

Anesthesia Oral Board Review



KNOCKING
OUT THE
BOARDS

edited by
Jessica A. Lovich-Sapola

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ANESTHESIA ORAL BOARD REVIEW: Knocking Out the Boards

The accreditation process for anesthesia in the United States is considered one of the most difficult in all medical specialties, with residents required to pass both an oral and a written exam to gain certification. This book is specially designed for the American Board of Anesthesiology Oral Examination. The evidence-based approach is presented in a concise outline-oriented format, with an emphasis on the knowledge necessary to pass the anesthesia oral boards. The Knockout Treatment Plan demonstrates the correct method of managing the case to the satisfaction of the examiners, whereas the Technical Knockout sections give additional tips for successfully passing the examination. The straightforward format of this book makes it suitable not only as an oral board review book but also as an introduction to anesthesia rotations for medical students, anesthesiologist assistant students, and nurse anesthetist students; furthermore, the book can be used as a technical study guide for anesthesia residents. More than 100 topics in this book have already been board-review tested by residents.

Dr. Jessica A. Lovich-Sapola is Assistant Clinical Professor of Anesthesiology at Case Western Reserve School of Medicine and Director of Pre-Surgical Testing in the Department of Anesthesiology at MetroHealth Medical Center, Cleveland, Ohio.

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Edited by

Jessica A. Lovich-Sapola

MetroHealth Medical Center, Cleveland, Ohio



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Every effort has been made in preparing this book to provide accurate and up-to-date information that is in accord with accepted standards and practice at the time of publication. Although case histories are drawn from actual cases, every effort has been made to disguise the identities of the individuals involved. Nevertheless, the authors, editors, and publishers can make no warranties that the information contained herein is totally free from error, not least because clinical standards are constantly changing through research and regulation. The authors, editors, and publishers therefore disclaim all liability for direct or consequential damages resulting from the use of material contained in this book. Readers are strongly advised to pay careful attention to information provided by the manufacturer of any drugs or equipment that they plan to use.

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Contributing Editors

EDITOR

Jessica A. Lovich-Sapola, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Pre-Surgical Testing
Co-Director of Remote Anesthesia
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Associate Editor

Brian M. Sapola

Adjunct Instructor of Biology
Cuyahoga Community College
Cleveland, Ohio
M.S. Candidate in Physiology
Kent State University
Kent, Ohio

Consulting Editors

Norman Bolden, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Obstetric Anesthesia
Vice Chairman
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Jeffrey A. Brown, D.O.

Clinical Instructor
University of Alabama at Birmingham School of Medicine
Department of Anesthesiology
Birmingham, Alabama

Gregory Kitagawa, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Obstetrics and Gynecology
MetroHealth Medical Center
Cleveland, Ohio

Edward S. Kosik, D.O.

Instructor of Anesthesiology
Texas Tech University Health Sciences Center
Lubbock, Texas

Christopher R. McHenry, M.D.

Professor of Surgery
Case Western Reserve University School of Medicine
Vice Chairman
Department of Surgery
MetroHealth Medical Center
Cleveland, Ohio

Kasia Petelenz Rubin, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Anesthesiology
University Hospitals/Rainbow Babies and Children's
Hospital
Cleveland, Ohio

Kanwaljit Rani Sidhu, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Charles E. Smith, M.D.

Professor
Case Western Reserve University School of Medicine
Director of Cardiothoracic Anesthesia and Trauma
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Kutaiba Tabbaa, M.D.

Clinical Associate Professor
Case Western Reserve University School of Medicine
Director of Chronic Pain Management
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Contributing Authors

Brendan J. Astley, M.D.

Senior Clinical Instructor
Case Western Reserve University School of Medicine
Department of Anesthesiology and Pain Management
MetroHealth Medical Center
Cleveland, Ohio

Michael Dale Bassett, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Andrew M. Bauer, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Lisa L. Bethea, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Norman Bolden, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Obstetric Anesthesia
Vice Chairman
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Vera V. Borzova, M.D., FAAP

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio
Pediatric Anesthesia Fellow
Department of Pediatric Anesthesia
Cleveland Clinic Foundation
Cleveland, Ohio

Jeffrey A. Brown, D.O.

Clinical Instructor
University of Alabama at Birmingham School of Medicine
Department of Anesthesiology
Birmingham, Alabama

Melvyn J. Y. Chin, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Sheila Chiu-Miller, D. O., M.S.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Varun Dixit, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Jennifer Eismon, M.D.

Senior Clinical Instructor
Case Western Reserve University School of Medicine
Co-Director of Remote Anesthesia
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Ryan J. Gunselman, M.D.

Chief Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Maureen S. Harders, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Ambulatory Anesthesia
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Contributing Authors

Alma Hoxha, M.D.

Senior Clinical Instructor
Case Western Reserve University School of Medicine
Department of Anesthesiology
Louis Stokes Cleveland VA Medical Center
Cleveland, Ohio

Kelly P. Jones, M.D.

Chief Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Matthew A. Joy, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Robert C. Lee, M.D.

Resident Physician
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Jessica A. Lovich-Sapola, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Pre-Surgical Testing
Co-Director of Remote Anesthesia
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Joni Maga, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Regional and Acute Pain Medicine
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Donn Marciniak, M.D.

Staff Anesthesiologist
Department of Cardiothoracic Anesthesia
Cleveland Clinic Foundation
Cleveland, Ohio

Cristian M. Prada, M.D.

Senior Clinical Instructor
Case Western Reserve University School of Medicine
Director of the Post-Anesthesia Care Unit
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Aditya Reddy, M.D.

Cardiothoracic Anesthesia Fellow
Department of Cardiothoracic Anesthesia
Cleveland Clinic Foundation
Cleveland, Ohio

Kasia Petelenz Rubin, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Anesthesiology
University Hospitals/Rainbow Babies and Children's
Hospital
Cleveland, Ohio

Kamaljit K. Sidhu, M.D.

Resident Physician
Department of Physical Medicine and Rehabilitation
MetroHealth Rehabilitation Institute of Ohio
Cleveland, Ohio

Kanwaljit Rani Sidhu, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Donald M. Voltz, M.D.

Assistant Clinical Professor
Case Western Reserve University School of Medicine
Director of Vascular Anesthesia
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

Karl Wagner, M.D.

Senior Clinical Instructor
Case Western Reserve University School of Medicine
Department of Anesthesiology
MetroHealth Medical Center
Cleveland, Ohio

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I want to especially thank my wonderful husband, Brian, and my two great kids, Rachel and Jeremy, for allowing me the time to work on this project. They understand how important it is for me to help my colleagues to not only pass this exam and become board certified but also become amazing anesthesiologists.

To all of the authors and consulting editors, I appreciate all of your time and hard work, more than you could imagine. This book would not have been possible without all of your help. Thank you!

I want to send a special thanks to my father, James Lovich, who put in hours of his time to draw many of the book's illustrations.

I also want to thank the anesthesia department at MetroHealth Medical Center in Cleveland, Ohio, for helping me to become the anesthesiologist I am today.

Letter from the Associate Editor

I would like to make a few comments about Dr. Lovich-Sapola – about the person, the physician, and the professional – to give you, the reader, some insight into how this book has come about, my role as assistant editor, and our intended purpose for this book.

First, let me introduce you to “Jessica,” as I call her. Of course, I could tell you about how we met, got married, had two beautiful children, and so on, but what is important to you is this: I have never met a more ambitious learner than Jessica. That is how this book came about. She was so intent on mastering the ABA oral examination that when she was studying she took meticulous notes on every possible question the examiners could ask. She became infatuated with that test. Of course, she passed and became board certified. But then she had a bunch of notes, and she formulated these into case studies and reviews that she shared with her residents and candidates for the test. The residents said, “This could be a book, Dr. Lovich.” And that’s how this book happened.

If you have read the list of editors and contributors, you might be wondering why I’m the associate editor for the book. You might be saying, “This guy is not a board-certified anesthesiologist. He’s not even a doctor!” I do teach anatomy and physiology to nursing students, but I’ve only been in an operating room as a patient. So, why did she ask me, other than for the sake of convenience?

Well, I have something in common with you. We’re both *not* board certified anesthesiologists! I have edited every line of this book to make each chapter independently readable, understandable, and free of jargon. I wanted this book to introduce the science, the physiology, the acronyms, and the materials and methods in a way that makes sense. Therefore, not only is this book intended to be the must-have anesthesia oral board review book for the ABA candidate, but it should be an invaluable reference for the anesthesia resident as well as preparation for medical students or anesthesiologist students on an anesthesiology rotation at a hospital. I was the medical knowledge litmus test for my wife. I made sure that this book didn’t intimidate you or confuse you by assuming you know all of the anesthesia acronyms. She made sure that it didn’t insult your anesthesia knowledge. Both of us want you to become a competent, board-certified anesthesiologist.

If you are a medical student and you are going into an anesthesia rotation, you can look up cases and read the clinical issues so that you know what’s going on in the operating room. If you are a PGY-1 intern, this book will give you some background knowledge that you can use from CA-1 anesthesia residency day one. If you are an anesthesia resident, you can read the Knockout Treatment Plan to be knowledgeable for when the attending starts grilling you. And, of course, to the ABA candidates, you want to read this entire book, including the TKO technical knockout sections that tell you how to approach the answer on the day of your oral exam in order to satisfy the examiners and pass. In fact, you’re going to do more than pass – you’re going to KNOCK IT OUT!

Brian M. Sapola

How to Use This Book

This book is an anesthesia oral board review book. I have tried to cover all of the essential topics required to pass the anesthesia oral boards. It does not have every question that you could be asked, nor is it exhaustively complete. This book should be used as a guide to understand and memorize facts so that you can easily discuss the topics as an anesthesia consultant. For a more in-depth understanding of the topics, the major textbooks or journals should be referenced. However, you should know all of the information presented in this book like the back of your hand. The more prepared you are, the easier it will be for you to think on your feet during the actual exam. Memorizing this book will not guarantee a passing score. You must take this knowledge and practice, practice, practice. The key to passing the oral boards is oral preparation. You must take this basic knowledge and then practice answering questions out loud. Study hard. Good luck!

Format

Each chapter starts with a **sample case**. This is an example of how the topic may be presented on the real exam. The **clinical issues** section covers specific details that require your time to understand, learn, and memorize. The final section is the **KO treatment plan**. This section usually references the sample case. It gives you a written dialogue demonstrating how you could answer the sample case questions on the actual exam. Prior to reading this section, try to talk out loud about what you would say and do in the sample scenario. Then read this section. At times, there will be technical **TKO** sections that stress the points that you need to memorize in order to pass the exam.

Oral Boards Tips for Success¹

Criteria for Becoming a Board-Certified Anesthesiologist

1. Complete an approved anesthesia residency accredited by the ACGME.
2. Pass the ABA Written Board Exam.
3. Pass the ABA Oral Board Exam.
4. Have adequate physical and sensory faculties.
5. Be free from the influence of or dependency on chemical substances.
6. Have no felony on record.

Oral Exam

Starting Note

1. You walk in the door **PASSING**.
2. You have 70 minutes to **NOT** prove otherwise to the examiners.
3. Statistically, your best chance for passing the exam is the first time you take it.
4. The ABA's general recommendations are these:
 - a. Study, especially the topics you are the least comfortable with.
 - b. Practice daily.
 - c. Use your daily cases as a chance to talk through your plan.
 - d. Read journal articles.

Location of the Oral Board Exam

1. The hotel location is chosen almost 5 years in advance.
2. It must be in a city with a big airport.
3. The hotel must not be prohibitively expensive.
4. The hotel must be large enough to accommodate the exam.
5. The location must be in an area where the weather is generally good.

Dress Code

1. Men: coat and tie.
2. Women: office attire.



Photo.1. Picture of a hotel in which the oral boards have been given. Photo credit: J. Lovich-Sapola, MD.

3. I recommend a black suit for men.
4. Most women also wear a black pants suit.

Behavior

1. Maintain good eye contact.
2. Speak up.
3. Maintain good posture.
4. Act professional.
5. Do not argue with the examiners.
6. Give the examiners a firm handshake at the beginning and the end of the exam, even if you feel that you did poorly.
7. Avoid slang and informality.
8. Don't play with your pen or jewelry.
9. Talk with the examiners like you are a colleague.
10. Do not talk down to the examiners or be overly cocky or smug.

What to Bring to the Exam

1. Basically nothing.
2. You can't bring anything into the room except a pen and your identification.



Photo.2. Picture of an oral board exam room. Photo credit: J. Lovich-Sapola MD.

Exam Room

1. The exam is taken in an actual hotel room.
2. Every room is adjusted for equal lighting and temperature.
3. In each room you are given water, a pen, and a piece of paper.
4. The examiners will verify the case with you.
5. They will check your wrist band.
6. The examiners will introduce themselves to you.
 - a. At this time you can switch rooms if you feel that you know an examiner.
 - b. There may be an observer in the room who does not grade you.

A Day in the Life of an Examinee

1. You will arrive at the hotel 15 minutes before your set time. You cannot go to the assigned room any earlier.
2. Take the elevator to your assigned room.
3. Bring your ID, sign in, and get a wrist band.
4. Briefing lasts about 1 hour.
5. You get the first exam to look at for about 10 minutes.
6. Examiners walk you to your assigned hotel room.
7. Sit in the chair and continue to read and write down notes about your case.
8. The examiners come and get you.
9. Exam #1.
10. Knock on the door.
11. Sit in the next seat and take the copy of the test off the door.
12. You get 10 minutes to prepare.
13. Suck it up between cases!
14. Relax and take a deep breath before entering the room.
15. Exam #2.
16. Go home.

Exam

1. The exam is based on general knowledge of all anesthesia-related fields.
2. The examiners follow a strict script.
3. The scripted format started 10 years ago, with strict enforcement of the scripting within the last 5 years.
4. The scripts are based solely on rescue scenarios.

Who Writes the Exam?

1. Practicing anesthesiologists who serve as examiners submit the cases.
2. The ABA takes care to ensure reasonable content sampling.

What Facts Do They Expect the Examinee to Know?

1. In-depth knowledge of all drugs used and their effects on normal and abnormal body functions.
2. Pathogenesis.
3. Alternate methods of management.
4. Mechanism of drug action.
5. Methods of measurement including routine lab studies and normal measurements.
6. Ability to anticipate, diagnose, and provide rational therapy for any complications that are likely to arise.

Format

1. Briefing session.
2. Two parts, 35 minutes each.

Part A

1. 10 minutes to look at the information. Take notes.
2. Intra-operative: 10 minutes (senior examiner).
3. Post-operative/critical care: 15 minutes (junior examiner).
4. Three extra topics: 10 minutes (senior examiner).

Part B

1. 10 minutes outside the exam room to look at the case. Take notes.
2. Pre-operative: 10 minutes (senior examiner).
3. Intra-operative: 15 minutes (junior examiner).
4. Three extra cases: 10 minutes (senior examiner).

Cases and Examiners

1. The same case is being presented in all of the exam rooms on all of the hotel floors at the same time.
2. No case is reused during the week.
3. You have two examiners at each session, four for your entire exam.
4. The examiners also change rooms during each set of exams.

Audits of the Exam

1. The actual exam questions are graded by the examiner for content and difficulty prior to giving the exam.
 - a. Content: excellent, good, acceptable, marginal.
 - b. Difficulty: too difficult, appropriate level of difficulty, or too easy.
2. This score is also used in the final grading.

Examiners Are Briefed on Their Roles

1. They get the exam the night before.
2. They are able to look up the general topics.
3. They are told to limit their preparatory research.

What the Examiners Know About You

1. Your name.
2. That is it!!!

A Day in the Life of an Examiner

1. The examiners are in a single room for only part A and B of a single exam. They trade rooms. Rarely is the same team of examiners used throughout the week.
2. They finish their grading within 1-2 minutes of the completion of the exam. They do not discuss the examinee until they turn in the score sheet.

Audits of the Examiners

1. The examiners are audited a few times during the week.
2. The ABA maintains strict quality control.
3. If the ABA has a problem with an examiner, he or she is not asked to serve as an examiner again.
4. Each examiner is ranked yearly as being an easy, moderate, or hard examiner.

What Are Examiners Audited For?

1. Questioning
 - a. Asking vague questions.
 - b. Asking confusing questions.
 - c. Asking for facts instead of judgment (giving a superficial exam).
 - d. Being unprepared to ask another question.
 - e. Giving inappropriate positive or negative reinforcement.
 - f. Asking rhetorical questions.
 - g. Acting in an aggressive or threatening manner.
 - h. Asking multiple questions without waiting for a response.
 - i. Pursuing factual minutiae.
 - j. Staying on schedule.
 - k. Covering all of the script.
 - l. Knowing when to change topics.
 - m. Being well prepared and informed.

- n. Asking too many yes/no questions. They should ask more open-ended questions.
 - o. Examiners should be unemotional and give no feedback.
2. Evaluating
 - a. Not taking into account the difficulty of the question.
 - b. Not recognizing non-gradable questions/answers.
 - c. Trying to guess the co-examiner's rating and matching those ratings.
 - c. Fretting over a split with a co-examiner leading to failure to concentrate on the next examination.

Diplomate Attributes

1. Application of knowledge
 - a. Be able to apply the factual knowledge to a clinical scenario. The primary goal is not the recall of cognitive information.
 - b. Show the ability to assimilate and analyze data so as to arrive at a rational treatment plan.
2. Judgment
 - a. Use sound judgment in making and applying decisions.
3. Adaptability
 - a. Be able to respond to a change in the patient's clinical condition.
 - b. Be willing to change your plan in response to a change in the situation or patient's condition.
4. Organization and presentation
 - a. Do you communicate well with peers, patients, family, and community?
 - b. Are you an anesthesia consultant?
 - c. Can you be a leader of an anesthesia care team?
 - d. Can you prioritize and organize your presentation?
 - e. Can you structure your answers?
 - f. Are you able to define the priorities in the care of the patient?

Scoring

1. You are not scored on one question. You are scored overall.
2. In the past, a person may have failed over one missed critical question. This is not true of the current exam.
3. The score is related to the difficulty of the test.
4. The score is also related to the difficulty of the examiner.
5. The score is scaled.
6. The score is based on the exam and the examiner.
7. The analysis is multifaceted.
8. The failure rate is consistently about 20%.
9. One examiner can't fail you!!!!

Be Able to Answer

1. Why?
2. Why not?
3. Why not something else?

Tips for Answering the Question

1. At times, there is NO right or wrong answer!
2. Don't be too regimented.
3. It is OK to say you are not comfortable with a certain technique, but you must know that it is possible.

Questions?

1. Just answer the question.
2. Do not ask questions. Examiners don't have any more information than they have told you.
3. You can ask for a clarification if you really don't know what they are asking.
4. Assume ...
5. Always assume that your patient is healthy. The examiner will let you know if this is not the case.

You Are Asked a Question ...

1. Listen to the question and answer it.
2. Then immediately justify why that was your answer.
3. Say I am doing "X" and this is why.
4. Examiners don't want to hear all the things you could do. Pick one!
5. Say "I would," not "I could ..."
6. They expect you to be able to defend your selected plan of management.
7. They will interrupt when you have said enough.
8. Explain things to the examiners assuming they do not understand anesthesia.
9. The explanation is more important than the answer.

More Tips

1. Imagine yourself in the OR. Only do things that you would normally do.
2. Don't be afraid to "consult" another service or physician. This shows that you know when to ask for help as opposed to compromising the patient's safety.
3. Write down any numbers or labs the examiners give you.
4. If you do not know the answer, say "I don't remember at this time." Don't ever make up answers.
5. Don't quote a book or article unless you are prepared to have a detailed discussion.
6. Always keep the patient safe!!!!

Bad Things Happen

1. Bad things are going to happen, no matter how good you are.
2. They are written into the script.
3. Treat the problem and don't stress over whether it was your fault.

So You Realize You Made a Mistake ...

1. They don't want you to be wishy-washy, so stick to your guns.
2. But don't go down with the sinking ship.
3. If you realize that you made a big, killing mistake, say, "I am sorry, but I ..."

Problem Candidates ...

1. Have superficial knowledge.
2. Have good knowledge, but ...
 - a. They can't apply it or adapt to clinical conditions.
 - b. They can't express or defend why they do something.
 - c. They don't realize that "we do it every day at my hospital" does not count.
3. Try to control the exam by
 - a. Asking too many questions.
 - b. Giving deliberately slow responses.
4. Talk a lot, but say little.
5. Can't express ideas or defend a point of view in a convincing manner.
6. Are indecisive.
7. Show faulty judgment.
8. Do not take the test seriously and are not prepared.

Failure

1. In case you have to retake the exam
 - a. You will never be re-examined by the same people.
 - b. The examiners have no way of knowing if this is your 1st or 20th time taking the exam.

BIBLIOGRAPHY

1. 2008 ABA Non-examiner workshop.
Fort Lauderdale, FL
April 14-15, 2008

PART ONE

General Information

1. Standard ASA Monitors¹

Karl Wagner, MD

SAMPLE CASE

A 30-year-old male comes to the radiology suite for an MRI of his lower spine for cauda equina syndrome. He has lower extremity weakness, morbid obesity, and obstructive sleep apnea. He is too afraid to enter the scanner and would rather be paralyzed than get into the machine. The only way an MRI will be obtained is if the patient has general anesthesia as per the radiologist. He is fasted and ready to go in the scanner when you get the call.

CLINICAL ISSUES

The “Standard ASA Monitors” is a buzz phrase that we use every day in practice. We will also use the term when we sit for the boards. Don’t forget what the monitors are.

1. Standard I: A person who is qualified to monitor, evaluate, and care for the patient must be present in the room.
2. Standard II: During all anesthetics, the patient’s oxygenation, ventilation, circulation, and temperature should be continuously evaluated.
 - a. Oxygenation
 - i. Oxygen analyzer: used to measure the oxygenation of the inspired gas.
 - ii. Pulse oximeter: used to measure the oxygenation of the blood.
 - b. Ventilation
 - i. The adequacy of ventilation should be monitored at all times.
 - ii. Monitor expired carbon dioxide (EtCO₂)
 - iii. Respiratory volumes (if on a ventilator) should be measured.

- iv. Disconnect alarms on the ventilators should be used.
 - v. It is important to have audible alarms when practical and available.
 - c. Circulation
 - i. EKG, blood pressure, and heart rate must be measured and evaluated at least every five minutes.
 - ii. We also must be able to palpate pulses, auscultate heart sounds, and/or visualize a continuous arterial wave form or pulse using plethysmography.
 - d. Body temperature
 - i. Temperature should be continuously monitored in all patients receiving anesthesia.
 - ii. This is especially important if clinically significant changes in body temperature are anticipated.



KO TREATMENT PLAN

This patient will be paralyzed, secondary to the cauda equine syndrome, if he does not get the MRI scan. The surgeons cannot operate without the scan. No matter what technique you choose (laryngeal mask airway or endotracheal tube) you have to monitor all of his vital signs. Don’t let the radiology technicians tell you it is only a short scan and that you do not need monitors. Also, don’t fall for a line such as “you can’t take these monitors in the scanner.” Every anesthetic gets the standard ASA monitors.

BIBLIOGRAPHY

1. These standards were approved by the House of Delegates on October 21, 1986, and last amended on October 25, 2005. They can be viewed at the ASA website www.asahq.org/publicationsAndServices/standards/02.pdf.

2. Pulse Oximetry^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

You are called to the emergency room to evaluate the airway of a patient found unconscious after a failed suicide attempt. He was found in his garage with his car running. His pulse oximetry reading is 97%. He is very somnolent. You determine that the patient needs to be intubated. The medical student standing nearby wants to know why you would intubate him if his “oxygenation” is normal.

CLINICAL ISSUES

Mechanism of Function

1. The color of the blood is a function of oxygen saturation.¹
2. The change in color results from the optical properties of hemoglobin and its interaction with oxygen.¹
3. The ratio of oxyhemoglobin and reduced hemoglobin can be determined by absorption spectrophotometry.¹
4. Light-absorbance measurements of pulsatile blood are used to determine the concentration of various species of hemoglobin.²
5. Adult blood contains four species of hemoglobin: oxyhemoglobin, reduced hemoglobin, methemoglobin, and carboxyhemoglobin.²
6. Each of these species of hemoglobin has a different light absorption profile.²
7. The oxygen saturation is determined by using the Beer-Lambert law.¹
8. Two wavelengths of light are used to distinguish oxyhemoglobin from reduced hemoglobin.¹
9. Light-emitting diodes (LEDs) in the pulse sensor emit red (660 nm) and near infrared (940 nm) light.¹
10. The percentage of oxygenated and reduced hemoglobin is determined by using a ratio of infrared to red light sensed by the photodetector.¹
11. The pulse oximeter then determines the difference in the amount of oxygenated hemoglobin in the pulsatile arterial blood from the nonpulsatile venous blood.¹

Indications

1. Standard ASA monitors.
2. Measurement of the pulse rate.
3. Measurement of the oxygen saturation of hemoglobin.
4. Tool for early warning of hypoxemia.

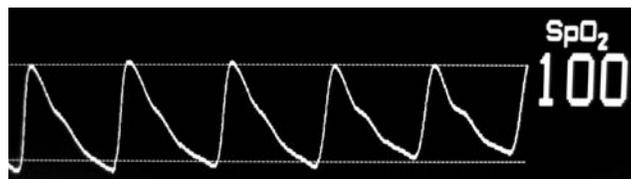


Figure 2.1. Pulse oximetry reading. Photo credit: J. Lovich-Sapola, MD.

Benefits

1. Noninvasive
2. Continuous monitor
3. Can be used on patients of all age groups
4. Simple
5. Autocalibrating
6. Reliable

Causes of Inaccuracy

1. Dyshemoglobins
2. Vital dyes
3. Nail polish (greatest effect with blue)
4. Ambient light
5. Light-emitting diode variability
6. Motion artifact
7. Background noise
8. Electrocautery
9. Loss of signal with hypoperfusion
10. Black henna

Errors in Pulse Oximetry

1. Carboxyhemoglobin: overestimates the fraction of hemoglobin available for oxygen transport.
2. Methemoglobin: at high levels, the SpO₂ approaches 85%, independent of the actual oxygen arterial saturation (SaO₂).
3. Hemoglobin Köln: reduction of 8–10%.
4. Transient decrease with indigo carmine, indocyanine green, high dose isosulfan blue, and methylene blue.

Co-oximeter

1. The SpO₂ measured by pulse oximetry is not the same as the arterial saturation (SaO₂) measured by a laboratory co-oximeter.¹
2. The pulse oximeter measures “functional” saturation.¹

3. The co-oximeter uses multiple wavelengths to distinguish other types of hemoglobin by their characteristic absorption.¹
4. The co-oximeter measures the “fractional” saturation.¹
5. If other hemoglobin moieties are present, the SpO₂ measurement will be higher than the SaO₂ reported by the laboratory.¹
6. Methemoglobin and carboxyhemoglobin are usually at such low concentrations in a normal patient that the functional saturation approximates the fractional value.¹



KO TREATMENT PLAN

The sample patient obviously has carbon monoxide poisoning. Despite a “normal” pulse oximetry reading, one must assume that his true arterial oxygen saturation is likely low.

An arterial blood gas should be sent immediately for a co-oximetry reading. This will give you the true levels of oxyhemoglobin and carboxyhemoglobin. Not only is the falsely high pulse oximetry reading deceiving, but also the characteristic cherry red appearance of the patient with high carbon monoxide levels can be deceiving as to the actual extent of the hypoxia. Carboxyhemoglobin is treated with oxygen therapy. The patient should be receiving 100% oxygen. Since this patient is somnolent and likely not able to protect his airway, intubation may be necessary.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 672.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1213–5, 1448–53.

3. Capnography^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

The patient is an 85-year-old female undergoing an anterior cervical fusion. One hour into the case her end-tidal carbon dioxide (EtCO₂) begins to drop from 35 to 15 mm Hg. What is your differential diagnosis? How will you treat this?

CLINICAL ISSUES

Terminology

1. Capnography: the numerical measurement and graphic waveform display of the CO₂ concentration versus time or expired volume. Capnography provides three sources of information: numerical CO₂ values, the capnogram shape, and the arterial and end-tidal PCO₂ difference.¹
2. Capnograph: the machine that generates the waveform.¹
3. Capnogram: the continuous graphic waveform representation of the CO₂ concentration over time. Characteristic waveforms can help in the diagnosis of underlying clinical or technical abnormalities such as partial airway obstruction, accidental extubations, circuit disconnects, and hypermetabolic states. This early recognition of a life-threatening problem allows for early intervention before irreversible damage occurs.^{1,3}
4. Capnometry: the measurement and numerical display of maximum inspiratory and expiratory CO₂ concentrations during a respiratory cycle.^{1,3}

5. Capnometer: the device that performs the measurement and displays the readings.¹
6. EtCO₂: end-tidal carbon dioxide; the measurement of the concentration of CO₂ at the end of exhalation. Normal value of partial pressure ranges between 35 and 45 mm Hg.¹
7. PaCO₂: the partial pressure of CO₂ in the arterial blood.¹
8. a-ADO₂: the difference between EtCO₂ and PaCO₂. This is normally 2 to 5 mm Hg. The number increases with age, emphysema, pulmonary embolism, decreasing cardiac output, hypovolemia, and with anesthesia. The number decreases with large tidal volumes and low frequency ventilation.¹

Clinical Application

1. Causes of decreased EtCO₂
 - a. Decrease in metabolic rate
 - i. Hypothermia
 - ii. Hypothyroidism
 - b. Change in elimination
 - i. Increased dead space/COPD
 - ii. Hyperventilation
 - iii. Decreased cardiac output/cardiac arrest
 - iv. Decreased CO₂ production
 - v. Circuit leak or occlusion
 - vi. Pulmonary embolism (air, thrombus, gas, fat, marrow, or amniotic)

3. Capnography

- c. Other
 - i. Increased muscle relaxation
 - ii. Increased depth of anesthesia
 - iii. Surgical manipulation of the heart or thoracic vessels
 - iv. Wedging of the pulmonary artery catheter
- 2. Causes of increased EtCO₂
 - a. Increased metabolic rate
 - i. Increased CO₂ production (malignant hyperthermia, thyrotoxicosis, and hyperthyroidism)
 - ii. Hyperthermia
 - iii. Shivering or convulsions
 - iv. Sepsis
 - b. Change in elimination
 - i. Rebreathing (valve prolapse, failed CO₂ absorber)
 - ii. Hypoventilation
 - iii. Depression of the respiratory center with a decrease in tidal volume
 - iv. Reduction of ventilation (partial paralysis, neurologic disease, high spinal anesthesia, weakened respiratory muscles, or acute respiratory distress)
 - v. Increased or improving cardiac output
 - vi. Right to left intracardiac shunt
 - c. Other
 - i. Excessive catecholamine production
 - ii. Administration of blood or bicarbonate
 - iii. Release of an aortic/arterial clamp or tourniquet with reperfusion to ischemic areas
 - iv. Glucose in the IV fluid
 - v. Parenteral hyperalimentation
 - vi. CO₂ used to inflate the peritoneal cavity during laparoscopy, pleural cavity during thoracoscopy, or a joint during arthroscopy
 - vii. Subcutaneous epinephrine injection
- 3. Causes of minimal to zero EtCO₂ or a sudden drop to near zero
 - a. Equipment malfunction
 - b. Endotracheal tube (ETT) disconnect, obstruction, or total occlusion
 - c. Bronchospasm
 - d. No cardiac output
 - e. Cardiac arrest
 - f. Bilateral pneumothorax
 - g. Massive pulmonary embolism
 - h. Esophageal intubation
 - i. Application of positive end expiratory pressure (PEEP)
 - j. Cricoid pressure occluding the tip of the ETT
 - k. Sudden, severe hypotension

4. Errors in capnography

- a. Water vapor
- b. Disconnect



KO TREATMENT PLAN

Intra-operative

1. Capnography is useful for verifying the position of the ETT, providing information about CO₂ production, pulmonary perfusion, alveolar ventilation, respiratory patterns, and the elimination of CO₂ from the anesthesia circuit and ventilator.¹
2. Capnography is a rapid and reliable method to detect life-threatening conditions, such as malposition of the ETT, ventilatory failure, circulatory failure, and defective breathing circuits.¹
3. With the sample patient, you need to quickly rule out all of the potentially life-threatening conditions associated with a decrease in EtCO₂, including decreased cardiac output, cardiac arrest, ETT obstruction/malposition, ventilator malfunction, embolism, and oversedation. Call for help. Look at the patient's vital signs. Feel for a pulse. Take the patient off the ventilator and then hand-bag while listening for bilateral breath sounds. If you presume the patient to be in a low cardiac output state, turn off your anesthetics and start ACLS as needed. If you presume a ventilatory problem, troubleshoot for the problem.
4. During ACLS resuscitation, exhaled CO₂ is a better guide to the presence of circulation than the EKG, pulse, or blood pressure. The effectiveness of the resuscitation can be measured by capnography. The capnogram is not susceptible to the mechanical artifacts associated with chest compressions. However, if high dose IV epinephrine or bicarbonate is used, the EtCO₂ would not be an effective indicator to the resuscitation. A sudden increase in the EtCO₂ during the resuscitation is an early clue that spontaneous cardiac output has been restored.¹

BIBLIOGRAPHY

1. D'Mello J, Butani M. Capnography. *Indian J Anaesth*, 2002;46(4):269-78.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1455-62.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 670-1.

4. Electrocardiogram (EKG)^{1,2,3}

Karl Wagner, MD

SAMPLE CASE

A 75-year-old male comes to the operating room (OR) for a laparoscopic cholecystectomy. His past medical history is significant for hypertension controlled with metoprolol and hydrochlorothiazide (HCTZ) and chronic smoking. He reports good functional capacity and is able to care for his lawn and house. He does not have an EKG on file. Should you delay the case in order to get an EKG?

CLINICAL ISSUES

This patient overall enjoys good health and he is considered an ASA class 2. However, he has a few minor indicators of cardiovascular disease. He is an older male with chronic hypertension and he smokes.

Mechanism of Function

1. The electrocardiogram (EKG) is a measurement of the electrical activity of the heart.
2. The waveforms generated by the electrical impulses of the conduction system give a glimpse into the function of the heart.
3. The measurements are time (seconds) on the x-axis and electromotive force (mV) on the y-axis.
4. The paper speed is 25 mm per second
5. By convention, 1 mm corresponds to 0.04 seconds (x) or 0.1 mV (y).

How to Read an EKG

1. When reading the EKG it is important to have a system/method for evaluation to ensure maximum diagnostic value.
2. Below is how I approach reading EKG's, but this was learned from reading other authors.^{1,2,3}

Rhythm: sinus or non-sinus

Regularity: regular or irregular

P wave: generated by atrial depolarization.

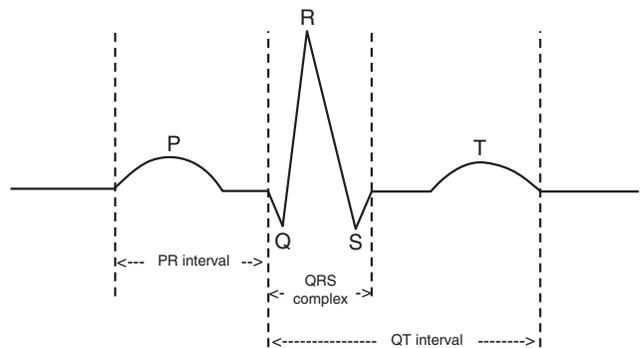


Figure 4.1. Basic EKG. Drawing credit: J Lovich-Sapola MD.

Table 4.1. EKG Rate

Rate	Diagnosis
60–100	Normal
<60	Bradycardia
>100	Tachycardia

1. It should be 2.5 mm long and 2.5 mm high.
2. Best viewed in lead II and V1. Therefore, lead II is commonly monitored in the OR because it is the most sensitive for diagnosing arrhythmias.

PR interval: generated by the conduction of the electrical impulse through the atria and the AV node.

1. This should not be longer than 0.2 seconds (5 mm).
2. The duration and comparisons of the PR intervals give an insight into the depolarization of and conduction through the atria.
3. First degree heart block has a PR interval longer than 0.2 seconds. The shape of the wave is unchanged.
4. Second degree heart blocks
 - a. Mobitz Type 1 (Wenckebach)
 - i. Repeating cycles of lengthening PR intervals until there is a dropped beat.
 - b. Mobitz Type 2
 - i. Dropped beats with uniformly prolonged PR intervals.

4. Electrocardiogram

5. Third degree heart block is a complete dissociation of the atria and ventricles.

- a. There is not a QRS complex after every P wave.

TKO: Remember that second degree type 2 and third degree heart blocks require cardiac pacing.

Q wave: should not be longer than 0.03 seconds (mm).

- a. Greater than 0.03 seconds (mm) can be a sign of transmural infarction.
- b. A prolonged Q wave is a sign of a post-infarction scar and not a sign of acute ischemia.

QRS complex: this is the time for ventricular depolarization and contraction.

- a. Normal duration is up to 0.12 seconds.
- b. Longer duration is a sign of hemi-block in the His bundles.
- c. Look for bundle branch blocks here as well.
 - 1. If there is an R and R' in lead V1, then suspect a right bundle branch block (RBBB).
 - 2. If there is R and R' in lead V6, then suspect a left bundle branch block (LBBB).

ST segment: this should be isoelectric.

- a. If it is depressed, then there is myocardial ischemia.
- b. If it is elevated, then there is myocardial necrosis.
- c. This is measured by the computer on the monitors and we need to take them seriously! Our patients are usually asleep and can not tell us if they have chest pain.
- d. The most sensitive lead for diagnosis of ischemia is chest lead V5.
- e. The next most sensitive is chest lead V4.

T wave: this depicts ventricular repolarization.

- a. It should not be longer than 0.2 seconds.
- b. It should be concordant with the total amplitude of the QRS complex.

Axis: normal values range between -30° and $+110^\circ$.

- a. Look at the total amplitude of lead I and lead aVf.
- b. The vector of these two leads is where the axis lies.

The EKG is a diagnostic test for the heart. We monitor the EKG every day in the OR because we love the heart. We can not always use a 12 lead EKG for continuous monitoring, however; we do use limb lead II to watch for arrhythmias and chest lead V5 to watch for ischemia. There are also other physiologic abnormalities that can cause EKG changes that will be mentioned in later sections. It is important to keep in mind that patients who are asymptomatic but do have chronic cardiac disease may have a normal EKG.⁴ Additionally, patients who have EKG abnormalities pre-operatively along with chronic cardiac disease have a higher risk associated with non-cardiac surgery.³



KO TREATMENT PLAN

- 1. This sample patient should get an EKG. He is greater than 50 years old and has a history of hypertension.
 - a. ASA recommendations for a preoperative EKG
 - i. Age greater than 50 years old
 - (1) Good for one year if age 50–69.
 - (2) Good for 6 months if age >69.
 - ii. History of cardiovascular disease or hypertension
 - (1) EKG is only good for 6 weeks in a patient with significant cardiovascular disease.
 - (2) EKG is mandatory if the patient has a change in cardiac symptoms: shortness of breath, chest pain
 - iii. History of diabetes mellitus
 - (1) EKG is required if the patient is >40 years old.
 - (2) EKG is required if he has had diabetes for >10 years regardless of the patient's age.
 - iv. Central nervous system disease

BIBLIOGRAPHY

1. O'Keefe JH, et al. *The Complete Guide to ECG's*, 2nd ed. Royal Oak, MI: Physicians' Press, 2002.
2. Dubin D. *Rapid Interpretation of EKG's*, 6th ed. Tampa: COVER Publishing, 2000.
3. Fleisher, Lee A. *Evidence-Based Practice of Anesthesiology*. Philadelphia: Saunders, 2004, pp 27–9.

5. Blood Pressure Monitoring¹

Karl Wagner, MD

SAMPLE CASE

A one-month-old male comes to the operating room (OR) for a pyloromyotomy. He has been volume resuscitated and has normal electrolytes. He has a 24-gauge IV in his right hand. What kind of blood pressure monitoring would you like for this patient?

CLINICAL ISSUES

One of the standards of anesthesia practice is to measure the blood pressure at least every five minutes. This can be done either non-invasively or invasively.

Non-invasive Blood Pressure (NIBP) Monitoring

1. Techniques
 - a. Manual cuff
 - b. Automatic cuff
2. Mechanism of function for the manual cuff
 - a. The cuff is inflated with a manometer to a pressure that is high enough to stop the flow of blood.
 - b. The pressure at which the flow returns is the systolic pressure.
 - c. The vessel is partially occluded, the flow at this point is turbulent, and the sounds heard are Korotkoff's sounds.
 - d. When the flow becomes laminar again, the sounds stop and this correlates with the diastolic pressure.
 - e. The bladder in the cuff should be large enough to cover 60% of the circumference of the arm and the width should be approximately 40% of the length of that limb segment.
3. Mechanism of function for the automatic cuff
 - a. Used more commonly
 - b. The cuff is inflated above the systolic pressure and the flow of blood is stopped.
 - c. The pressure in the cuff is decreased slowly, and once there is a return of blood flow through the artery, oscillations are detected.
 - d. The mean pressure is the point at which the oscillations are at their maximum.

e. The systolic and diastolic pressures are calculated values based on the mean and the rate of change in the oscillations.

4. Complications of NIBP monitoring
 - a. The cuff, while measuring pressure, is preventing blood flow to the extremity.
 - i. Patients have been reported to get compartment syndromes and neuropathies from overuse of the cuff.
 - b. There is a change of 0.7 mm Hg in pressure for each centimeter the cuff is above (lower readings) or below (higher readings) the heart.
 - i. Improper positioning may lead to inappropriate medical management.
 - c. The cuff measures oscillations; any disturbance of the cuff or patient's arm during the reading can introduce error into the results.

Invasive Arterial Blood Pressure Monitoring

1. Techniques
 - a. Catheters are inserted intra-arterially.
 - b. The most common arteries are the radial, brachial, axillary, femoral, and dorsalis pedis.
2. Mechanism of function
 - a. Pulse waves are transmitted along a column of saline to a transducer and the signal is converted into an electronic signal.
 - b. A wave form can be graphically displayed and used for analysis.
 - c. Waves can be amplified while traveling back and forth along the vessels.
 - i. This amplification also occurs in the tubing used to transmit the signal to the transducer.
 - ii. This amplification can result in "whip," an increased systolic pressure reading if the system is under-damped.
 - iii. If the system is over-damped it will read as an artificially low blood pressure.
 - iv. Use short, non-compliant tubing with saline to conduct the impulse to the transducer; this will minimize error in the pressure readings.

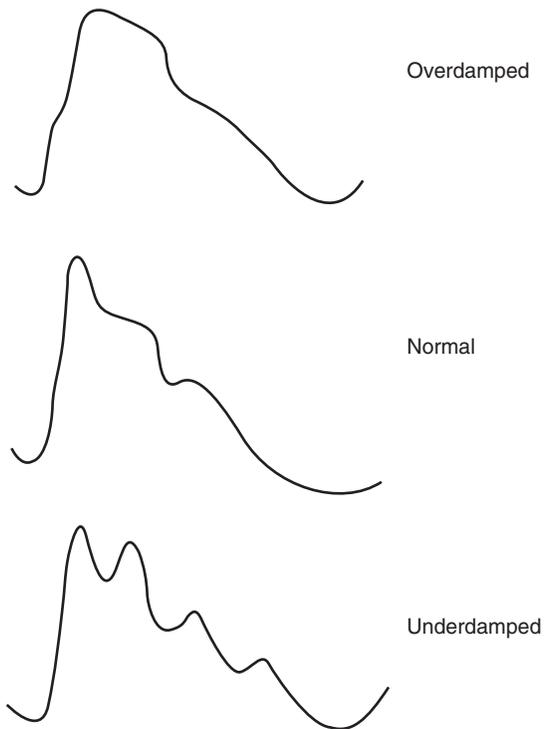
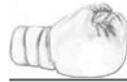


Figure 5.1. Arterial line dampening. Drawing credit: James Lovich.

d. The natural frequency of the measuring system is much higher than that of the vascular system and is thought to minimize error in readings because the system does not naturally increase its wave amplitude.

e. The system is described as “critically damped” when the readings are neither artificially high nor low, but are just right.

3. Complications of invasive arterial monitoring (see Chapter 6).



KO TREATMENT PLAN

Remember that this type of case is a medical emergency and not a surgical one. The patient is not an operative candidate until he has been adequately volume resuscitated. Once that occurs, then using a non-invasive blood pressure monitor is reasonable unless there are other variables or indications for an invasive line.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 672-6.

6. Indications, Complications, and Waveforms for an Arterial Line, Pulmonary Artery Catheter (PAC), and Central Venous Pressure Monitor (CVP)^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

ARTERIAL LINE^{1,2,3}

SAMPLE CASE

A 62-year-old female is scheduled for a carotid surgery. She has a history of Raynaud’s disease. Your colleague places a left radial arterial line for the surgery. You are called to the post-anesthesia care unit to evaluate the patient. Her left hand is blue.

CLINICAL ISSUES

Indications^{1,2}

1. Continuous, real-time blood pressure monitoring
2. Planned pharmacologic or mechanical cardiovascular manipulation
3. Repeated blood samplings
 - a. Arterial blood gas (ABG)
 - b. Hematocrit
 - c. Glucose

6. Indications, Complications, and Waveforms



Figure 6.1. Pseudoaneurysm of the radial artery. Photo credit: J. Lovich-Sapola, MD.

4. Failure of indirect arterial blood pressure measurement
5. Supplementary diagnostic information from the arterial waveform
 - a. Arterial pulse contour analysis
 - i. Systolic pressure variation
 - ii. Pulse pressure variation
6. Patient with end organ disease
7. Patient with large fluid shifts

Complications^{1,2}

1. Distal ischemia secondary to thrombosis, proximal emboli, or prolonged shock
2. Pseudoaneurysm
3. Arteriovenous fistula
4. Hemorrhage
5. Hematoma
6. Infection
7. Skin necrosis
8. Peripheral neuropathy and damage to adjacent nerves
9. Misinterpretation of data
10. Cerebral air embolism secondary to retrograde flow with flushing

Patients at Increased Risk of Complications^{1,2,3}

1. Severe atherosclerosis
2. Diabetes
3. Low cardiac output
4. Intense peripheral vasoconstriction: Raynaud's disease

Waveform Abnormalities^{1,2}

1. Aortic stenosis: pulsus parvus (narrow pulse pressure) and pulsus tardus (delayed upstroke)
2. Aortic regurgitation: bisferiens pulse (double peak) and wide pulse pressure

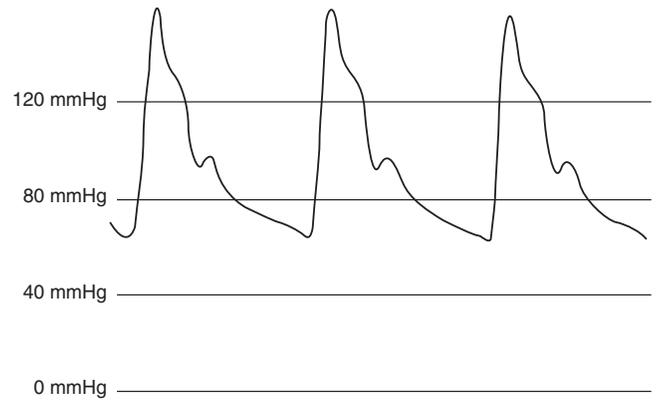


Figure 6.2. Normal arterial line pressure tracing. Drawing credit: James Lovich.

3. Hypertrophic cardiomyopathy: spike and dome (midsystolic obstruction)
4. Systolic left ventricular failure: pulsus alternans (alternating pulse pressure amplitude)
5. Cardiac tamponade: pulsus paradoxus (exaggerated decrease in systolic blood pressure during spontaneous inspiration)
6. Hypovolemia: exaggerated decrease in systolic blood pressure or pulse pressure during mechanical ventilation



KO TREATMENT PLAN

Pre-operative Treatment

1. Raynaud's disease is the episodic vasospastic ischemia of the digits.
 - a. Affects women more than men
 - b. Characteristic digital blanching and cyanosis after cold exposure

Intra-operative Treatment

1. Protect the patient's hands and feet from cold exposure.
2. Maintain the patient's core body temperature.
3. Non-invasive blood pressure monitoring techniques are recommended over invasive techniques secondary to the increased risk of ischemic injury.
4. The risk/benefit ratio of radial arterial cannulation must be considered for each patient.
5. Consider using a larger artery, such as the brachial or femoral artery, if an arterial line is necessary.

Post-operative Treatment

1. Go evaluate the patient.
2. Warm the patient and her left extremity especially.
3. Determine if this is a Raynaud's event or ischemia secondary to the arterial line.

6. Indications, Complications, and Waveforms

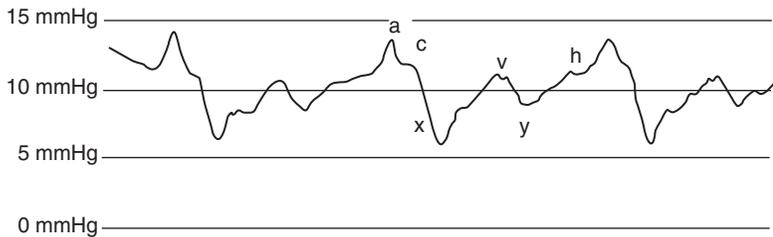


Figure 6.3. Normal CVP waveform. Drawing credit: James Lovich.

4. A stellate ganglion block may improve blood flow.
5. Vascular surgery should be consulted to evaluate the patient for a possible embolectomy.

CENTRAL VENOUS ACCESS AND CENTRAL VENOUS PRESSURE MONITORING (CVP)^{1,2}

CLINICAL ISSUES

Indications^{1,2}

1. CVP monitoring
2. Transvenous cardiac pacing
3. Required for the insertion of pulmonary artery catheters
4. Temporary hemodialysis
5. Drug administration: drugs that are irritating to peripheral veins
 - a. Vasoactive drugs
 - b. Hyperalimentation
 - c. Chemotherapy
 - d. Prolonged antibiotic therapy
6. Rapid infusion of fluids: trauma, major surgery
7. Major surgery with large fluid shifts
8. Aspiration of a venous air embolus
9. Inadequate peripheral access
10. Sampling site for repeated blood testing

Complications and Limitations¹

1. Mechanical injury: arterial, venous, nerve injury, and cardiac tamponade
2. Respiratory compromise: airway compression by a hematoma and pneumothorax
3. Arrhythmias
4. Thromboembolic events: venous or arterial thrombosis, pulmonary embolism, and catheter/guidewire embolism
5. Infectious: infection at site, catheter infection, blood stream infection, and endocarditis
6. Misinterpretation of data

Waveform Components¹

1. a wave: end diastole; atrial contraction
2. c wave: early systole; isovolumic ventricular contraction, tricuspid motion toward the right atrium
3. v wave: late systole; systolic filling of the atrium

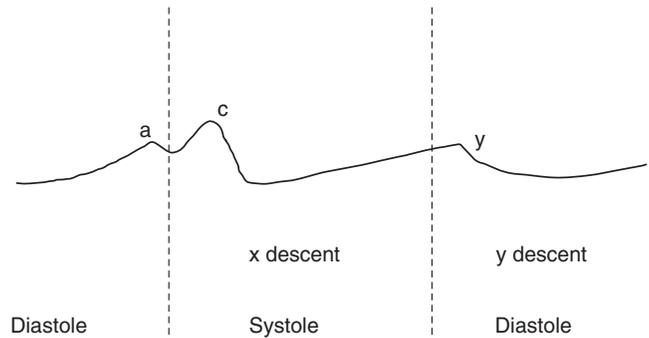


Figure 6.4. Normal CVP waveform showing cardiac cycle. Drawing credit: James Lovich.

4. h wave: mid-to-late diastole; diastolic plateau
5. x descent: midsystole; atrial relaxation, descent of the base, systolic collapse
6. y descent: early diastole; early ventricular filling, diastolic collapse

Waveform Abnormalities by Diagnosis^{1,2}

1. Atrial fibrillation/flutter: loss of the a wave and prominent c wave
2. Atrioventricular dissociation: cannon a wave
3. Tricuspid regurgitation: tall systolic c-v wave and loss of x descent
4. Tricuspid stenosis: tall a wave and attenuation of y descent
5. Right ventricular ischemia and pericardial constriction: tall a and v waves, steep x and y descent, and M or W configuration (fusion of a prominent a and v wave)
6. Cardiac tamponade: dominant x descent and attenuated y descent
7. Respiratory variation during spontaneous or positive-pressure ventilation: measure pressures at end-expiration

Waveform Abnormalities by Waveform^{1,2}

1. Loss of the a wave: atrial fibrillation/flutter
2. Cannon a waves: right ventricular hypertrophy, tricuspid/pulmonary stenosis, acute or chronic lung disease associated with pulmonary hypertension, and junctional or nodal rhythm
3. Large v waves: tricuspid regurgitation and right ventricular papillary muscle ischemia

6. Indications, Complications, and Waveforms

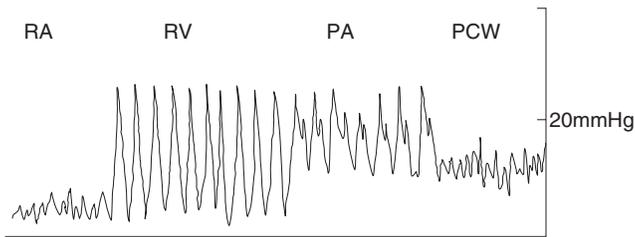


Figure 6.5. Pressure tracing during the insertion of a PAC.
Drawing credit: James Lovich.

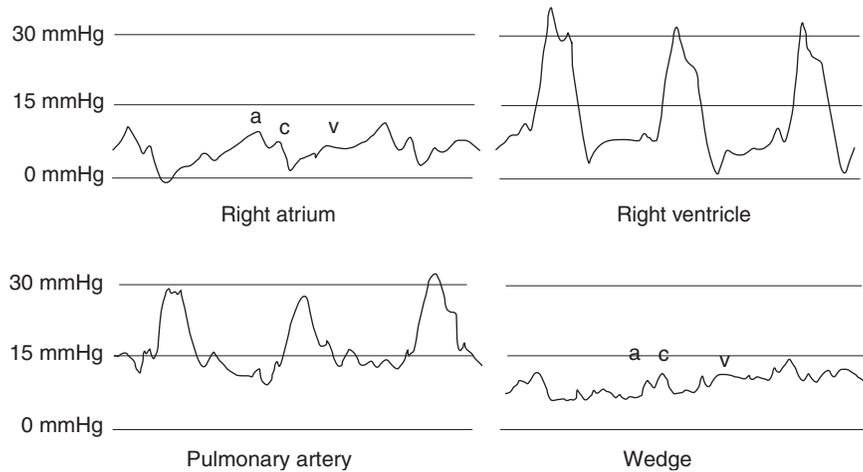


Figure 6.6. PAC pressure tracings. Drawing credit: James Lovich.

PULMONARY ARTERY CATHETER (PAC)^{1,2}

SAMPLE CASE

A 77-year-old female had a PAC placed for a routine triple vessel coronary artery bypass grafting (CABG). The patient was transported to the intensive care unit (ICU). Upon arrival, you note significant hemoptysis. The patient was an easy intubation.

CLINICAL ISSUES

PAC Measurements²

1. Cardiac output (CO)/cardiac index (CI)
2. Pulmonary artery pressure (PAP)
3. Central venous pressure (CVP)
4. Calculation of oxygen delivery
5. Assessment of cardiac work
6. Mixed venous oxygen saturation (MVO₂)
7. Pulmonary capillary wedge pressure (PCWP)
8. Systemic vascular resistance (SVR)

Possible Indications¹

1. Cardiac
 - a. Congestive heart failure (CHF)

- b. Low ejection fraction (EF)
- c. Left sided, valvular heart disease
- d. CABG
- e. Aortic cross clamp
2. Pulmonary
 - a. Chronic obstructive pulmonary disease (COPD)
 - b. Adult respiratory distress syndrome (ARDS)
3. Complex fluid management
 - a. Shock
 - b. Burns
 - c. Acute renal failure
4. High risk obstetrical care
 - a. Eclampsia
 - b. Placental abruption
5. Neurological
 - a. Sitting craniotomy
 - b. Venous air embolus

Complications^{1,2}

1. Venous access and PAC placement
 - a. Arterial puncture
 - b. Arrhythmias
 - i. Right bundle branch block
 - ii. Complete heart block
 - iii. Ventricular fibrillation/tachycardia
 - c. Postoperative neuropathy

6. Indications, Complications, and Waveforms

- d. Pneumothorax
- e. Air embolism
- 2. Catheter residence
 - a. Catheter knots
 - b. Infection
 - c. Thrombophlebitis
 - d. Thromboembolism
 - e. Pulmonary infarct
 - f. Endocarditis
 - g. Valvular injury
 - h. Pulmonary artery rupture
 - i. Pulmonary artery pseudoaneurysm
- 3. Death
- 4. Misinterpretation of data

Waveform Abnormalities by Diagnosis¹

- 1. Mitral regurgitation: tall regurgitant c-v wave and obliteration of the x descent
- 2. Ventricular septal defect (VSD): tall anterograde v wave
- 3. Mitral stenosis: tall a wave and attenuation of y descent
- 4. Myocardial ischemia: tall a and v waves

Waveform Abnormalities by Waveform¹

- 1. Large a waves: mitral stenosis, atrial myxoma, myocardial ischemia, and acute CHF
- 2. Large v waves: mitral regurgitation (papillary muscle dysfunction), CHF, myocardial ischemia, and ventricular septal defect (VSD)



KO TREATMENT PLAN

Pulmonary Artery Rupture^{1,4}

- 1. Most life-threatening complication of the PAC
- 2. Uncommon: 0.02–0.2% of catheterized patients
- 3. Often avoidable with meticulous attention to insertion and monitoring techniques
 - a. Avoid unnecessary catheter manipulation, excessive insertion distance, persistent or prolonged balloon inflation, and balloon inflation with liquid rather than air.
- 4. Mortality rate is about 50%.
- 5. Occurs more commonly in females than males.
- 6. Right pulmonary artery is more commonly injured: about 90% of cases.
- 7. More common in patients undergoing cardiopulmonary bypass (CPB)
- 8. Increased risk of occurrence is associated with
 - a. Hypothermia
 - b. Anticoagulation

- c. Advanced age
- d. Pulmonary hypertension
- 9. Mechanism of injury
 - a. Balloon inflation
 - i. Especially the over-wedged, distally placed catheter
 - ii. Chronic erosion
- 10. Hallmark: hemoptysis
- 11. Other presenting signs and symptoms
 - a. Hypotension
 - b. Respiratory compromise
 - c. Hemothorax: visualized on a chest X-ray
- 12. Treatment
 - a. Stabilize the cardio-respiratory condition.
 - b. Maintain adequate gas exchange.
 - i. Endobronchial intubation with a single or double lumen endotracheal tube for selective unilateral lung ventilation and protection of the unaffected lung
 - ii. Positive end-expiratory pressure (PEEP) applied to the affected lung
 - iii. Bronchoscopy can be used to localize the site of bleeding.
 - c. Stop the bleeding.
 - i. Reverse anticoagulation, unless on cardiopulmonary bypass.
 - ii. Bronchial blocker may be placed into the bronchus to tamponade the bleeding and prevent contamination of the unaffected lung.
 - d. It is controversial as to whether or not to pull the PAC.
 - i. Some anesthesiologists suggest leaving it in place to monitor the pulmonary artery pressure and guide antihypertensive therapy targeted at reducing the bleed.
 - e. Early pulmonary angiography can be used for diagnosis and treatment by embolization.
 - f. The patient may require definitive surgical therapy, including oversewing the involved pulmonary artery or resecting the involved lung lobe or segment.

BIBLIOGRAPHY

- 1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1272–327.
- 2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 674–80.
- 3. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 165–6.
- 4. Urschel JD, Myerowitz PD. Catheter-induced pulmonary artery rupture in the setting of cardiopulmonary bypass. *Annals of Thoracic Surgery*, 1993 (56): 586–9.

7. False Measurements in Thermodilution Cardiac Output Readings^{1,2,3,4,5}

Jessica A. Lovich-Sapola, MD

CLINICAL ISSUES

Thermodilution Cardiac Output (CO) Monitoring

1. This is the clinical standard for measuring cardiac output.
2. Cardiac output is the total blood flow generated by the heart.
3. Normal cardiac output in an adult ranges from 4 to 6.5 L/min.
4. The thermodilution method uses a thermal indicator.
5. The temperature of the patient's blood is measured continuously by a thermistor at the tip of the pulmonary artery catheter (PAC).
6. The injectate temperature is measured by a second thermistor at the injectate port.
7. To perform an accurate CO, a fixed volume of iced or room-temperature fluid is injected as a bolus into the proximal CVP lumen of the PAC. The resulting change in pulmonary artery blood temperature is recorded by the thermistor at the catheter tip.
8. The thermodilution technique measures right ventricular output and pulmonary artery blood flow.

Assumptions and Errors of Thermodilution Cardiac Output Measurements

1. Assumptions
 - a. Flow is constant.
 - b. Blood volume is constant (no rapid IV fluid administration).
 - c. Pulmonary artery (PA) temperature is constant.
 - d. There is no significant venous pooling.
 - e. Volume of injectate must be correct.
 - f. Temperature of injectate must be accurate.
 - g. There are no intracardiac shunts.
 - h. The tip of the PA catheter must be in the PA and not covered by fibrin or clot.
 - i. There is insignificant tricuspid or pulmonic valve regurgitation.
2. Errors

Although thermodilution is the most commonly used CO technique, precision is poor compared with other methods.

A 10% difference may result depending on when the injection occurs during the respiratory cycle, likely due to changes in pulmonary blood flow during respiration. The readings or traces are nonsensical if the PA catheter is wedged or if there is a thrombus covering the tip. The computer algorithm bases the CO calculation on a specific injectate volume at a specific temperature, and assumes that there is no communication between the pulmonary and systemic circulations.

False Increase in Thermodilution Cardiac Output Readings

1. Small injectate volume
2. Increased temperature of injectate
3. Thrombus on thermistor
4. The patient is in a very low cardiac output state.

False Decrease in Thermodilution Cardiac Output Readings

1. Large injectate volume
2. Decreased temperature of injectate
3. Inflation cycle of lower limb sequential compression devices (SCDs)
4. Either rapid or continuous infusion of intravenous fluid through the pulmonary artery catheter secondary to the cooling effect on the blood

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1327-31.
2. Nishikawa T, Dohi S. Errors in the measurement of the cardiac output by thermodilution. *Canadian J Anesthesia* 1993; 40:142-53.
3. van Grondelle A, Ditchey RV, Groves BM, et al. Thermodilution method overestimates low cardiac output in humans. *Am J Physiol Heart Circ Physiol* 1983;245: H690-2.
4. Killu K, Oropello JM, Manasia Ar, et al. Effects of lower limb compression devices on the thermodilution cardiac output measurement. *Crit Care Med* 2007;35(5):1307-11.
5. Hensley FA, Martin DE, Gravlee GP. *A Practical Approach to Cardiac Anesthesia*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2007, chapter 3, Monitoring the cardiac surgical patient.

8 Intra-Operative Transesophageal Echocardiography^{1,2,3,4}

Donald M. Voltz, MD

CLINICAL ISSUES

The role for anesthesiologist performance of intra-operative transesophageal echocardiography (IOTEE) has been well established for the assessment and management of patients undergoing valvular and coronary surgery. The roles of IOTEE in non-cardiac surgery cases or for the unstable intensive care unit (ICU) patient is not yet completely clear. What follows is a discussion about the indications, contraindications, and utility of IOTEE in the management of patients for non-cardiac surgery.

Utility of TEE in Critically Ill Patients or Those Undergoing Non-Cardiac Surgery

1. Numerous modalities have been utilized throughout the years to assess, monitor, and optimize the cardiovascular system in patients who are critically ill or undergoing non-cardiac surgery.
 - a. These techniques include
 - i. Invasive blood pressure monitoring
 - ii. Pulmonary artery pressure monitoring
 - iii. Minimally invasive cardiac output monitoring
2. Although these technologies have aided anesthesiologists in optimization and diagnosis, they are limited in the ability to provide assessment of cardiac chamber size, function, and valvular integrity.
3. The deficiencies of these indirect, continuous monitoring modalities can be overcome with IOTEE.
4. The ASA has addressed these issues in their guidelines for the use of IOTEE.¹
5. Evidence-based information has demonstrated an improvement in outcomes for patients undergoing both cardiac and non-cardiac surgery with the use of IOTEE.⁴

Indications for IOTEE in Critically Ill Patients or Those Undergoing Non-Cardiac Surgery

1. In multiple studies, for about 2–10% of patients undergoing cardiac surgery, information obtained from the IOTEE altered the course of surgical care.⁴
 - a. Due to this fact, the use of IOTEE is becoming routine in cardiac surgery cases and is being done either by anesthesiologists trained in its use or by a cardiologist.

2. The use of IOTEE in other non-cardiac surgery cases is not yet clear.
 - a. In patients where there is concern about the cardiovascular system and its optimization, IOTEE can provide additional information to assist with clinical management.
 - i. Better assessment of ventricular filling and function than a pulmonary artery catheter
 - ii. Considered by some to be an alternative monitoring tool in peri-operative optimization of the heart
3. The following are some situations where one might consider using IOTEE over a pulmonary artery catheter.
 - a. Anticipated hemodynamic swings that may not be well tolerated by the patient
 - b. Patients who are persistently hypotensive despite interventions that should have corrected the hypotension³
 - c. Patients undergoing procedures where there is a significant risk for venous air emboli
 - d. Any case where a cardiac abnormality is suspected and where knowledge of the abnormality would alter clinical care
4. The utility of IOTEE with cardiac surgery patients has stimulated many to investigate its utility in the critically ill patient.
 - a. TEE can provide additional information to guide physicians in the management of these patients.
 - b. Patients that may benefit from the information obtained with a TEE:
 - i. Patients with hemodynamic instability despite therapies to correct it
 - ii. Critically injured patient with a suspected aortic injury
 - iii. Patients suspected to have endocarditis or paradoxical embolic disease
 - iv. Patients in atrial fibrillation with a concern for a left atrial thrombus
 - c. The risks are low and the information obtained about cardiac structure and function far exceed those obtained with alternative hemodynamic monitors such as a pulmonary artery catheter.
 - d. The main limitation to widespread use of TEE in the critical care setting is the number of physicians with experience to perform and interpret the results.

Contraindications for IOTEE in Critically Ill Patients or Those Undergoing Non-Cardiac Surgery

1. Relative contraindications
 - a. Paraesophageal hernia
 - b. Cervical spine instability
 - c. Atlantooccipital disease or instability
 - d. Dysphagia
 - e. History of esophageal or upper gastrointestinal bleeding
 - f. Esophageal scarring from radiation
 - g. Esophageal surgery
 - h. Hiatal hernia
2. Absolute contraindications
 - a. Esophageal perforation
 - b. Esophageal strictures
 - c. Esophageal varicies
 - d. Esophageal diverticula

Complications That Can Occur with TEE Probe Placement

1. The overall complication rate for IOTEE is 0.1–0.3%.²
2. Pharyngeal trauma
3. Laryngeal trauma
4. Aspiration
5. Dental injuries
6. Esophageal injury
7. Bleeding
8. Hemodynamic swings
9. Arrhythmias

Modes of IOTEE and the Information They Provide

1. 2D
 - a. By utilizing numerous crystals arranged in linear or phased-arrays, numerous views can be obtained and synthesized into a two-dimensional representation of the heart.
 - b. With 2D echocardiography, about 30 images per second can be collected.
 - c. 2D allows for the recognition of normal and abnormal cardiac structures as well as the ability to assess function of the myocardium and valvular apparatus in real time.
 - d. Adjustment of the probe direction or the angle of the crystals within the probe results in the collection of numerous views of the heart.
 - e. Newer technologies are increasing the number of images obtained and software advancements are allowing for three-dimensional representation of the cardiac system.
2. M-mode
 - a. This was the first technique developed for TEE.
 - b. It consists of a single image plane through tissue.
 - c. The information returned to the transducer is recorded in a linear moving strip and can produce up to 1,000 images per second.

d. This mode of TEE continues to be used for rapidly moving structures within the heart such as valve leaflets or the thickening of the myocardium.

e. However, since it is a single narrow plane, it is difficult to spatially relate the information to the overall function of the heart.

3. Color-flow Doppler

a. Color-flow Doppler is derived from pulse-wave Doppler where many areas of blood flow velocities are sampled.

b. These velocities are then depicted as color depending on their direction of flow in relation to the transducer.

i. Red: toward the probe

ii. Blue: away from the probe

c. This technique is useful to determine color jets that occur during blood flow through stenotic valves or regurgitation of flow when valves are incompetent.

4. Pulse-wave (PW) Doppler

a. Using the principles of the Doppler shift, we can obtain information about the blood flow velocity within the heart.

b. The working end of a TEE probe consists of either one or more piezoelectric crystals that when stimulated transmit waves at a certain wavelength and frequency.

c. When the wave contacts something in its path, it is reflected back to the crystal allowing for the generation of an image (such as in M-mode or 2D echo).

d. When the wave contacts something that is moving, such as a red blood cell, the frequency of the wave becomes changed and the returning wave frequency is higher or lower than the original wave sent out.

i. This is the Doppler shift and can be used to assess the flow velocity of blood throughout the cardiac system.

e. In PW Doppler, a pulse of waves is sent out of the crystal and their returning frequencies are collected and measured.

f. The use of small pulses of waves allows the user to focus the information on a small area and therefore to determine an instantaneous blood flow velocity as a specific location within the heart.

g. The utility of this technique depends on the direction of the ultrasound beam within the cardiac chambers.

i. When the angle one is attempting to measure with PW Doppler exceeds 20 degrees from the direction of the probe, the Doppler shift measurement is attenuated to produce meaningless results.

5. Continuous-wave (CW) Doppler

a. Unlike PW Doppler, CW Doppler utilizes two separate crystals to obtain information about blood flow velocities within the heart.

i. One crystal continually emits waves.

ii. The other crystal is used to collect frequency information about the returning waves.

b. One problem with CW is that the collecting crystal cannot determine which of the transmitted waves it is measuring and therefore can not define the location within the path where that wave's frequency was shifted.

8 Intra-Operative Transesophageal Echocardiography

- c. Although one loses location information, the returning frequency shift can be used to obtain a maximal velocity along the path where the waves were sent.
- d. This information, along with the Bernoulli equation, is useful to assess peak pressure/flow gradients across stenotic valves.
- e. CW Doppler can measure higher blood flow velocities than PW; however, PW Doppler is able to define a more precise location for the velocity.

Cases or Pathophysiology in Which IOTEE Is Useful

1. Severe ischemic cardiomyopathy
2. Suspected pulmonary emboli with hemodynamic embarrassment
3. Intra-thoracic aortic injury, suspected dissection, or aneurysm
4. Patients with symptomatic or suspected valvular disease
5. Major vascular surgery
6. Liver transplantation
7. Major burn cases
8. Cardiac surgery cases: coronary revascularization and valvular surgery
9. Monitoring and management of cases with a high probability for venous air emboli
10. Assessment of patients with severe cardiopulmonary disease
11. Cardiac assessment in patients where central venous access is difficult or impossible

Minimal IOTEE Views That Should Be Obtained by an Anesthesiologist

1. Mid-esophageal basal short-axes view of the aortic valve
 - a. This position is obtained with the TEE probe placed 28–32 cm from the upper incisors.
 - b. With the probe in this position, the direction of the ultrasound beam will cut the heart in a coronal plane.
 - c. The aortic valve and ascending aorta exit the heart at a 25–35 degree angle.
 - i. Rotation of the transducer by this amount will bring the aortic valve into a cross-sectional view allowing for assessment of the valve leaflets and opening of the valve.
 - ii. Color-flow Doppler can reveal both stenotic and regurgitant abnormalities of the aortic valve.

2. Mid-esophageal four chamber view
 - a. Rotation of the transducer back to zero degrees and slight advancement of the TEE probe into the esophagus will lead to a mid-esophageal four chamber view often referred to as “home-base.”
 - i. It is in this view where one can assess the four cardiac chambers simultaneously.
 - ii. Assessment of chamber size and function of all four cardiac chambers can occur in this view.
 - (1) It does not provide a complete evaluation of myocardial function.
 - iii. Color flow can be used to assess for tricuspid and mitral valve abnormalities.
3. Transgastric short-axis view
 - a. Further advancement of the TEE probe into the proximal stomach and flexing of the probe reveals a cross-sectional view across the left and right ventricle at the level of the papillary muscles.
 - b. This view provides assessment of ventricular filling status as well as myocardial function.
 - c. The transgastric short-axis view shows myocardium from all of the coronary blood distributions.
 - i. The left anterior descending artery supplies the anterior myocardial wall and part of the septum.
 - ii. The circumflex artery supplies the anterior-lateral and lateral wall of the left ventricle.
 - iii. The right coronary artery supplies the inferior wall as well as part of the intraventricular septum.
 - d. It is this short axis view of the myocardium, at the level of the papillary muscles, that is the best for detection of intra-operative myocardial ischemia.
 - e. The size of the ventricular chambers can also be well assessed in this view to make a determination of the pre-load on the myocardium.

BIBLIOGRAPHY

1. Youngberg JA, Roizen MF, Lake CL, et al. Cardiac, Vascular, and Thoracic Anesthesia. *Churchill Livingstone*, 2000, pp 272–306.
2. Rafferty, T, et al. Quality assurance for intraoperative transesophageal echocardiographic monitoring: A report of 846 procedures. *Anesth Analg* 1993;76:228.
3. Reichert, CL, et al. Transesophageal echocardiography in hypotensive patients after cardiac operations. Comparison with hemodynamic parameters. *J Thorac Cardiovasc Surg* 1992; 104:321.
4. Eltzschig HK, et al. Impact of intraoperative transesophageal echocardiography on surgical decisions in 12,566 patients undergoing cardiac surgery. *Ann Thorac Surg* 2008 Mar; 85(3):845–52.

9. Types of Anesthesia Circuits^{1,2,3}

Karl Wagner, MD

SAMPLE CASE

A two-year-old female presents to the operating room for a repair of her club foot. Do you think that a circle system is appropriate for her?

CLINICAL ISSUES

Breathing Systems

1. Insufflation

- a. Most basic type of oxygen delivery
- b. Blowing of a gas across a patient's face
- c. This utilizes a high fresh gas flow and does not require direct contact between the patient and oxygen source.
- d. The FiO_2 delivered to the patient is dependent on the respiratory rate, tidal volume, and fresh gas flow.
- e. Advantages
 - i. Useful for children who are afraid of having a mask on their face during an inhalational induction
 - ii. Useful for patients who are covered with a drape during an operative intervention but are breathing spontaneously without an endotracheal tube or a laryngeal mask airway (LMA), to prevent carbon dioxide accumulation
 - iii. A high fresh gas flow can prevent hypercarbia in the spontaneously breathing patient.
 - iv. No rebreathing
 - v. Can be used to maintain arterial oxygenation during short periods of apnea
- f. Disadvantages
 - i. Not able to deliver anesthetic gases
 - ii. Not able to scavenge with this system
 - iii. Not able to control ventilation
 - iv. The inspired gas contains unpredictable amounts of atmospheric gas.
 - v. No conservation of exhaled heat or humidity

2. Simple face mask, face tent, venturi mask, and non-rebreather face mask

- a. Simple circuits that allow delivery of oxygen from the wall supply to the patient
- b. They may or may not have valves, and there can be variable amounts of rebreathing.
- c. An Ambu bag, such as that commonly used for resuscitation in the wards, differs in that it has a one-way valve to prevent rebreathing and delivers nearly to 100% oxygen.

d. Advantages

- i. They allow a greater increase in FiO_2 when comparing the face mask to insufflation.
- ii. They are lightweight and portable.

e. Disadvantages

- i. Not capable of scavenging waste
- ii. Except for the ambu bag, all rely on spontaneous ventilation.

3. Mapleson circuits

a. Semi-open system

b. Allows delivery of oxygen and with some modifications can be used to deliver anesthetic gases

c. The amount of rebreathing with the different types is dependent on

- i. Fresh gas flow
- ii. Minute ventilation
- iii. Mode of ventilation: spontaneously breathing or controlled ventilation
- iv. Tidal volume
- v. Respiratory rate
- vi. I:E ratio
- vii. Peak inspiratory flow rate
- viii. Volume of the reservoir tube
- ix. Volume of the breathing bag

d. They are considered to be valveless, because the direction of flow is not limited by valves, although there is a pop-off pressure valve.

e. Advantages

- i. Simple to use
- ii. Decreased resistance to respiratory flow since they do not have valves or an absorber
- iii. Lightweight
- iv. Not complicated
- v. Easily used while transporting patients
- vi. Inexpensive
- vii. Easy to sterilize
- viii. Ability to change the depth of anesthesia rapidly
- ix. Lack of rebreathing of exhaled gases, provided the fresh gas flow is adequate

f. Disadvantages

- i. Do not conserve heat, humidity, or anesthetic gases
- ii. They require high fresh gas flows
- iii. They have no CO_2 absorbers
- iv. Limited ability to scavenge waste gases

9. Types of Anesthesia Circuits

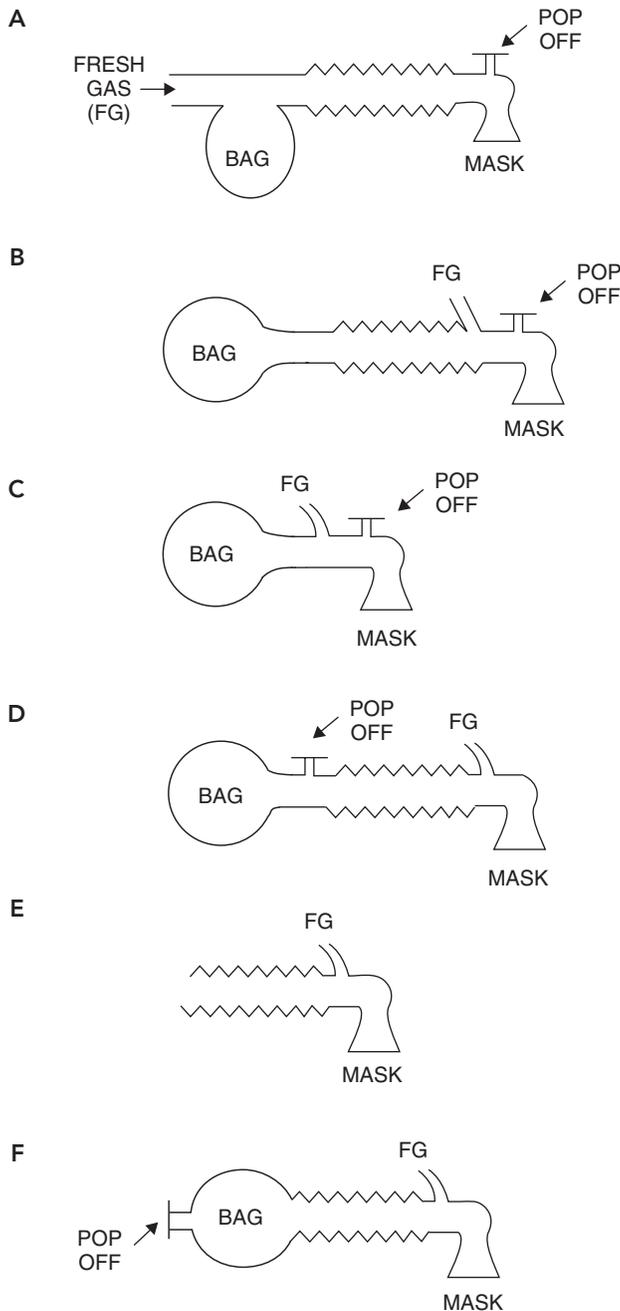


Figure 9.1. Mapleson-type circuit. Drawing credit: K. Wagner.

g. Modifications of the Mapleson circuit

- i. For your exam you may be asked to draw the different circuits, so be prepared to do so.
- ii. The Mapleson A circuit is best used for a spontaneously ventilating patient.
- iii. The Mapleson D is best for controlled ventilation.

4. Circle system

- a. The circle system contains
 - i. Inspiratory limb
 - ii. Expiratory limb

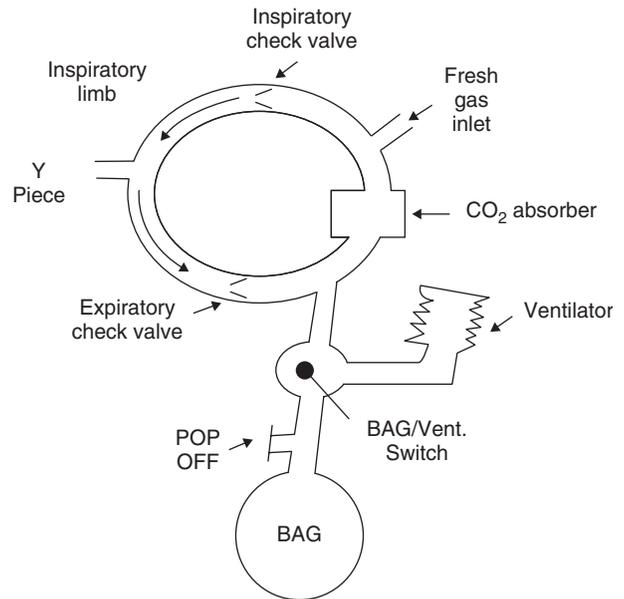


Figure 9.2. Circle system. Drawing credit: K. Wagner.

- iii. One-way valves to ensure flow direction
 - iv. CO₂ absorber
 - v. Gas reservoir bag
 - vi. Pop-off valve on the expiratory limb
- b. It is considered a semi-closed system (instead of a completely closed system) because there is not complete rebreathing.
- i. Some of the tidal volume during exhalation goes to the scavenging system.
 - ii. The amount of volume that is sent through the scavenger is a function of the total fresh gas flow rate, pressure in the system, and minute ventilation.
 - iii. Once a threshold pressure is reached, the gas volume is released to the scavenging system via a valve.
- c. Advantages
- i. Can be used to conserve heat and humidity
 - ii. Uses low flows of fresh gas thereby conserving volatile anesthetics
 - iii. There is a CO₂ absorber.
 - (1) Prevents rebreathing of CO₂
 - (2) Allows partial rebreathing of other anesthetic gases
 - iv. Scavenging can easily be done to prevent contamination of the operating room.
 - (1) Because there is a scavenger built into the system, the exhaled gas can either be rebreathed or go out to the scavenger.
 - v. Good control of anesthetic depth
 - vi. They come in different sizes and tubing compliances.
 - (1) Remember that compliance is the change in volume per unit of pressure.

9. Types of Anesthesia Circuits

vii. Good for pediatric patients

(1) The pediatric population benefits from less compliant tubing so that the tidal volume delivered from the machine is not utilized in expanding the tubing but is being delivered to the patient.

(2) Heat conservation is critical in small children as is volume control against dehydration; these are all accomplished with the circle system.

d. Disadvantages

i. These circuits are more complicated than the Mapleson type and are not as portable.

ii. There is an increase in resistance with the tubing and valves, which can increase the work of breathing in a spontaneously breathing patient.



KO TREATMENT PLAN

Assuming the patient requires general anesthesia, she would benefit from the conservation of heat and humidity provided by the circle system that is not existent with the Mapleson-type circuits. We would use pediatric tubing that is smaller and less compliant than the adult tubing.

BIBLIOGRAPHY

1. Morgan, Mikhail. *Murray: Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006, pp 31-42.
2. Barash, Cullen. *Stoelting: Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 578-80.
3. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 295-6.

10. Sodium^{1,2,3,4}

Jennifer Eisman, MD

HYPERNATREMIA

SAMPLE CASE

A 42-year-old male from a group home, who is baseline non-verbal with mental retardation, is brought into the hospital for confusion, muscle weakness, lethargy, poor oral intake, and abdominal pain. The patient is found to have an abdominal hernia. His laboratory results reveal a sodium level of 157 mEq/L. The surgeon is concerned that the hernia is incarcerated and wants to operate immediately. What are your concerns? Would you like to order any more labs or tests? How would you treat the hypernatremia? Would you delay this surgery if it was an elective hernia repair? If this is emergent, what is your anesthetic plan?

CLINICAL ISSUES

Definition of Hypernatremia

1. Sodium (Na⁺) concentration of greater than 145 mEq/L
2. Produces a state of hyperosmolality
3. Maintenance of osmotic equilibrium in hypernatremia results in intracellular fluid volume contraction.
4. Mechanism
 - a. Primary Na⁺ gain
 - b. H₂O deficit: majority of cases

Appropriate Response to Hypernatremia in an Awake, Otherwise Normal Patient

1. Increased water (H₂O) intake stimulated by thirst
2. Excretion of minimal volume, maximally concentrated urine reflecting anti-diuretic hormone (ADH) secretion in response to an osmotic stimulus

Causes of Hypernatremia

1. Inadequate intake of fluids
 - a. Lack of thirst: thirst is regulated by the hypothalamus.
 - i. Primary hypodipsia: uncommon
 - (1) Usually as a result of damage to the hypothalamic osmoreceptors

(2) Associated with abnormal osmotic regulation of ADH secretion

(3) Causes of damage to hypothalamic osmoreceptors include

- (a) Granulomatous disease
- (b) Vascular occlusion
- (c) Tumors

b. Access to H₂O is limited or due to poor intake.

- i. Infants
- ii. Elderly: there is a predisposition for hypernatremia in the elderly due to poor urinary concentrating ability and hypodipsia.
- iii. Patients who are physically and mentally handicapped
- iv. Those with impaired mental status: coma
- v. Intubated patient/intensive care unit (ICU) setting

2. Renal losses of hypotonic fluid

a. Drug induced: diuretics produce iso-osmotic solute diuresis.

b. Osmotic diuresis: most common cause of H₂O loss

- i. Glucosuria
- ii. Mannitol
- iii. Increased endogenous production of urea: high protein diet

c. Diabetes insipidus: central and nephrogenic

i. Central diabetes insipidus: impaired ADH secretion secondary to the destruction of the neurohypophysis

- (1) Idiopathic
- (2) Post-traumatic: basal skull fracture, severe head injury
- (3) Post-surgical: pituitary surgery
- (4) Space-occupying sellar lesions: cysts, tumors, tuberculosis (TB), and sarcoidosis
- (5) Vascular: infarction, aneurysms, Sheehan's syndrome
- (6) Inflammatory lesions: encephalitis, meningitis, granulomatous disease, Guillain-Barré, Lyme disease

ii. Nephrogenic diabetes insipidus: end-organ kidney resistance to ADH

- (1) Inherited
 - (a) Congenital nephrogenic diabetes insipidus
 - (b) X-linked recessive or autosomal mutation

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- (2) Acquired: sporadic nephrogenic diabetes insipidus
 - (a) Renal disease: medullary sponge kidney, amyloid, myeloma, Sjorgen's syndrome
 - (b) Drugs: lithium, foscarnet, glyburide, demeclocycline, methoxyflurane, amphotericin B
 - (c) Electrolytes: hypercalcemia, hypokalemia
 - (d) ADH released by the placenta
 - (i) Resulting in second and third trimester nephrogenic diabetes insipidus as a result of the excessive release of vasopressinase from the placenta
- d. Intrinsic renal disease
- e. Post-obstructive diuresis
- 3. Extrarenal: nonrenal loss of H₂O
 - a. Evaporation of water from the skin or respiratory tract
 - b. Insensible loss of water increased with
 - i. Fever
 - ii. Heat exposure
 - iii. Exercise
 - iv. Severe burns
 - v. Controlled mechanical ventilation and hyper-ventilation
 - vi. Sweating: Na⁺ concentration of sweat decreases with profuse perspiration therefore increasing solute free H₂O loss.
 - c. Water loss from gastrointestinal tract
 - i. Osmotic diarrhea
 - (1) Lactulose
 - (2) Sorbitol
 - (3) Malabsorption of carbohydrates
 - ii. Viral gastroenteritis: results in H₂O loss greater than Na⁺ loss
 - iii. Vomiting
 - iv. Nasogastric drainage
 - v. Enterocutaneous fistula
- 4. Primary Na⁺ gain: urine sodium greater than 800 mEq/L
 - a. Administration of hypertonic NaCl or NaHCO₃:
 - i. 3% NaCl contains 513 mEq sodium per liter.
 - ii. 7.5% sodium bicarbonate solution routinely used in a 50 ml ampule contains 44.5 mEq sodium.
 - b. Hypertonic tube feeds: total parenteral nutrition (TPN), tube feeds
 - c. Ingestion of NaCl or seawater
 - i. A tablespoon of table salt is equal to 350 mEq of NaCl and can raise serum sodium by 8 mEq/L in a 70 kg adult.
 - d. Hypertonic enemas or dialysis

Clinical Features of Hypernatremia

- 1. The severity of the clinical manifestations are determined by the acuity.
 - a. Chronic hypernatremia is less symptomatic.
- 2. The major symptoms are neurologic and are related to the osmotic decrease in brain volume.

- a. Altered mental status: lethargy, confusion, irritability
- b. Advanced neurologic deficit: coma, seizures, intracerebral bleeding due to the rupture of cerebral veins as a result of brain shrinkage
- 3. Other symptoms
 - a. Expanded intravascular volume: signs of hypervolemic hypernatremia
 - i. Pleural effusion
 - ii. Ascites
 - iii. Peripheral edema
 - iv. Heart failure
 - b. Complaints of thirst or polyuria
 - c. Nausea and vomiting
 - d. Neuromuscular irritability: myoclonus, muscular tremor/rigidity, hyperactive reflexes, muscle weakness

Diagnosis of Hypernatremia

- 1. History and physical examination
 - a. List of current medications
 - b. Mental and neurologic exam
 - c. Determine which of the three categories of volume status in the hypernatremic state the patient has: hypovolemic, hypervolemic, or isovolemic.
 - d. Establish water intake, access to water, and salt intake.
- 2. Look for signs and symptoms including thirst, diaphoresis, diarrhea, vomiting, polyuria, and features of extracellular fluid volume contraction.
- 3. Measure urine osmolality, sodium concentration, and the integrity of the ADH-renal system.
 - a. The normal response of a patient with a plasma osmolality above 295 mosmol/kg (represents a plasma Na⁺ above 145–147 mEq/L) is to secrete ADH to concentrate the urine and to activate the thirst mechanism.
 - i. Therefore with intact ADH secretion and renal function, urine osmolality (U_{osm}) will be 700–800 mosmol/kg and no further increase in urine osmolality is seen with supplemental ADH administration.
 - ii. Causes of hypernatremia with U_{osm} 700–800 mosmol/L
 - (1) Unreplaced insensible loss: urine sodium concentration should be less than 25 mEq/L.
 - (2) Gastrointestinal losses: urine sodium concentration should be less than 25 mEq/L.
 - (3) Sodium overload: urine sodium concentration above 100 mEq/L
 - (4) Defect in thirst: rare
 - b. Urine osmolality (U_{osm} less than 300 mOsm/L) is lower than plasma osmolality: consider diabetes insipidus.
 - c. Administer ADH to distinguish between nephrogenic and central diabetes insipidus: give 10 mcg nasal synthetic desmopressin (DDAVP) or 5 units vasopressin subcutaneously (SC).
 - i. Nephrogenic: little or no response to ADH
 - ii. Central: appropriate increase in U_{osm} by greater than 50%

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- iii. Consultation with nephrologist, endocrinologist, or neurologist to aid in the diagnosis of diabetes insipidus: do not be afraid to ask for help from an expert.
- d. Measure urine solute concentration to confirm osmotic diuresis: glucose and urea.

Treatment of Hypernatremia

1. Determine the volume status of the patient and if applicable stop the ongoing H₂O loss by treating the cause and correcting the H₂O deficit.
 - a. Hypernatremia with hypervolemia: use of diuretics and hemodialysis may be necessary to lessen symptoms of volume overload.
 - b. Free H₂O deficit (in liters of H₂O) = $\{(\text{plasma Na}^+ / 140) - 1\} \times \text{kg} \times 0.6$ in males
 - i. 0.5 should be used for the calculation in females.
 - (1) Goal should be a Na⁺ less than or equal to 145 mEq/L.
 - (2) Replace half of the free water deficit in the first 24 hours and the remainder over the following 2–3 days.
 - (3) Use 5% dextrose in water or 0.45% NaCl to correct the deficit.
 - (4) Monitor serum sodium every 1–2 hours to ensure a gradual correction.
2. The rate of Na⁺ correction should not exceed 0.5 mEq/L per hour or 10–12 mEq/L per day.
3. If the rate of correction is too rapid, it may produce cerebral edema, seizures, and irreversible neurologic damage or death.

Treatment of Central Diabetes Insipidus

1. Desmopressin (DDAVP)
 - a. Intravenous (IV) dosing: 2–4 mcg, 1–2 times per day
 - b. Nasal spray: 10–40 mcg 1–2 times per day
 - i. Titrate to urine output, serum sodium, and osmolality
2. Low Na⁺ diet
3. Low-dose thiazide diuretic
4. Carbamazepine: enhances vasopressin secretion
5. Nonsteroidal anti-inflammatory drugs (NSAIDs)
 - a. Impair renal prostaglandin synthesis
 - b. Potentiate ADH action resulting in increased urine osmolality and decreased urine volume.

Treatment of Nephrogenic Diabetes Insipidus

1. Treat the underlying cause.
 - a. Example: eliminate the offending drug.
2. Treat symptomatic polyuria.
 - a. Low Na⁺ diet
 - b. Thiazide diuretic: reduces urine flow by inhibiting distal tubule sodium reabsorption
 - c. Vasopressin and analogs have no role in treatment of nephrogenic diabetes insipidus.



KO TREATMENT PLAN

Pre-operative

1. The patient in the sample case gives you an intuitive reason for the hypernatremia: poor access to water, poor oral intake due to mental retardation, and perhaps a reliance on caregivers for oral intake. There is a good chance that this patient has hypovolemic hypernatremia.
2. Verify the cause of the hypernatremia by obtaining a urine sodium and urine osmolality.
3. Treat the hypernatremia acutely.
 - a. Restore blood pressure by giving fluid replacement with 0.9% NaCl.
 - b. Once the patient's blood pressure is stabilized, address the water deficit.
4. If this is an elective case, postpone the case until the serum sodium approaches normal levels.
 - a. Miller recommends patients undergoing surgery to have Na⁺ less than 150 mEq/L prior to anesthesia.
 - b. The goal of treatment is Na⁺ less than 145 mEq/L.
5. Treat the patient's hypernatremia with fluid replacement and frequent serum sodium evaluation.

Intra-operative

1. If this is an emergency and the decision has been made to proceed with the case:
 - a. Monitors
 - i. Standard ASA monitoring
 - ii. Pre-induction invasive arterial blood pressure monitoring
 - b. Intravenous (IV) access
 - i. Obtaining central IV access is necessary to measure central venous pressure and aid in volume resuscitation.
 - ii. If there is no need for central venous pressure monitoring, peripheral IV access would be acceptable.
 - c. Regional versus general anesthesia: due to this patient's mental retardation and questionable cooperativeness general anesthesia is indicated for this procedure rather than a regional anesthetic.
 - i. In an otherwise cooperative patient, regional anesthesia would be preferred in an emergent surgery on a hypernatremic patient secondary to the ability to monitor his mental status.
 - d. Induction
 - i. Etomidate is a good choice for induction due to the patient's associated hypovolemia.
 - ii. Consider a rapid sequence induction (RSI) using succinylcholine if the patient's mental status puts him at increased risk for aspiration.
 - (1) Avoid succinylcholine if it is contraindicated.
 - (2) Avoid RSI if the patient has an anticipated difficult airway.

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- e. Maintenance anesthesia
 - i. Inhalational agent
 - ii. Short-acting narcotics like remifentanyl (to ensure a “quick” wake up and lessen residual anesthesia) would be ideal due to this patient’s lethargy and baseline mental status.
- f. Other considerations
 - i. Frequent electrolyte checks

Post-operative

1. Follow-up plan: despite his lethargy, this patient after the procedure can be extubated (assuming he meets extubation criteria) and transferred to the intensive care unit (ICU) for further monitoring of his serum sodium every 1-2 hours, replacement of fluid deficits, and urinary volume output measurement.
2. Investigate how he became so dehydrated and verify his safety in his group home setting.

HYPONATREMIA

SAMPLE CASE

A 32-year-old female presents to the emergency department. She reports being a contestant in a water drinking competition to win a car from a radio station. She complains of nausea, headache, lethargy, and confusion. What are your concerns? How will you diagnose and treat the problem? What would your anesthetic plan be if she requires anesthesia?

CLINICAL ISSUES

Definition of Hyponatremia

1. Na^+ less than 135 mEq/L.
2. Most common electrolyte disorder
3. Hyponatremia is commonly caused by cellular edema with the presence of hypotonicity.

Causes of Hyponatremia Defined by Volume Status

1. Hypovolemic hyponatremia
 - a. Nonrenal
 - i. Gastrointestinal (GI) losses: vomiting, diarrhea, gastrointestinal tube drainage, fistula, bowel obstruction, pancreatitis
 - ii. Integumentary loss: excessive sweating, burns
 - iii. Third spacing: ascites, peritonitis, pancreatitis, burns
 - iv. Cerebral salt wasting syndrome: traumatic brain injury, aneurismal subarachnoid hemorrhage, intracranial surgery
 - (1) Decreased Na^+ also occurs via desalination.
 - (a) When urine tonicity (sum concentration Na^+ and K^+) exceeds that of the administration of IV fluids (including isotonic saline)

- (b) This accounts for some cases of acute postoperative reduction in Na^+ and cerebral salt wasting after neurosurgery.
- b. Renal loss
 - i. Diuretics
 - ii. Osmotic diuresis
 - iii. Hypoaldosteronism
 - iv. Salt-wasting nephropathy
 - v. Post-obstructive diuresis
 - vi. Nonoliguric acute tubular necrosis
 - vii. Acute and chronic renal failure
 2. Euvolemic hyponatremia: normal Na^+ stores and total body excess of free water
 - a. Polydipsia
 - i. Psychogenic compulsive H_2O consumption
 - (1) May overwhelm the normally large renal excretory capacity of 12 L/day
 - ii. Exercise-induced water intoxication
 - iii. Drugs that enhance thirst
 - (1) Phenothiazines: cause dry mouth
 - (2) MDMA (3,4-methylenedioxy-*N*-methylamphetamine): Ecstasy
 - b. Administration hypotonic IV or irrigation fluids
 - c. Infants given too much free water
 - d. Beer potomania: patients with poor dietary protein and electrolytes consume large volumes of beer which may exceed the renal excretory capacity resulting in hyponatremia.
 - e. Syndrome of inappropriate anti-diuretic hormone secretion (SIADH): most common cause of decreased Na^+ due to the non-physiologic release of ADH from posterior pituitary or an ectopic source
 - i. Causes of SIADH
 - (1) Neurologic: head injury, meningitis, subdural hematoma, subarachnoid hemorrhage, neurosurgery
 - (2) Pulmonary disease: pneumonia, tuberculosis, lung abscess, chronic obstructive pulmonary disease (COPD), pneumothorax, HIV infection
 - (3) Malignant tumors: bronchogenic carcinoma, lymphoma, pancreatic cancer, mesothelioma
 - (4) Major surgery: severe post-operative pain and nausea stimulate ADH secretion.
 - (5) Pharmacologic agents: carbamazepine, chlorpromamide, cyclophosphamide, tricyclic anti-depressants, SSRIs
 - ii. Classification of SIADH: four types
 - (1) Ectopic production
 - (2) Normal regulation of ADH release around lower osmolality set point (cachexia, malnutrition)
 - (3) Normal ADH response to hypertonicity with failure to suppress completely at low osmolality (incomplete pituitary stalk section)
 - (4) Normal ADH secretion with increased sensitivity to its actions (rare)
 3. Hypervolemic hyponatremia: total body Na^+ increases but total body water increases to a greater extent.

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- a. Renal: acute/chronic renal failure
 - b. Nonrenal
 - i. Heart failure
 - ii. Hepatic cirrhosis
 - iii. Nephrotic syndrome
 - (1) All three cause a decreased effective circulating arterial volume leading to increased thirst and increased ADH levels.
4. Redistributive hyponatremia
- a. Increased plasma osmolality: water shifts from intracellular to extracellular components with resultant dilution Na^+ .
 - i. Hyperglycemia: plasma Na^+ falls by 1.4 mmol/L for every 100 mg/dL rise the in plasma glucose concentration.
 - ii. Mannitol
5. Pseudohyponatremia
- a. Normal plasma osmolality: aqueous phase diluted by excess protein lipids; total body water and Na^+ unchanged
 - i. Hyperlipidemia
 - ii. Hyperproteinemia
 - iii. Glycine solutions: post-transurethral resection of prostate/bladder tumor
6. Endocrine disorders resulting in hyponatremia
- a. Adrenal insufficiency
 - i. Cortisol deficiency that leads to hypersecretion of ADH
 - (1) Indirectly: secondary to volume depletion
 - (2) Directly: co-secreted with corticotrophin-releasing factor
 - b. Hypothyroidism: leads to decreased Na^+ secondary to the resulting decrease in cardiac output and glomerular filtration rate (GFR), and the increased ADH secretion in response to hemodynamic stimuli.

Clinical Manifestations of Hyponatremia

1. Symptoms are primarily neurologic and severity is determined by the rapidity of serum Na^+ loss.
2. Symptoms are related to fluid shift leading to an increase in intracellular fluid volume – specifically, brain cell swelling or cerebral edema.
 - a. Increasing severity of hyponatremia with falling Na^+ levels:

Diagnosis of Hyponatremia

1. Thorough history and physical examination
 - a. Assessment of extracellular fluid volume status:
 - i. Decreased Na^+ , increased extracellular fluid volume, decreased effective circulating volume
 - (1) Congestive heart failure
 - (2) Hepatic cirrhosis
 - (3) Nephrotic syndrome
 - ii. Decreased Na^+ , near normal extracellular fluid volume, decreased effective circulating volume

Table 10.1. Symptoms Associated with Hyponatremia Levels

Symptoms	Associated Serum [Na^+] (mEq/L)
Asymptomatic	Over 125, or chronic over 115
Anorexia, nausea, malaise	120–125
Headache, lethargy, confusion, agitation, obtundation	110–120
Stupor, seizures, coma	Less than 110

- (1) Hypothyroidism
- (2) Adrenal insufficiency
- iii. Decreased Na^+ , euvolemic
 - (1) SIADH diagnosis
 - (a) Hypotonic hyponatremia
 - (b) Inappropriately concentrated urine
 - (c) Exclude hemodynamic stimulus to vasopressin
 - (d) Signs of extracellular volume depletion must be absent.
 - (e) Cardiac and hepatic systems must be normal.
 - (f) No proteinuria
- b. Review of medication history
- c. Check serum Na^+ concentration and plasma osmolality.
- d. Check concentration of urine.

Lab Tests Once Hyponatremia Is Determined

1. Plasma osmolality
 - a. Normally ranges from 275 to 290 mOsm/kg
 - b. >290 mOsm/kg: consider hyperglycemia or administration of mannitol
 - c. 275–290 mOsm/kg: consider hyperlipidemia or hyperproteinemia
 - d. <275 mOsm/kg: plasma is hypotonic; the next step is evaluation of urine osmolality and volume status.
2. Urine osmolality
 - a. <100 mOsm/kg: consider excessive water intake or primary polydipsia
 - b. >100mOsm/kg: indicates impaired renal diluting ability; the next step is to determine extracellular fluid volume status.
3. Extracellular fluid volume status: distinguish between hypovolemia and hypervolemic hyponatremia by checking the urine Na^+ concentration.
 - a. Euvolemia: SIADH, endocrinopathies, postoperative states (pain, nausea, stress), diuretics
 - b. Hypovolemia
 - i. Urine Na^+ concentration:
 - (1) >20 mEq/L: renal solute loss: consider diuretics, osmotic diuresis, Addison's disease
 - (2) <10 mEq/L: non-renal solute loss: consider GI loss, skin
 - c. Hypervolemia

10. Sodium

- i. Urine Na^+ concentration
 - 1) >20 mEq/L: consider renal failure.
 - 2) <10 mEq/L: consider edematous states such as congestive heart failure (CHF), cirrhosis, nephrotic syndrome.
4. Fractional excretion of sodium (FENa): helps to distinguish renal from nonrenal failure
5. Thyroid stimulating hormone: distinguishes hypothyroidism
6. Albumin: distinguishes paraproteinemia
7. Potassium (K^+) concentration
8. Bicarbonate level

Treatment of Hyponatremia

1. Therapy can be guided by the assessment of plasma osmolality:

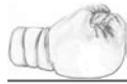
- a. Treatment of isotonic hyponatremia should be directed at underlying hyperlipidemia and paraproteinemia.
- b. Treatment of hypertonic hyponatremia should be directed at correcting hyperglycemia.
- c. Treatment of hypotonic hyponatremia as follows:
 - i. Therapy is directed at raising extracellular fluid tonicity to shift water out of the intracellular space alleviating cellular edema.
 - ii. The rate of correction must be carefully monitored; otherwise, too rapid a correction may produce central pontine myelinolysis (CPM).
 - (1) CPM is an osmotic demyelination syndrome associated with irreversible neurologic deficits which develop 2–6 days after treatment: dysarthria, dysphagia, incoordination, quadriplegia, coma.
 - (a) Rate of correction
 - (i) Symptomatic: increase Na^+ level by 8–10 mEq/L in 4–6 hours with hypertonic saline.

TKO: Exact rate of correction with hypertonic saline

1. 10 mEq/L in the first 24 hours
2. 20 mEq/L in the first 48 hours
3. In dire situations where serum Na^+ is less than 105 mEq/L, the rate of correction is 1–2 mEq/L for the first few hours using 3% hypertonic saline.

TKO: To use 3% hypertonic saline

1. Calculate the amount of sodium to be administered by multiplying the target change in serum sodium by the patient's total body water (TBW).
 - a. $\text{TBW} = 0.6 \times \text{kg}$
 - b. Example: 70 kg male
 - i. $0.6 \times 70 = 42$
2. Example: to change the serum Na^+ in the first 24 hours by an equivalent of 10 mEq/L in a 70 kg male: $10 \times 42 = 420$ mEq
3. 3% saline has a Na^+ concentration of 513 mEq/L, therefore $420/513 = 0.82$ L or 820 mL which should be given in the first 24 hours.



KO TREATMENT PLAN

Pre-operative

1. The patient's hyponatremia is related to excessive water drinking not likely related to psychiatric illness.
 - a. Patients with pure water excess appear euvolemic.
2. The patient should be transferred from the emergency department to the medical intensive care unit.
 - a. She should be electively intubated due to her mental confusion.
 - b. Peripheral access should be obtained.
 - c. Central access should be obtained to monitor central venous pressure and help establish her volume status.
 - d. An arterial catheter should be placed for frequent serum laboratory tests.
 - e. A Foley catheter should be inserted to closely monitor urine output.
 - f. A chest X-ray should be obtained to rule out pulmonary edema.
3. Laboratory
 - a. Serum Na^+ should be monitored as fluid restriction occurs along with measurement of urine output.
 - b. The normal response to significant water ingestion is to excrete maximally dilute urine (urine osmolality less than 100 mOsm/kg).
 - c. Usually, low blood urea nitrogen and serum uric acid concentration is the only laboratory evidence of increased total body water.
 - d. Toxicology screen should be done to rule out the use of ecstasy.
 - i. Ecstasy can have the resulting side effect of significant hyponatremia.
4. The patient can be started on an infusion of 3.0% saline for symptomatic hyponatremia.
 - a. Patients with psychogenic polydipsia require fluid restriction and/or 3% saline, depending on the presence of symptoms and acuity of the hyponatremia.

Intra-operative

1. If this patient does need emergent surgery, meticulous attention should be paid to her volume status.
 - a. 3% hypertonic saline should be infused along with serial monitoring of serum sodium concentrations.
 - b. Pre-induction arterial line for frequent electrolyte measurements
 - c. Central venous line to measure her volume status
 - d. Careful titration of anesthetics secondary to her already confused and lethargic state
 - e. She will require RSI secondary to her nausea and likely full stomach; she did drink a lot of water.

TKO: Delay any case until the patient's hyponatremia improves unless it is an absolute emergency. This patient

should not go to the operating room for an elective case since it puts her at a significantly increased risk for morbidity and mortality.

Post-operative

1. Close monitoring in the intensive care unit
2. If she required an emergent surgery, delay extubation until her hyponatremia begins to resolve.
 - a. Her preoperative confusion and lethargy will not resolve until her serum sodium values are normalized.

BIBLIOGRAPHY

1. Humes DH, Dupont HL, Gardner LB, et al. *Kelley's Textbook of Internal Medicine*, 4th ed. Philadelphia: Lippincott, Williams & Wilkins, 2000, pp 1150–61.
2. Kasper DL, Braunwald E, Hauser SL, et al. *Harrison's Principles of Internal Medicine*, 16th edition. New York: McGraw Hill Medical, 2005, pp 252–8.
3. Winshall JS, Lederman RJ. *Tarascon Internal Medicine & Critical Care Pocketbook*, 3rd edition. Lompoc: Tarascon Publishing, 2004, pp 146–9.
4. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th edition. New York: Churchill Livingstone, 2005, pp 1764–8.

11. Potassium^{1,2,3,4,5,6,7}

Jennifer Eison, MD

HYPERKALEMIA

SAMPLE CASE

A 65-year-old male patient with hypertension, diabetes, and chronic renal failure on hemodialysis presents for an elective left arm arterio-venous fistula grafting. The patient unfortunately missed his dialysis and his last K^+ was 6.7 mEq/L. The patient is asymptomatic. What are your concerns? What tests and labs do you want? Would you postpone the surgery? Would you demand hemodialysis first? Would you be less concerned if the patient was not on dialysis? If this case suddenly became an emergency, what is your anesthetic plan?

CLINICAL ISSUES

Definition

Hyperkalemia is defined as potassium (K^+) concentration greater than 5 mEq/L.

Causes of Hyperkalemia

1. Pseudohyperkalemia: artificially increased serum K^+ concentration
 - a. Hemolysis
 - b. Prolonged use of a tourniquet with or without fist clenching: releases K^+ from the ischemic muscle
 - c. Marked leukocytosis: white cell count $>70,000/cm^3$ causes spurious hyperkalemia due to K^+ leakage from the cells when the specimen is allowed to clot.

- d. Thrombocytosis: platelet count $>1,000,000/cm^3$ can release K^+ from platelets during clotting.
2. Endogenous K^+ : K^+ released from cells
 - a. Tumor lysis syndrome
 - b. Rhabdomyolysis
 - c. Exercise-induced hyperkalemia related to the degree of exertion
 - d. Thermal and electrical burns
3. Exogenous K^+
 - a. Increased K^+ intake: rarely a cause
 - b. Iatrogenic K^+ administration
 - c. Transfusions with near-expired blood products
 - i. Increased K^+ content
4. Renal conditions
 - a. Renal insufficiency/chronic renal failure
 - b. Acute oliguric renal failure: increased K^+ release from the cells (acidosis, catabolism) and decreased K^+ excretion
 - c. Nephropathy due to
 - i. Drug-induced interstitial nephritis
 - ii. Lupus nephritis
 - iii. Sickle cell disease
 - iv. Diabetic nephropathy
 - v. Obstructive uropathy
 - vi. Postrenal transplantation
 - d. Hyperkalemic distal (type 4) renal tubular acidosis: due to hypoaldosteronism or chloride shunt
5. Acidosis
 - a. Metabolic acidosis: raises K^+ an average of 0.7 mEq/0.1 pH unit.
 - b. Respiratory acidosis: raises K^+ an average of 0.1 mEq/0.1 pH unit.

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c. Diabetic ketoacidosis results in hyperkalemia from high extracellular fluid osmolality pulling both water and K^+ from the intracellular space. Consequence of

- i. Insulin deficiency
- ii. Hypertonicity from hyperglycemia

6. Drugs

a. Succinylcholine

- i. It raises the K^+ level 0.5–0.7 mEq/L in otherwise normal patients.
- ii. Exaggerated release of K^+ from the cellular membrane can be seen when succinylcholine is used in patients who have the following conditions:

- (1) Massive trauma
- (2) Burns
- (3) Neuromuscular disease

b. β -blocker: elevates K^+

c. Severe digitalis toxicity: due to Na^+ - K^+ ATP-ase pump altering the K^+ distribution across cellular membranes

d. Angiotensin-converting enzyme inhibitor (ACEI): results in impaired aldosterone release which in turn increases the K^+ in patients who are more susceptible.

These include patients with

- i. Diabetes
- ii. Renal insufficiency
- iii. Decreased effective circulation volume
- iv. Bilateral renal artery stenosis
- v. Concurrent use of K^+ sparing diuretics and non-steroidal anti-inflammatory drugs (NSAIDs)

e. Heparin: inhibits aldosterone biosynthesis, therefore leading to renal sodium wasting and potassium retention

f. Cyclosporine: suppresses renin levels and may impair aldosterone secretion by directly interfering with tubular K^+ secretion

g. Prostaglandin synthetase inhibitors (indomethacin): reduce renin release and produce reversible hyporeninemic hypoaldosteronism

h. Medications that impair renal K^+ excretion

- i. Spironolactone: competitive mineralocorticoid antagonist
- ii. Amiloride or triamterene: blocks the principal cell apical Na^+ channel and thus K^+ excretion causing hyperkalemia via an aldosterone-independent mechanism.
- iii. Trimethoprim and pentamidine (antivirals): block distal nephron Na^+ reabsorption which may contribute to elevated K^+ levels in patients with HIV being treated for *P. carinii pneumonia*.

7. Endocrine abnormalities

a. Primary adrenal insufficiency (Addison's) or congenital adrenal enzyme deficiency: both result in decreased aldosterone synthesis leading to renal sodium wasting and potassium retention.

b. Pseudohypoaldosteronism: a rare familial disorder which presents with hyperkalemia, metabolic acidosis, renal Na^+ wasting, hypertension, and increased renin and aldosterone levels.

Table 11.1. Approximate Intracellular and Extracellular Concentrations of Electrolytes (mEq/L)

	Intracellular	Extracellular
Sodium (Na^+)	12	150
Potassium (K^+)	150	4
Chloride (Cl^-)	4	115
Phosphate (PO_4^{3-})	40	2

Adapted from Seeley RR, Tate P, Stephens TD. *Anatomy & Physiology*, 8th ed. New York: McGraw-Hill, 2008.

c. Hyporeninemic hypoaldosteronism: results in mild increase in K^+ due to euvoemia or extracellular fluid volume expansion and suppressed renin aldosterone levels, seen in

- i. Mild renal insufficiency
- ii. Diabetic nephropathy
- iii. Chronic tubulointerstitial disease

8. Hyperkalemia periodic paralysis: a rare autosomal dominant disorder presenting with episodic paralytic attacks that results in shifts of K^+ from the cellular compartment.

a. Precipitated by

- i. Excitement
- ii. Cold
- iii. Fasting
- iv. Stress
- v. Infection
- vi. General anesthesia

9. Chronic hyperkalemia: associated with decreased renal K^+ excretion:

a. Impaired secretion

- i. Impaired Na^+ reabsorption
- ii. Increased chloride reabsorption

b. Diminished distal tubule solute delivery

- i. Protein malnourishment: low urea excretion
- ii. Extracellular fluid volume contraction: decreased distal tubule Na^+Cl^- delivery

Clinical Features of Hyperkalemia

1. Weakness, tingling, and paresthesias
2. Flaccid paralysis particularly of the lower extremities
3. Hypoventilation if respiratory musculature is involved
4. Increased K^+ inhibits renal production of ammonia and reabsorption in the thick ascending loop of Henle; therefore net acid excretion is impaired leading to metabolic acidosis exacerbating K^+ movement out of cells.
5. Cardiac toxicity: does not correlate well with the plasma concentration of K^+
 - a. Cardiac toxicity usually precedes neurologic toxicity.

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b. EKG changes with increasing severity of hyperkalemia as follows:

- i.** Increased T wave amplitude occurs as the K^+ levels approach 6–7 mEq/L.
- ii.** Flattened P wave, prolonged PR interval, and widening of QRS complex with deepening of the S wave
- iii.** AV conduction delay
- iv.** Loss of P wave
- v.** Progressive widening of the QRS complex and merging of T wave: resembles a sine-wave pattern.
- vi.** Ventricular fibrillation/flutter and asystole occur as the K^+ levels approach 10–12 mEq/L.

Diagnosis of Hyperkalemia

1. Obtain a history and physical examination including review of current medication therapy.
2. Check the serum chemistry.
 - a.** If the serum concentration of K^+ is elevated, treatment takes precedence over diagnosis.
 - b.** Obtain an EKG to ascertain if cardiac toxicity is occurring.
 - c.** Rule out pseudohyperkalemia or spurious laboratory values; check a complete blood count (CBC) and peripheral blood smear.
 - d.** Rule out transcellular K^+ shift.
 - i.** Review the clinical circumstances.
 - ii.** Determine the patient's acid-base status.
 - iii.** Check other labs, including
 - (1) Creatinine phosphokinase
 - (2) Myoglobin
 - (3) Urinalysis
 - (4) Serum drug levels: digoxin
3. Rule out renal etiology of the hyperkalemia.
 - a.** GFR less than 10 mL/min
 - i.** Acute renal failure
 - ii.** Oliguria
 - iii.** K^+ load
 - (1) Exogenous source
 - (2) Endogenous source
 - b.** GFR greater than 20 mL/min
 - i.** Measure plasma renin and aldosterone levels.
 - (1) Low aldosterone and high renin suggests adrenal insufficiency: confirmed by measuring plasma cortisol.
 - (2) Low aldosterone and renin: hyporeninemic hypoaldosteronism
 - (3) Normal aldosterone and renin: primarily renal tubular defect

Treatment of Hyperkalemia

1. Treatment action is determined by the severity of patient's EKG changes and symptomatology.
 - a.** Non-emergent treatment
 - i.** Reduce K^+ intake: eliminate K^+ -rich foods from diet.

ii. Increase K^+ output by promoting gastrointestinal loss with cation exchange resins which promote the exchange of Na^+ for K^+ .

- a.** Onset 1–2 hours
- b.** Duration 4–6 hours

iii. Intravenous diuretics: loop and thiazide diuretics
iv. Consider dialysis in a patient with renal failure.
v. Avoid medication that can interfere with K^+ elimination.

b. Emergent treatment goals

- i.** Protect the myocardium.
 - (1) Calcium chloride given in a central line or calcium gluconate in peripheral vein
 - (a) The onset of the effect is within minutes but the duration is short-lived (30–60 minutes).
 - (b) The dose should be repeated every 5–10 minutes as necessary.
 - (c) Avoid in suspected digitalis toxicity.
- ii.** Shift K^+ into cells temporarily.
 - (1) Give sodium bicarbonate 50 mEq intravenously (IV) over 5 minutes.
 - (a) Note that a patient in end stage renal failure may not respond.
 - (2) D50 plus insulin IV
 - (a) Onset 10–20 minutes
 - (b) Duration 4–6 hours
 - (3) Albuterol (nebulized β_2 -adrenergic agonist): promotes cellular K^+ uptake.
 - (a) Onset in 30 minutes
 - (b) Duration 2–4 hours
- iii.** Continue with non-emergent treatments as listed above once the myocardium is stabilized.



KO TREATMENT PLAN

Pre-operative

1. The ideal goal K^+ level is below 5 mEq/L prior to surgery.
 - a.** If the patient is symptomatic, the surgery should be postponed.
 - i.** Keep in mind though, that clinical signs and symptoms do not correlate predictably with K^+ serum concentrations.
 - b.** Acute hyperkalemia
 - i.** The surgery should be postponed and treatment should be initiated immediately.
 - c.** Many authors describe thresholds for postponing surgery varying from 5.5 to 5.9 mEq/L.
 - d.** Some studies suggest that this may be an arbitrary number when dealing with patients who have chronic hyperkalemia. Chronic hyperkalemia does not automatically contraindicate surgery.
 - i.** First determine whether the hyperkalemia is chronic or acute.

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- b.** Extrarenal: urine K^+ less than 20 mEq/day
 - i.** Gastrointestinal loss
 - (1) Diarrhea
 - (2) Laxative abuse: test stool and urine for phenolphthalein derivatives and consider proctoscopy for confirmation of melanosis coli.
 - (3) Villous adenomas, VIPomas (vasoactive intestinal peptide tumors), and non-beta cell pancreatic islet tumor: create profuse diarrhea.
 - (4) Intestinal bypass or fistula
 - (5) Vomiting and gastric suctioning: chloride depletion contributes to hypokalemic metabolic alkalosis.
 - ii.** Integumentary loss
- c.** Renal: urine K^+ greater than 20 mEq/day
 - i.** Drugs
 - (1) Diuretics
 - (2) High-dose steroids
 - (3) Penicillin derivatives
 - (4) Hypomagnesium inducing drugs: amphotericin B, aminoglycosides, cisplatin, foscarnet
 - (a) Hypokalemia is resistant to correction unless magnesium replacement occurs first.
 - ii.** Renal tubular acidosis (RTA): seen clinically as metabolic acidosis, renal K^+ wasting, and hypokalemia
 - (1) Distal RTA (type I): seen most often
 - (2) Proximal RTA (type II): common during usage of high-dose bicarbonate
 - iii.** Diabetic ketoacidosis: several components lead to hypokalemia.
 - (1) Delivery of beta hydroxybutyrate to collecting ducts and K^+ loss
 - (2) Uncontrolled hyperglycemia leads to K^+ depletion from osmotic diuresis.
 - (3) Treatment with insulin leads to decreased K^+ by stimulation of Na^+H^+ anti-porter secondary to the Na^+K^+ ATP-ase pump.
 - iv.** Mineralocorticoid excess
 - (1) Primary Hyperaldosteronism: characterized by low renin and hypertension.
 - (a) Conn's syndrome (adrenal adenoma)
 - (b) Adrenocortical hyperplasia
 - (c) Carcinoma
 - (2) Secondary Hyperaldosteronism: characterized by high renin and hypertension.
 - (a) Malignant hypertension
 - (b) Renin-secreting tumors
 - (i) Renal cell carcinoma
 - (ii) Ovarian carcinoma
 - (iii) Wilm's tumor
 - (c) Renal artery stenosis
 - (3) Circumstances producing mineralocorticoid excess
 - (a) Inhibition of 11β -hydroxysteroid dehydrogenase: an enzyme necessary for the conversion of cortisol

(i) Glycyrrhizin acid: found in licorice and chewing tobacco

(ii) Carbenoxalone

(b) Cushing's syndrome

- v.** Rare syndromes that produce hypokalemia
 - (1) Bartter's syndrome
 - (2) Gitelman's syndrome
 - (3) Liddle's syndrome

Clinical Features of Hypokalemia

- 1.** Non-cardiac symptoms
 - a.** K^+ less than 3 mEq/L: symptoms are unlikely if K^+ is above this value.
 - i.** Fatigue
 - ii.** Myalgia
 - iii.** Muscular weakness of the proximal lower extremities
 - iv.** Constipation and intestinal ileus
 - v.** K^+ depletion manifests as polydipsia and polyuria.
 - vi.** Glucose intolerance from decreased K^+ due to impaired insulin secretion or peripheral insulin resistance
 - b.** Severe hypokalemia: less than 2 mEq/L
 - i.** Progressive weakness
 - ii.** Hypoventilation due to respiratory involvement
 - iii.** Complete paralysis
 - iv.** Increased risk of rhabdomyolysis: due to impaired muscle metabolism and blunted hyperemic response to exercise
- 2.** Cardiac symptoms due to hypokalemia
 - a.** Cardiac symptoms due to delayed ventricular repolarization: does not correlate well with specific plasma K^+ concentration.
 - b.** Cardiac problems frequently involve
 - i.** Arrhythmias
 - ii.** Atrial fibrillation
 - iii.** Premature ventricular contractions (PVC)
 - c.** Early Hypokalemia: K^+ less than 3 mEq/L
 - i.** Flattening or inversion of T wave
 - ii.** Prominent U wave: more than 1 mm
 - iii.** ST segment depression
 - iv.** Prolonged QT interval
 - v.** Hypokalemia predisposes to digitalis toxicity.
 - d.** Increasing severity of hypokalemia
 - i.** Prolonged PR interval
 - ii.** Decreased voltage
 - iii.** Widening of QRS
 - iv.** Increased risk of ventricular arrhythmia

Diagnosis of Hypokalemia

- 1.** Determined by the patient's history
- 2.** Exclude diuretic use, laxative abuse, and dietary causes.
- 3.** Exclude marked leukocytosis by obtaining a CBC.

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- a. Acute myeloid leukemia: white blood cells uptake plasma K^+ leading to a low K^+ concentration
4. Pseudohypokalemia: prevented by storing blood samples on ice
5. Determine acid-base disturbances
 - a. Arterial blood gas (ABG) sample
 - b. Urine K^+ and Cl^-
 - c. Measure renin and aldosterone levels.
 - d. Serum magnesium level
6. Determine if the cause is renal.
 - a. In the presence of renal K^+ loss, urine K^+ greater than 20 mEq/day, and metabolic alkalosis, a urine Cl^- is invaluable to differentiate etiology.
 - i. Urine Cl^- greater than 10 mEq/day suggests etiology of mineralcorticoid or glucocorticoid excess production.
 - ii. Urine Cl^- less than 10 mEq/day suggests a functioning kidney: hypokalemia is due to K^+ loss from the skin, GI tract, or from vomiting, or remote diuretic use.
 - b. The presence of renal K^+ loss, urine K^+ greater than 20 mEq/d, and metabolic acidosis suggests RTA.
 - c. Extrarenal etiology is suggested when urine K^+ is less than 20 mEq/day.

Treatment of Hypokalemia: total body K^+ deficit correlates poorly with serum levels

1. Identify the etiology and correct the underlying disorder.
2. Asymptomatic or minor symptoms (potassium of 2.5–3.5 mEq/L)
 - a. Give oral potassium replacement therapy: KCl 20–40 mEq PO q 4–6 hours.
 - b. Encourage K^+ -rich foods such as dried fruits, nuts, lima beans, tomatoes, carrots, potatoes, bananas, oranges, ground beef, lamb, and veal.
 - i. 1 inch banana or 1 oz orange juice: 1.6 mEq K^+
3. Cardiac manifestations or significant symptoms are present.
 - a. More aggressive therapy is warranted.
 - b. If the potassium level is less than 2.5 mEq/L, intravenous potassium replacement therapy should be given.
 - i. Intravenous supplementation: maximum 20–40 mEq/h ideally through central access with continuous cardiac monitoring
 - ii. Also, hospital admission or emergency room observation is indicated.
 - iii. Repeat serum K^+ level measurement every 1–3 hours.
4. Serum K^+ levels are difficult to replenish with coinciding hypomagnesemia. Therefore magnesium may need to be replaced prior to correcting K^+ .
5. If the patient is manifesting cardiac arrhythmias
 - a. Appropriate pharmacologic therapy should be initiated.
 - b. Place cardiac monitoring.
 - c. Establish intravenous access.
 - d. Assess respiratory status.
- e. Cardiac pacing should be considered in scenarios of severe bradycardia.
6. An internist or a nephrologist should be consulted for admission or follow-up care.



KO TREATMENT PLAN

Pre-operative

1. Case delay
 - a. According to Miller, available studies do not demonstrate an increased morbidity and mortality when patients undergo anesthesia with a plasma K^+ of 2.6 mEq/L or greater.
 - b. Proceeding with anesthesia is also dependent on the clinical setting
 - i. If the patient is having cardiac dysrhythmias, surgery should be postponed.
 - ii. Is hypokalemia acute or chronic?
 - (1) In the acute setting it is ideal to postpone surgery especially in the face of cardiac dysrhythmias
 - (2) In the setting of chronic depletion of K^+ without cardiac changes, it may be reasonable to proceed with surgery and plan to replace K^+ intra-operatively.
2. Labs and tests
 - a. Obtain serum magnesium: replacement of K^+ can be inhibited by hypomagnesemia.
 - b. EKG: rule out cardiac dysrhythmias.
 - c. Urine K^+ : rule out renal involvement.
3. In regard to the patient being underweight, tachycardic, and having low serum K^+ , you should determine if she uses or abuses diuretics and laxatives; whether she has an eating disorder that could contribute to her presentation. Express to the patient your concern for intra-operative cardiac dysrhythmias and complications her low serum K^+ has on her anesthetic management. As an aside, she could be taking other diet and weight loss aids with amphetamine derivatives that can make her more susceptible for cardiac events. Otherwise, if she denies the above scenario, you should investigate other causes for her low serum K^+ .

Intra-operative

1. Emergent surgery in a hypokalemic patient
 - a. Proceed with anesthesia once the patient is hemodynamically stable.
 - b. Use invasive blood pressure monitoring.
 - c. Check the electrolytes frequently.
 - d. Continue treatment of the hypokalemia in the operating room.
2. Non-emergent surgery
 - a. In the case scenario given, it is reasonable to proceed with surgery assuming no cardiac manifestations are present; however, investigation of underlying disorders should be made.

b. I would perform general anesthesia with endotracheal intubation due to surgery length and plan K⁺ replacement to be initiated through a peripheral IV.

i. Correcting K⁺: replace this patient's serum K⁺ no more than 40 mEq/L, and the infusion rate should not exceed 20 mEq/h unless the patient exhibits paralysis or malignant ventricular arrhythmia.

2. Induction

a. After standard ASA monitors are placed and preoxygenation occurs, induction can be carried out with fentanyl, propofol, and rocuronium assuming she has a good airway evaluation.

b. Avoid sympathomimetics, such as ketamine, due to her resting tachycardia.

3. Maintenance

a. The patient can be maintained on volatile anesthetics along with narcotics when necessary.

Post-operative

1. Plan for follow-up care and oral supplementation of her K⁺ level should be arranged with her primary care physician.

2. She should be counseled on the gravity of using diuretics and laxatives for weight loss.

BIBLIOGRAPHY

1. Humes HD, DuPont HL, Gardner LB, et al. *Kelley's Textbook of Internal Medicine*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2000, pp 1162-71.
2. Fauci AS, Braunwald E, Kasper DL, et al. *Harrison's Principles of Internal Medicine*, 16th ed. New York: McGraw Hill Medical Publishing Division, 2005, pp 258-63.
3. Winshall JS, Lederman RJ. *Tarascon Internal Medicine & Critical Care Pocketbook*, 3rd ed. Lompoc: Tarascon Publishing, 2004, pp 149-51.
4. Olson RP, Schow AJ, McCann R, et al. Absence of Adverse Outcomes in Hyperkalemic Patients Undergoing Vascular Access Surgery. *Canadian J of Anesthesia* 2003; 50: 553-57.
5. Palmer BF. *Approach to Fluid and Electrolyte Disorders and Acid-Base Problems*. Primary Care: Clinics in Office Practice 2008; 35(2): 195-213.
6. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1768-70.
7. Seeley RR, Tate P, Stephens TD. *Anatomy & Physiology*, 8th ed. New York: McGraw-Hill, 2008, pp 1005.

12. Calcium^{1,2,3,4,5,6,7}

Melvyn J. Y. Chin, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 25-year-old otherwise healthy male was involved in a motorcycle accident resulting in multiple long bone and pelvic fractures, and blood in his pelvis and abdomen. Massive blood loss occurred in the operating room, and the patient was rapidly transfused with 15 units of packed red blood cells (pRBC), 12 units of fresh frozen plasma (FFP), and two 6-packs of platelets. Despite the fluid resuscitation, the patient remained hypotensive. The astute resident in the case noted that the ionized calcium level on the last arterial blood gas was low and promptly administered 1 gm of intravenous calcium chloride. The blood pressure rapidly improved. Why was the patient's calcium low? Why did treating the hypocalcemia improve the blood pressure?

CLINICAL ISSUES

1. Total serum calcium = protein-bound (40%) + chelated fraction (10%) + ionized fraction (50%).
2. Ionized calcium is the physiologically active form of calcium.

- a.** Therefore, even if total serum calcium is low, it may not be clinically significant if the ionized calcium level is within the normal range.
3. Calcium is essential for
 - a.** Normal muscle function: excitation-contraction coupling
 - b.** Generation of cardiac pacemaker activity and cardiac action potential
 - c.** Cardiac contractility
 - d.** Neurotransmitter release
 - e.** cAMP and phosphoinositide function
 - f.** Membrane and bone structure
4. Main regulators of serum calcium
 - a.** Parathyroid hormone, calcitriol, and vitamin D
 - b.** Calcitonin
 - c.** Homeostasis of calcium is maintained by processes in the intestinal tract (absorption vs. secretion), bones (resorption vs. deposition), and kidneys (absorption vs. excretion).
5. Correction for hypoalbuminemia
 - a.** Add 1 mg/dL to the serum calcium level for every 1 g/dL of albumin concentration below 4.0 g/dL.

12. Calcium

HYPOCALCEMIA

Definition

1. Ionized calcium less than 1.0 mmol/L or 4.0 mg/dL
2. Total serum calcium less than 2.1 mmol/L or 8.5 mg/dL

Causes of Hypocalcemia

1. Parathyroid hormone (PTH) deficiency
 - a. Parathyroid gland damaged or removed
 - b. Parathyroid suppression from
 - i. Severe hypomagnesemia
 - ii. Burns
 - iii. Sepsis
 - iv. Pancreatitis
2. Vitamin D deficiency
3. Hyperphosphatemia: renal failure, tumor lysis, rhabdomyolysis
4. Renal failure: results in hyperphosphatemia and decreased calcitriol
5. Citrate toxicity: from massive transfusions (citrate binds to calcium, decreasing the ionized calcium concentration)
6. Acute alkalemia
7. Post-cardiopulmonary bypass
8. Acute pancreatitis

Clinical Manifestations of Hypocalcemia

1. Cardiovascular
 - a. ECG changes
 - i. Dysrhythmias: heart block in severe cases
 - ii. QT prolongation
 - b. Heart failure
 - c. Hypotension
 - d. Digitalis insensitivity
 - e. Impaired β -adrenergic action
2. Neuromuscular
 - a. Tetany:
 - i. Chvostek's sign: tapping on the facial nerve elicits a contraction of facial muscles.
 - ii. Trousseau's sign: inflating a blood pressure cuff to 20 mmHg above systolic blood pressure causing radial and ulnar nerve ischemia producing carpal spasms.
 - b. Muscle spasm
 - c. Papilledema
 - d. Seizure
 - e. Weakness
 - f. Fatigue
 - g. Paresthesias: around the mouth or fingertips
 - h. Irritability
 - i. Mental status changes
3. Respiratory
 - a. Apnea
 - b. Laryngeal spasm: can be seen in the immediate post-operative period after thyroidectomy or parathyroidectomy surgery.

- c. Bronchospasm
4. Psychiatric
 - a. Anxiety
 - b. Dementia
 - c. Depression
 - d. Psychosis

Treatment

1. Symptomatic and acute: seizures, laryngospasm, tetany, and cardiovascular depression
 - a. Intravenous calcium
 - b. Magnesium must also be replaced if it is low.
 - c. Correct metabolic and/or respiratory alkalosis.
2. Asymptomatic and less acute
 - a. Treat the underlying cause.
 - b. Oral calcium supplements
 - c. Oral vitamin D



KO TREATMENT PLAN

Pre-operative

1. Identify the underlying cause of hypocalcemia.
2. Treat the hypocalcemia.
 - a. Administer calcium
 - i. Intravenous infusion of 10% calcium chloride or calcium gluconate
 - ii. Oral administration of 500–1,000 mg elemental calcium every 6 hours
 - b. Administer vitamin D
 - i. Ergocalciferol 1200 mcg/day
 - ii. Dihydrotachysterol 200–400 mcg/day
 - iii. 1,25-dihydroxycholecalciferol 0.25–1.0 mcg/day
3. Monitor the electrocardiogram for dysrhythmias and prolonged QT.
4. Monitor for hypomagnesemia and treat if necessary.
5. Symptomatic hypocalcemia must be treated prior to proceeding to the operating room.

Intra-operative

1. Once in the operating room, try to avoid things that will continue to decrease the calcium level.
 - a. Avoid hyperventilation.
 - b. Avoid the administration of bicarbonate.
2. Hypocalcemia can occur with massive transfusion of pRBC or FFP because the citrate preservative in the blood product chelates the calcium in the blood.
 - a. The hypocalcemia can then result in hypotension secondary to the resulting poor cardiac function.
3. Repleting the calcium improves the contractility of the heart, which in turn increases the patient's blood pressure.

12. Calcium

4. An arterial line can be placed for frequent serum calcium blood draws.

HYPERCALCEMIA

Definition

1. Ionized calcium greater than 1.5 mmol/L.
2. Total serum calcium greater than 10.5 mg/dL: customarily defined in terms of total serum calcium

Causes

90% of cases are caused by malignancy (most common in inpatients) and primary hyperparathyroidism (most common in outpatients).

1. Hyperparathyroidism
2. Malignancy
 - a. Bone destruction from metastasis
 - b. Hormone-secreting malignant tissue
 - c. Hematologic malignancies
3. Vitamin D intoxication
4. Sarcoidosis and other granulomatous diseases
5. Hyperthyroidism
6. Immobilization
7. Milk-Alkali Syndrome
8. Drugs
 - a. Thiazide diuretics
 - b. Lithium

Clinical Manifestations

1. Mild to moderate hypercalcemia (11–13 mg/dL)
 - a. Lethargy
 - b. Anorexia
 - c. Nausea
 - d. Polyuria
 - e. At this level, some patients may even be asymptomatic.
2. Severe hypercalcemia (>13 mg/dL)
 - a. Neuromuscular
 - i. Muscle weakness: should receive decreased doses of nondepolarizing muscle relaxants.
 - ii. Depression
 - iii. Impaired memory
 - iv. Emotional lability
 - v. Lethargy
 - vi. Stupor
 - vii. Coma (usually occurs at 15–18 mg/dL)
 - b. Cardiovascular
 - i. Hypertension
 - ii. Dysrhythmias
 - iii. Widening QRS complex
 - iv. Short QT interval
 - v. Heart block
 - vi. Cardiac arrest

- vii. Digitalis sensitivity
- c. Other
 - i. Calcification in kidneys, skin, vessels, lungs, heart, and stomach
 - ii. Renal insufficiency: hypercalcemia impairs renal concentrating ability and excretory capacity for calcium.



KO TREATMENT PLAN

Pre-operative

1. Administration of normal saline to dilute plasma calcium and promote diuresis.
 - a. Hypercalcemia is usually associated with hypovolemia.
 - b. Normal saline restores the intravascular volume and increases urinary excretion of calcium.
 - c. Normal saline should be started at rates of 200–300 mL/hr.
 - d. Once volume depletion is corrected, start loop diuretics.
2. Diuresis
 - a. Along with fluid therapy, loop diuretics (furosemide) further enhance calcium excretion by increasing tubular sodium.
 - i. The sodium ions compete with calcium ions for reabsorption in the proximal renal tubules and loop of Henle.
 - b. The goal urine output should be 200–300 mL/hr.
3. Bisphosphonates (i.e., pamidronate 30–90 mg IV)
4. Calcitonin (2–8 U/kg IV, SQ, or IM every 6–12 hours)
5. Phosphate: risk of ectopic calcification: renal damage, fatal hypocalcemia
6. Glucocorticoids: active only in certain malignancies
7. Dialysis: useful in renal failure
8. Increasing physical activity
9. Treat underlying cause.
10. Ideally, surgery should be postponed until the calcium level is normalized.

Intra-operative

1. If the surgery is an emergency, then you must try to treat the hypercalcemia as quickly as possible intra-operatively: maintenance of hydration and urine output.
 - a. Place an arterial line for frequent blood samples.
 - b. Place a central line and central venous pressure monitor to monitor fluid resuscitation and diuresis.
 - c. Place a Foley catheter to monitor urine output.
2. Closely monitor the EKG for cardiac conduction abnormalities.
3. Dosing of muscle relaxants should be guided by neuromuscular monitoring.
 - a. Patients should receive a decreased dose of nondepolarizing muscle relaxants, especially if muscle weakness, hypotonia, or loss of deep tendon reflexes is present.

BIBLIOGRAPHY

1. Barash, P., Cullen, B.F., and Stoelting, R.K. *Clinical Anesthesia, 5th ed.* Philadelphia: Lippincott Williams & Wilkins, 2006, pp 198–201.
2. Braunwald, E., et al. *Harrison's Principles of Internal Medicine, 15th ed.* New York: McGraw-Hill, 2001, pp 2194–5, 2209–26.
3. Hensley, F., Martin, D., and Gravlee, G. *A Practical Approach to Cardiac Anesthesia, 4th ed.* Philadelphia: Lippincott Williams & Wilkins, 2008, pp 232–3.
4. Winshall, J., and Lederman, R. *Tarascon Internal Medicine & Critical Care Pocketbook, 3rd ed.* Lompoc: Tarascon Publishing, 2004, pp 63–4.
5. Miller, R., et al. *Miller's Anesthesia, 6th ed., vol. 2.* Philadelphia: Elsevier, 2005, pp 177–2.
6. Hines, Marschall. *Stoelting's Anesthesia and Co-Existing Disease, 5th ed.* Philadelphia: Churchill Livingstone, 2008, Chapter: Disorders of Calcium.
7. Goldman L., Ausiello D. *Cecil Medicine, 23rd ed.* Philadelphia: Saunders Elsevier, 2008, Chapter 266.

13. Magnesium^{1,2,3,4,5,6}

Melvyn J. Y. Chin, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 27-year-old female, G1P0 at 39 weeks gestational age, is currently receiving a magnesium sulfate infusion for pre-eclampsia. You are consulted for a possible cesarean section. You begin to interview her and notice that she is lethargic and has a hard time staying awake. You promptly check her deep tendon reflexes and notice that they are hyporeflexic. You immediately notify the obstetrician of your findings, obtain a serum magnesium level, and promptly stop the magnesium sulfate infusion. What are your concerns? How would this affect your anesthesia?

CLINICAL ISSUES OF MAGNESIUM

1. Ionized magnesium is the physiologically active form of magnesium.
2. Normal level of magnesium is 1.8–2.5 mg/dL.
3. Magnesium is essential for
 - a. Various enzymatic reactions
 - i. DNA and protein synthesis
 - ii. Energy metabolism
 - iii. Glucose utilization
 - iv. Fatty acid synthesis and breakdown
 - b. Regulation of
 - i. Sodium-potassium pumps
 - ii. Ca-ATPase enzymes
 - iii. Adenylate cyclase
 - iv. Proton pumps
 - v. Slow calcium channels: contributes to maintenance of normal vascular tone and prevention of vasospasm.
 - vi. Parathyroid hormone (PTH) secretion and sensitivity to PTH and vitamin D
 - vii. Membrane excitability

- c. Membrane and bone structure
4. Main regulators of serum magnesium
 - a. Absorbed through the gastrointestinal (GI) tract.
 - b. Filtered, reabsorbed, and excreted by the kidneys.

Magnesium Therapy Indications

1. Premature labor: tocolysis
2. Pre-eclampsia and eclampsia: anti-convulsant (therapeutic magnesium levels range from 5.0 to 7.0 mg/dL).
3. Tetanus and pheochromocytoma: blocks the release of catecholamines from adrenergic nerve terminals and adrenal glands.
4. Cardiology
 - a. Improves myocardial oxygen supply and demand.
 - b. Reduces the incidence of dysrhythmias in post-myocardial infarction (MI) patients and in congestive heart failure (CHF) patients.
 - c. Decreases short-term mortality after an acute MI.
 - d. Treatment for torsades de pointes ventricular tachyarrhythmia
5. Respiratory
 - a. Asthma management: contributes to smooth muscle relaxation of the bronchioles.

HYPERMAGNESEMIA**Definition**

1. Magnesium level greater than 2.5 mg/dL
2. Typically asymptomatic at levels between 2.5 and 5.0 mg/dL.
3. Less common than hypomagnesemia because a magnesium load is usually quickly excreted in a patient with normal renal function.

13. Magnesium

Causes

1. Majority of cases are iatrogenic: administration of magnesium in antacids, laxatives, enemas, parenteral nutrition, or infusions: especially in the setting of renal failure.
2. Other rare causes
 - a. Hypothyroidism
 - b. Addison's disease
 - c. Lithium toxicity

Clinical Manifestations

1. Magnesium levels of 5–7 mg/dL (The therapeutic level for pre-eclampsia/eclampsia)
 - a. Lethargy
 - b. Drowsiness
 - c. Flushing
 - d. Nausea
 - e. Vomiting
 - f. Diminished deep tendon reflexes (DTRs)
2. Magnesium levels above 7.0–12 mg/dL
 - a. Cardiovascular effects
 - i. Hypotension: direct vasodilation
 - ii. Dysrhythmias
 - iii. Increased QRS duration
 - iv. Prolonged PR and QT intervals
 - v. Depressed myocardial performance
 - b. Neuromuscular
 - i. Muscle weakness
 - (1) Hypermagnesemia antagonizes the release and effect of acetylcholine at the neuromuscular junctions.
 - (2) Potentiates the action of depolarizing and non-depolarizing muscle relaxants and decreases the potassium release in response to succinylcholine.
 - (a) Muscle relaxants should be carefully titrated by assessing the degree of neuromuscular blockade (i.e., use of a nerve stimulator).
 - (3) May precipitate severe muscle weakness in Eaton-Lambert syndrome or myasthenia gravis patients.
 - ii. Somnolence
 - iii. Loss of DTRs
 - b. Respiratory effects
 - i. Depressed respiration and apnea from muscle weakness and/or paralysis
 - ii. Bronchodilator effects
3. Magnesium levels above 12 mg/dL
 - a. Hypotension
 - b. Bradycardia
 - c. Diffuse vasodilation
 - d. Paralysis
 - e. Coma
 - f. Significant respiratory depression
 - g. Complete heart block
 - h. Cardiac arrest

Treatment of Hypermagnesemia

1. Stop or treat the underlying cause.
2. Forced diuresis with saline followed by a loop diuretic
3. Intravenous calcium
4. Hemodialysis



KO TREATMENT PLAN

Pre-operative

1. Identify and treat the underlying cause.
 - a. This usually means stopping the magnesium-containing preparation.
 - b. Draw a magnesium level.
2. Magnesium is quickly excreted by the kidneys.
 - a. Further diuresis can be induced with an infusion of normal saline and/or intravenous furosemide.
3. Intravenous calcium (5–10 mEq) will antagonize the neuromuscular and cardiac toxicity of hypermagnesemia.
4. Renal failure or life-threatening situations may require hemodialysis.
5. Non-urgent surgeries should be delayed until the patient's significant symptoms have resolved.

Intra-operative

1. Invasive cardiovascular monitoring
 - a. Measure and treat the associated hypotension and vasodilation.
 - b. Guide fluid resuscitation.
 - c. Guide the forced diuresis.
2. Avoid acidosis: it exaggerates hypermagnesemia.
3. Muscle relaxant
 - a. Magnesium enhances the action of non-depolarizing muscle relaxants.
 - b. Prolongs the action of depolarizing muscle relaxants.
 - c. Decreased initial and subsequent doses, especially in patients with muscle weakness.
 - i. Use your peripheral nerve stimulator as a guide.
4. If the sample patient requires an emergent cesarean section
 - a. Regional anesthesia is preferred.
 - i. Use an existing epidural or place a spinal.
 - b. If a general anesthetic is required
 - i. Rapid sequence induction with thiopental and succinylcholine
 - ii. Maintenance anesthetic with isoflurane and nitrous oxide
 - iii. Place an arterial line to measure and treat the associated hypotension, and for frequent blood samples.
 - iv. An attempt can be made at forced diuresis.
 - (1) Be careful to closely follow the volume status in a pre-eclamptic patient.
 - c. The patient is likely to require prolonged ventilation.

13. Magnesium

Post-operative

1. Hypermagnesemia and its associated skeletal muscle weakness often lead to difficulty in weaning from mechanical ventilation.
2. Patients often require a stay in the intensive care unit until the hypermagnesemia is corrected.

HYPOMAGNESEMIA

Definition

1. Magnesium level less than 1.8 mg/dL
2. Symptoms are rare at levels between 1.5 and 1.7 mg/dL.
3. Symptoms often occur when the magnesium level is <1.2 mg/dL.

Causes

1. Renal loss
 - a. Volume expansion
 - b. Hypercalcemia
 - c. Osmotic diuresis: diabetes mellitus
 - d. Renal disease with magnesium wasting: advanced renal disease may lead to magnesium retention.
 - e. Drugs
 - i. Loop diuretics
 - ii. Aminoglycosides
 - iii. Cisplatin
 - iv. Cyclosporine
 - v. Amphotericin B
 - vi. Ethanol

(1) Hypomagnesemia is prevalent in alcoholics: seen in about 30% of alcoholics admitted to the hospital.
2. Gastrointestinal loss/decreased intestinal absorption
 - a. Vomiting
 - b. Prolonged nasogastric suctioning
 - c. Gastrointestinal or biliary fistulas
 - d. Intestinal drains
 - e. Diarrhea: Crohn's disease and ulcerative colitis

Clinical Manifestations

Usually seen at magnesium levels below 1.2 mg/dL.

1. Cardiovascular
 - a. ECG changes
 - i. Arrhythmias: ventricular tachycardia, torsades de pointes, and ventricular fibrillation
 - ii. PR and QT prolongation
 - iii. U waves
 - iv. Nonspecific T wave changes
 - b. Heart failure
 - c. Hypotension
 - d. Aggravates digoxin toxicity
 - e. Coronary artery spasm

2. Neuromuscular/psychiatric

- a. Muscle spasm
 - i. Chvostek's sign: tapping on the facial nerve elicits a contraction of facial muscles.
 - ii. Trousseau's signs: inflating a blood pressure cuff to 20 mm Hg above the systolic blood pressure causing radial and ulnar nerve ischemia producing carpal spasms.
 - b. Muscle weakness
 - c. Lethargy
 - d. Paresthesias
 - e. Seizures
 - f. Depression
 - g. Confusion
 - h. Coma
 - i. Tetany
3. Endocrine
 - a. Decreases parathyroid hormone (PTH) secretion and impairs end-organ responsiveness (i.e., bone, kidney, intestine) to PTH.
 - i. Associated with hypocalcemia.
 - b. Hypokalemia
 - i. Renal potassium wasting: the sodium-potassium pump is magnesium dependent.

Treatment

1. Depends on the severity of the deficiency.
 - a. Treat patients with
 - i. Cardiac arrhythmias and seizure
 - ii. Associated severe hypocalcemia or hypokalemia
 - iii. Severe hypomagnesemia less than 1.4 mg/dL
 - (1) Treat with intravenous magnesium as a bolus.
 - (2) Repeat the bolus dose until symptoms resolve.
 - (3) Once symptoms resolve, continue a slower infusion for a few days.
 - b. Mild cases
 - i. Oral magnesium salts



KO TREATMENT PLAN

Pre-operative

1. Identify and treat the underlying cause.
 - a. 24-hour urinary magnesium excretion will help separate renal from non-renal causes of hypomagnesemia
 - i. Hypomagnesemia coupled with high urinary excretion of magnesium (>3–4 mEq/day) suggests a renal etiology.
2. Mild hypomagnesemia (magnesium level 1.5–1.8 mg/dL)
 - a. Oral magnesium replacement is effective.
3. Moderate to severe (magnesium level <1.2 mg/dL):
 - a. Parenteral magnesium is usually needed (usually in the form of intravenous magnesium sulfate 2 g over the first hour, with a cumulative dose of up to 6 g over 24 hours).

13. Magnesium

4. A non-emergent anesthetic should be delayed if the patient is displaying significant signs or symptoms: cardiac arrhythmias or seizures.

Intra-operative

1. Anticipate ventricular arrhythmias and be prepared to treat them.
2. Guide muscle relaxation with a peripheral nerve stimulator.
 - a. Hypomagnesemia is associated with both muscle weakness and excitation.
3. Prepare for possible prolonged ventilation secondary to the associated impairment of respiratory muscle power.
4. Be prepared for seizures.
5. Avoid fluid loading, particularly with sodium-containing solutions.

6. Avoid the use of diuretics: renal excretion of magnesium passively follows sodium excretion.

BIBLIOGRAPHY

1. Barash, P., Cullen, B.F., and Stoelting, R.K. *Clinical Anesthesia, 5th ed.* Philadelphia: Lippincott Williams & Wilkins, 2006, pp 202-4.
2. Braunwald, E., et al. *Harrison's Principles of Internal Medicine, 15th ed.* New York: McGraw-Hill, 2001, pp 2197-8.
3. Winshall, J., and Lederman, R. *Tarascon Internal Medicine & Critical Care Pocketbook, 3rd ed.* Lompoc: Tarascon Publishing, 2004, p 151.
4. Miller, R., et al. *Miller's Anesthesia, 6th ed., vol. 2.* Philadelphia: Elsevier, 2005, pp 1772-4.
5. Hines, Marschall. *Stoelting's Anesthesia and Co-Existing Disease, 5th ed.* Philadelphia: Churchill Livingstone, 2008, Chapter: Disorders of Magnesium.
6. Goldman L., Ausiello D. *Cecil Medicine, 23rd ed.* Philadelphia: Saunders Elsevier, 2008, Chapter 120.

14. Intravenous (IV) Anesthetics^{1,2}

Varun Dixit, MD and Jessica A. Lovich-Sapola, MD

CLINICAL ISSUES

Properties of the Ideal IV Anesthetic

Physical Properties

1. Long shelf life
2. Water soluble
3. Drug compatibility and stability in solution
4. No pain on injection, veno-irritation, or local tissue damage from extravasation
5. Non-teratogenic
6. Analgesic
7. Amnestic
8. Hypnotic

Pharmacological Properties

1. Rapid and smooth onset
2. Lack of cardiovascular and respiratory depression
3. Rapid metabolism to pharmacologically inactive metabolites
4. No histamine release or hypersensitivity reactions
5. Rapid emergence without excitation or delirium
6. Anti-emetic properties
7. No accumulation with infusion

TKO: You will be asked which IV anesthetic you plan to use and why. There is usually no single correct answer. Just pick one and defend it!!!!

Propofol

1. Physical attributes
 - a. 2,6 diisopropylphenol
 - b. Insoluble in water
 - c. Highly lipid soluble
 - d. Prepared in the form of an emulsion
 - e. Stable at room temperature
 - f. Not light sensitive
2. Metabolism
 - a. Rapidly metabolized in the liver to water-soluble compounds
 - b. Water-soluble compounds are then excreted by the kidneys
 - c. May have a component of lung metabolism

3. Exact mechanism of action
 - a. Primarily a hypnotic
 - b. Exact mechanism is unknown
 - c. Mechanism may be due to GABA potentiation
4. Pharmacokinetics
 - a. Elimination half-life is 4–7 hours
 - b. Clearance is 20–30 mL/kg/min
 - c. Apparent volume of distribution at steady state is 2–10 L/kg
5. Effects
 - a. Central nervous system (CNS)
 - i. Rapid onset of hypnosis after doses of 2.5 mg/kg (one arm-brain circulation) with a peak effect seen at 90–100 seconds
 - ii. Sense of well-being noted after administration secondary to an increase in dopamine concentrations
 - iii. Excitatory effects in 10 % of patients
 - iv. Decreases cerebral metabolic oxygen consumption (CMRO₂), cerebral blood flow (CBF), intracranial pressure (ICP), and intraocular pressure.
 - v. Decreased cerebral perfusion pressure (CPP) if the propofol causes a significant drop in systemic blood pressure
 - b. Respiratory
 - i. Apnea with the induction dose
 - ii. Maintenance infusion causes a decreased tidal volume and increased respiratory rate
 - iii. Reduced ventilatory response to hypoxia and carbon dioxide
 - iv. Causes bronchodilation in patients with chronic obstructive pulmonary disease (COPD)
 - v. Does not inhibit hypoxic pulmonary vasoconstriction
 - c. Cardiovascular
 - i. Reduces systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MAP)
 - ii. Decreased cardiac output (CO), cardiac index (CI), stroke index (SI), and systemic vascular resistance (SVR)
 - iii. No reflex tachycardia
6. Uses
 - a. Induction of general anesthesia (dose 1–2.5 mg/kg IV)
 - b. Maintenance of general anesthesia (dose 50–150 µg/kg/min IV)
 - c. Sedation (25–75 µg/kg/min IV)

14. Intravenous Anesthetics

7. Special characteristics

- a. Anti-emetic (dose 10–20 mg IV, infusion 10 µg/kg/min IV)
- b. Do not use if the patient has an egg (yolk) allergy.
- c. Free radical scavenger

8. Salient features

- a. Rapid emergence from the initial bolus due to redistribution
- b. Rapid recovery following prolonged infusion
- c. No tolerance
- d. Safe in malignant hyperthermia-susceptible patient
- e. Most commonly used induction agent
- f. Does not affect corticosteroid synthesis or alter the normal response to adrenocorticotrophic hormone (ACTH) stimulation
- g. Does not trigger histamine release
- h. Subhypnotic dose can be used to treat pruritus induced by spinal opiates

9. Side effects

- a. Pain on injection
- b. Myoclonus (rare)
- c. Apnea (with a bolus)
- d. Reduction in blood pressure
- e. Anaphylactoid reactions
- f. Thrombophlebitis of the vein into which it is injected (rare)
- g. Propofol infusion syndrome: rare and often fatal syndrome described in critically ill children undergoing a prolonged propofol infusion at high doses
 - i. Can occur in adults.
 - ii. Symptoms
 - (1) Cardiomyopathy with acute cardiac failure
 - (2) Rhabdomyolysis
 - (3) Severe metabolic acidosis
 - (4) Hyperkalemia
 - (5) Hepatomegaly
 - (6) Lipemia
 - (7) Renal failure

Barbiturate (Thiopental and Methohexital)

1. Physical attributes

- a. Derivatives of barbituric acid
- b. Thiopental is stable for one week if refrigerated after reconstitution and methohexital is stable for up to six weeks.
- c. Pancuronium, vecuronium, atracurium, sufentanil, alfentanil, and midazolam can not be coadministered or mixed in a solution with barbiturates secondary to the resultant precipitate formation and IV line occlusion.

2. Metabolism

- a. Hepatic
- b. The metabolites that form are almost all inactive, water soluble, and excreted in the urine.

3. Mechanism of action

a. Mechanism of action is largely unknown

- a. Likely related to its action on the GABA receptors.
- b. Rapid onset of action
- c. Short duration

4. Pharmacokinetics

- a. Elimination half-life is 7–17 hours
- b. Clearance is 3–4 mL/kg/min
- c. Apparent volume of distribution at steady state is 1.5–3 L/kg
- d. Thiopental can accumulate in the tissues because of its affinity for fat, relatively large volume of distribution, and low rate of hepatic clearance.
- e. Rapid termination of the barbiturate effect (awakening) is due to the redistribution of the drug to the other tissues rather than metabolism.
- f. Decreased induction dose of the barbiturates is required for patients with shock, dehydration, severe anemia, burns, malnutrition, widespread malignant disease, uremia, and intestinal obstruction.

5. Effects

a. CNS

- i. Reduced CMRO₂, ATP consumption, CBF, and ICP
- ii. Slowing of the EEG
- iii. Overall CPP is maintained.
- iv. Protection from incomplete cerebral ischemia
- v. Clinical effects result in loss of consciousness and amnesia.
- vi. Barbiturates produce CNS effects when they cross the blood-brain-barrier.

(1) Drugs with high lipid solubility and a low degree of ionization cross the blood-brain barrier rapidly and produce a fast onset of action.

(2) Barbiturates such as thiopental and methohexital usually exist in the non-ionized form and are relatively lipid soluble.

b. Respiratory

- i. Respiratory depression
- ii. Transient apnea

c. Cardiovascular

- i. Reduced contractility
- ii. Increased heart rate (HR)
- iii. Decreased cardiac output
- iv. Cardiac index is unchanged or reduced.
- v. Cardiovascular depression
- vi. Mean arterial pressure is maintained or slightly reduced.
- vii. Cardiac output is significantly decreased when thiopental is given to hypovolemic patients.

d. Renal

- i. Reduced urine output due to increased anti-diuretic hormone (ADH)

6. Uses

- a. Premedication
- b. Induction agent
- c. Maintenance of anesthesia

14. Intravenous Anesthetics

- d. Antiepileptic
 - e. Cerebral protection to patients at risk for incomplete ischemia
7. Salient features of thiopental
- a. Induction dose: 3–4 mg/kg
 - b. Prompt onset of action (10–30 seconds)
 - c. IV maintenance infusion: 50–100 mg every 10–12 minutes
 - d. Smooth induction
 - e. Relatively rapid emergence after a single bolus
 - i. Prolonged effects with an infusion
 - f. Excellent hypnotic
 - g. Does not have analgesic properties
8. Salient features of methohexital
- a. Induction dose: 1–1.5 mg/kg
 - b. Onset of action 10–30 seconds
 - c. IV maintenance infusion: 20–40 mg every 4–7 minutes
 - d. Hypnotic
 - e. Not analgesic
 - f. Cleared more rapidly than thiopental
9. Side effects
- a. Garlic taste in the mouth with IV induction
 - b. Allergic reaction
 - c. Local tissue irritation
 - i. Tissue necrosis on extravasation
 - d. Induces liver enzyme production.
 - e. Possible laryngospasm and broncho-constriction
 - f. Porphyria
 - g. Accidental intra-arterial injection
 - i. Treatment
 - (1) Dilution of the drug by the administration of saline into the artery
 - (2) Heparinizaion to prevent thrombosis
 - (3) Brachial plexus block
10. Contraindications
- a. Patients with respiratory obstruction or an inadequate airway
 - b. Severe cardiovascular instability or shock
 - c. Status asthmaticus
 - d. Porphyria

Ketamine

1. Physical attributes
- a. Phencyclidine derivative
 - b. NMDA receptor antagonist
 - c. Partially water soluble
 - d. Lipid solubility 5–10 times that of thiopental
 - e. Low molecular weight
 - f. Near physiologic pH
2. Metabolism
- a. Hepatic microsomal enzymes
 - b. The metabolites are then conjugated into water-soluble glucuronide derivatives that are excreted in the urine.
3. Pharmacokinetics
- a. Elimination half-life is 2.5–2.8 hours

- b. Clearance is 12–17 mL/kg/min
 - c. Apparent volume of distribution at steady state is 3.1 L/kg
 - d. Rapidly crosses the blood-brain barrier
4. Effects
- a. CNS
 - i. Unconsciousness
 - ii. Dissociate anesthesia
 - iii. Increased CMRO₂
 - iv. Increased cerebral metabolism, CBF, and ICP
 - v. Maintain many reflexes such as corneal, cough, and swallow, but may still be at risk for aspiration.
 - vi. Profound analgesia
 - vii. Pupils dilate
 - viii. Nystagmus
 - ix. Increased muscle tone
 - b. Respiratory
 - i. Minimal effect on the central respiratory drive
 - ii. Unaltered response to CO₂
 - iii. Bronchodilation
 - c. Cardiovascular
 - i. Stimulates the sympathetic system
 - ii. Increased CO, HR, and BP
 - iii. Increased O₂ consumption
5. Uses
- a. Pre-medication: can be given IV, intramuscularly (IM), orally, nasally, or rectally
 - b. Sedation: 0.2–0.8 mg/kg IV over 2–3 minutes or 2–4 mg/kg IM
 - c. Induction: 0.5–2 mg/kg IV or 4–6 mg/kg IM
 - d. Maintenance of anesthesia: 30–90 µg/kg/min IV
 - i. Good choice for induction and maintenance
 - (1) Patients with reactive airway disorders
 - (2) Healthy trauma with unknown blood loss
 - (3) Cardiac tamponade
 - (4) Restrictive pericarditis
 - (5) Congenital heart disease
 - (6) Septic shock
 - (7) Hemodynamic compromise based on hypovolemia or cardiomyopathy.
 - (8) ASA class IV patients with respiratory and cardiovascular system disorders (excluding ischemic heart disease).
 - (9) Right-to-left cardiac shunt
 - (10) Remote location: dressing changes, radiation therapy, dental work
 - (11) Treatment of opioid resistance: chronic pain
6. Salient features
- a. Minimal hemodynamic changes
 - b. Profound analgesia and amnesia
 - c. The patient maintains the cough, corneal, and swallow reflexes.
 - d. Bronchodilation
 - e. Dissociative anesthesia
 - f. Post-operative analgesia

14. Intravenous Anesthetics

g. Patients who have been critically ill for a prolonged period may have exhausted their catecholamine stores and may be subject to the circulatory depressant effects of ketamine.

7. Side effects

- a.** Increased lacrimation and salivation which can further increase the risk of laryngospasm
- b.** Increased skeletal muscle tone
- c.** Increased ICP
- d.** Emergence reactions
 - i.** Excitement
 - ii.** Confusion
 - iii.** Illusions: treat with benzodiazepines
 - iv.** Vivid dreaming
- e.** Not recommended for
 - i.** Open eye injury: increases intraocular pressure
 - ii.** Significant coronary artery disease (CAD)
 - iii.** Vascular aneurysm
 - iv.** Schizophrenia
 - v.** Patient with increased ICP or an intracranial mass lesion

Etomidate

1. Structural attributes

- a.** Imidazole derivative
- b.** Water insoluble

2. Metabolism

- a.** Hepatic
- b.** The metabolites are then excreted by the kidneys (85%) and bile (13%)
- c.** A small amount (2%) is excreted without being metabolized.

3. Pharmacokinetics

- a.** Elimination half-life is 2.9–5.3 hours
- b.** Clearance is 18–25 mL/kg/min
- c.** Apparent volume of distribution at steady state is 2.5–4.5 L/kg

4. Effects

- a.** CNS
 - i.** Hypnosis
 - ii.** No analgesic activity
 - iii.** Reduced CBF and CMRO₂
 - iv.** CPP is maintained or increased.
 - v.** Reduced ICP and intraocular pressure
 - vi.** Cerebral protection
 - vii.** Induces increased EEG activity and myoclonic activity
- b.** Cardiovascular
 - i.** Hemodynamic stability
 - ii.** Almost no change in HR, MAP, mean pulmonary pressure, central venous pressure (CVP), stroke

volume (SV), cardiac index (CI), and systemic vascular resistance (SVR)

c. Respiratory

- i.** Minimal respiratory depression
- ii.** May not totally ablate the sympathetic response to direct laryngoscopy and intubation

5. Uses

a. Patients with

- i.** History of a cerebral vascular accident
- ii.** CAD
- iii.** Reactive airway disease
- iv.** Increased ICP
- v.** Aortic surgery
- vi.** Cardioversion
- vii.** Neurosurgical patients
- viii.** Trauma patients
- ix.** Electroconvulsive therapy (ECT)
- x.** Retrobulbar block
- xi.** Can induce convulsions in epileptic patients, and therefore can be used for mapping of the seizure foci.
- xii.** Superior to propofol for evoked potential monitoring

b. Sedation: 5–10 µg/kg/min IV

c. Induction: 0.2–0.6 mg/kg IV

- i.** One arm-brain circulation time
 - ii.** Duration of anesthesia is about 100 seconds of loss of consciousness for each 0.1 mg/kg administered
- d.** Maintenance: 10 µg/kg/min IV with N₂O and an opiate

6. Salient features

- a.** Hemodynamic stability
- b.** Minimal respiratory depression
- c.** Cerebral protection
- d.** Rapid recovery after infusion
- e.** Short elimination time

7. Side effects

- a.** Suppresses the adreno-cortical function by inhibition of the enzymes 11 beta-hydroxylase and 17 alpha-hydroxylase.
 - i.** Inhibition of cortisol and aldosterone synthesis
- b.** Pain on injection
- c.** Superficial thrombophlebitis
- d.** Myoclonus
- e.** Nausea and vomiting

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 340–6.
2. Miller RD, Cucchiara RF, Miller ED, et al. *Anesthesia*, 5th ed. New York: Churchill Livingstone, 2000, pp 239–56.

15. Inhalational Anesthetics^{1,2}

Andrew M. Bauer, MD and Jessica A. Lovich-Sapola, MD

CLINICAL ISSUES

Factors that Influence the Choice of an Inhaled Agent

1. Speed of induction and emergence
2. Suitability for inhalational induction
3. Effects on physiology: cardiovascular, intracranial pressure
4. Coexisting diseases: obesity, pneumothorax
5. Cost

Isoflurane

1. Facts
 - a. Low minimum alveolar concentration (MAC): 1.2
 - b. Systemic and coronary vasodilator
2. Advantages
 - a. Lowest cost of the halogenated anesthetics
 - b. Nonflammable
 - c. Minimal cardiac depression: cardiac output is maintained by a rise in heart rate.
 - d. Reverses bronchospasm
 - e. Skeletal muscle relaxant
3. Disadvantages
 - a. High blood and lipid solubility
 - i. Slower onset and emergence
 - ii. May be more of an issue in morbidly obese patients and long cases.
 - b. Pungent odor may not be well tolerated for inhalational inductions
 - c. May produce tachycardia
 - d. Sometimes avoided in patients with significant coronary artery disease because of the theoretical risk of coronary steal syndrome: dilation of the normal coronary arteries with resultant diversion of blood away from the fixed stenotic lesion.
 - e. Not tolerated well in patients who are severely hypovolemic, due to its vasodilating effects.
 - f. Malignant hyperthermia trigger

Sevoflurane

1. Facts
 - a. Relatively low MAC: 2.0
 - b. Intermediate cost between isoflurane and desflurane
 - c. Low blood solubility but high lipid solubility

2. Advantages

- a. More rapid induction than isoflurane
- b. Sweet relatively non-pungent odor with minimal airway irritation
 - i. Best choice for inhalational induction
 - ii. Well suited to pediatric anesthesia
 - iii. Reverses bronchospasm: potent bronchodilator
- c. Faster emergence when compared to isoflurane
- d. Less decrease in systemic vascular resistance and arterial blood pressure when compared to isoflurane and desflurane
- e. Skeletal muscle relaxant

3. Disadvantages

- a. May start to accumulate over a long case, especially in an obese patient.
- b. Potential for emergence delirium
 - i. Some clinicians will change to isoflurane after induction.
- c. Cardiac output is not well-maintained secondary to the minimal rise in heart rate compared to isoflurane and desflurane.
 - i. Mild myocardial depressant
- d. May prolong QT.
- e. Contraindicated in patients with severe hypovolemia and intracranial hypertension.
- f. Malignant hyperthermia trigger
- g. Potential nephrotoxicity if given at flow rates less than 2 liters
 - i. Formation of compound A
- h. Can form carbon monoxide during exposure to dry CO₂ absorbents, and an exothermic reaction has occurred and resulted in fires.

Desflurane

1. Facts

- a. High MAC: 6.0

2. Advantages

- a. Very low solubility in blood and lipid
 - i. Fast induction and emergence
 - ii. Less residual anesthetic in peripheral tissues in obese patients and after long cases
- b. Near-absent metabolism
- c. Skeletal muscle relaxant

15. Inhalational Anesthetics

3. Disadvantages

- a. Expensive
- b. Most pungent: may cause coughing, bronchospasm, laryngospasm, salivation, and breath holding.
 - i. Unsuitable for inhalational inductions
 - ii. Not a good choice for pediatric anesthesia
 - iii. Not suitable for patients with significant pulmonary disease
- c. A rapid increase in the desflurane concentration level or high levels alone can result in tachycardia, hypertension, myocardial ischemia, and elevated catecholamine levels.
- d. Malignant hyperthermia trigger
- e. Contraindicated in patients with severe hypovolemia and intracranial hypertension
- f. Requires a heated, pressurized vaporizer.
 - i. Requires electrical power.
 - ii. Requires more servicing.
- g. Degrades to form carbon monoxide in extremely dry CO₂ absorbers to a greater extent than the other volatile anesthetics.

Nitrous Oxide

1. Facts

- a. Only inorganic anesthetic gas
- b. Colorless
- c. Essentially odorless
- d. Gas at room temperature
- e. Stored as a liquid under pressure

2. Advantages

- a. Inexpensive
- b. Very low solubility in blood and lipids
 - i. Allows the use of less halogenated agent when used in combination.
- c. Rapid induction and recovery
- d. Can speed induction of other anesthetics
 - i. Concentration effect
 - (1) Combination of low solubility and high inhaled partial pressures
 - (2) Increases the blood concentration of nitrous oxide.
 - (3) Speeds induction.
 - (4) Diffusion hypoxia
 - (a) Occurs during emergence if low FiO₂ used
 - (b) Opposite of concentration effect: rapid diffusion of nitrous oxide from the blood to the alveoli decreases the alveolar PO₂.

- ii. Second gas effect when used with a volatile agent
 - (1) Same principle as concentration effect
 - (2) Rapid diffusion of nitrous oxide from the alveoli to the blood increases the alveolar concentration of the volatile agent.
- e. Not a trigger for malignant hyperthermia; therefore, may be used as an adjunct to intravenous anesthesia in these patients.
- f. Nonexplosive and nonflammable
- g. Minimal cardiovascular effects
- h. Minimal change in minute ventilation: increased respiratory rate and decreased tidal volume
- i. Analgesic effect

3. Disadvantages

- a. Very high MAC (104%): cannot be used as sole agent.
- b. Will expand gas-filled spaces.
 - i. Do not use in patients with a pneumothorax, pneumocephalus, pulmonary hypertension, or intestinal obstruction.
 - ii. Do not use in certain eye or inner ear surgeries.
 - (1) Discuss with the surgeon if it is appropriate to use.
 - iii. Use clinical judgment for laparoscopy or bowel surgery.
 - iv. Do not use in cases that are high risk for a venous air embolism.
- c. Capable of supporting combustion
- d. Increased risk of post-operative nausea and vomiting.
- e. Does not produce skeletal muscle relaxation like the other volatile anesthetics.
- f. Potential toxic effect on cell function
 - i. Inactivation of vitamin B12
 - ii. Effect on embryonic development

TKO: It is important not only to choose an anesthetic but also to be able to explain why you chose it on the day of your exam!!!!

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 395–6.
2. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 3rd ed. New York: Lange Medical Books, McGraw-Hill, 2002, pp 137–46.

PART TWO

**Anesthetic-Related Critical
Events and Information**

16. Hypoxemia and Hypercarbia^{1,2}

Jessica A. Lovich-Sapola, MD

HYPOXEMIA

SAMPLE CASE

A 47-year-old female presents for an open reduction and internal fixation (ORIF) of the left femur after an automobile accident. Her past medical history is significant for hypertension. She is a 1 pack per day smoker for the past 20 years. The patient's oxygen saturation is 93% on 2 L nasal cannula. What pre-operative tests would you like? Why? How will her low oxygen saturation affect your anesthetic plan?

CLINICAL ISSUES

Definition of Hypoxemia: deficiency in the concentration of dissolved oxygen in the arterial blood

Definition of Hypoxia: abnormally low oxygen availability to the body, or a specific tissue/organ

Classification of Hypoxemia

Pathophysiological Mechanism of Classification

1. Decreased inspired oxygen
 - a. Mechanical failure of the anesthesia apparatus to deliver oxygen to the patient
 - b. Disconnection from the oxygen supply is the most common form of mechanical failure. This usually occurs at the junction of the endotracheal tube and the elbow connector of the circuit.
 - c. Empty or depleted oxygen cylinder
 - d. Gas pressure failure
 - e. Crossing of pipelines
 - f. Crossing of tanks
 - g. Fracture or sticking of flow meters
 - h. Transposition of rotameter tubes
 - i. Improper oxygen sensor calibration
2. Hypoventilation
 - a. Esophageal intubation
 - b. Endotracheal tube kinking, blockage with secretions, and herniated or ruptured cuff all lead to increased airway resistance and hypoventilation.
 - c. Right main-stem intubation

- d. Respiratory depression or failure secondary to anesthetic medications and paralysis
- e. Ventilator failure
3. Impaired diffusion
4. Ventilation-perfusion (V/Q) mismatch
5. Right-to-left intracardiac shunt
 - a. Patent foramen ovale
 - b. Tetralogy of Fallot
6. Intrapulmonary derangements

Structural-Anatomic Classification

1. Alveoli
 - a. Pulmonary edema
 - b. Acute lung injury/pulmonary contusion
 - c. Adult respiratory distress syndrome (ARDS)
 - d. Pulmonary hemorrhage
 - e. Pneumonia
2. Interstitium
 - a. Pulmonary fibrosis
 - b. Viral pneumonia
 - c. Allergic alveolitis
3. Heart and pulmonary vasculature
 - a. Pulmonary embolism
 - b. Intracardiac or intrapulmonary shunt
 - c. Congestive heart failure (CHF)
4. Airways
 - a. Asthma
 - b. Chronic obstructive pulmonary disease (COPD)
 - c. Mucus plugging
 - d. Right main-stem intubation
5. Pleura
 - a. Pneumothorax
 - b. Pleural effusion

Hypoxemia from V/Q mismatch is responsive to supplemental oxygen, while hypoxemia from right-to-left shunt is not responsive to supplemental oxygen. The cause can then be easily determined with the application of supplemental oxygen. Hypoventilation can be ruled out if the patient is not hypercapnic or acidemic. Impaired diffusion is usually a rare cause of hypoxemia because the red blood cell has plenty of time for the diffusion of oxygen even in the presence of intrinsic lung disease. Most patients with acute hypoxic respiratory failure have a combination of V/Q mismatch and right-to-left shunt.



KO TREATMENT PLAN

Pre-operative

1. When evaluating this patient it is important to think about the full differential diagnosis prior to the induction of anesthesia.
2. Think about the possible chronic causes of hypoxia, including COPD.
3. Think about the possible acute causes, including a pulmonary/cardiac injury secondary to the accident.
4. Also, consider over-sedation as the cause of hypoxia.
5. Would her saturations improve with increased oxygen and albuterol, or does she need a chest tube?
6. Since this is a trauma, you should work quickly. Listen to cardiac and breath sounds. Access her vital signs. Is she responsive? Where does she have pain?
7. Get a portable chest X-ray if it was not already done in the emergency room.
8. Consider a baseline ABG.
9. An EKG can be done to help rule out a cardiac cause for the hypoxemia.

Intra-operative

1. Apply the standard ASA monitors.
2. Preoxygenate the patient.
3. Apply in-line stabilization.
4. Assuming the airway is normal, perform a rapid sequence induction using propofol and succinylcholine. (Etomidate, thiopental, and ketamine can also be used. It does not matter which induction agent you use as long as it is safe, you understand the proper dosing, and you can provide reasons for why you are using it.)
5. Apply cricoid pressure during the induction.
6. Intubate.
7. Listen for bilateral breath sounds and verify end-tidal carbon dioxide (ETCO₂) prior to releasing cricoid pressure.
8. Maintain anesthesia with a combination of oxygen/sevo-flurane. I would use sevoflurane because it is less irritating to the airways, but you can use any volatile anesthetic; just have a reason for your choice.
9. I would not use nitrous oxide secondary to her hypoxia and the risk of potentially expanding a small pneumothorax induced by the trauma.
10. Obtain an arterial blood gas.

Intra-operative Acute Hypoxia

1. Check the color of the patient.
2. Check for a pulse.
3. Check all vital signs.
4. Check for ETCO₂.
5. Take the patient off the ventilator and hand bag with 100% O₂.
6. Call for help.

7. Check the O₂ monitor, peak airway pressure, and capnograph waveform.
8. Listen to the chest for bilateral breath sounds. Note chest rise.
 - a. Unilateral breath sounds: endotracheal tube (ETT) is too deep, pneumothorax, tension pneumothorax
9. Evaluate the ETT. Rule out kinking, mucus plug, and herniated cuff. Pass the suction catheter or fiber optic scope. Check for proper placement and position.
10. Listen to the chest for wheezing. Give aerosolized albuterol for wheezing.
11. If you think that the patient has bronchospasm, deepen the anesthetic.
12. If deepening the anesthetic does not treat the bronchospasm, give IV epinephrine.
13. Order a chest X-ray.

Tension Pneumothorax

1. Presentation
 - a. Unilateral absence of breath sounds
 - b. Tracheal deviation
 - c. Unexplainable hypotension
 - d. Distended neck veins
2. Treatment
 - a. Find the second intercostal space on the side of the pneumothorax.
 - b. Find the midclavicular line.
 - c. Insert a 14 gauge angiocatheter into this space over the top of the rib.
 - d. Listen for a decompressive air rush.
 - e. Leave the angiocatheter in place.
 - f. Place a chest tube.

Post-operative

1. Plan on extubating if she meets the extubation criteria.
2. Know that a pulmonary contusion may not be evident early on after the injury but may develop a few hours later.
3. A normal chest X-ray pre-operatively does not rule out pulmonary injury. If the patient had worsening oxygenation during the surgery, a repeat chest X-ray may be warranted.

HYPERCARBIA

CLINICAL ISSUES

Definition of Hypercarbia

1. Also known as hypercapnia
2. Too much carbon dioxide (CO₂) in the blood

Causes of Hypercarbia

1. Increased production of CO₂
 - a. Tourniquet release
 - b. Aortic cross-clamp release

- c. Malignant hyperthermia
 - d. Sepsis
 - e. Thyrotoxicosis
 - f. Fever
2. Decreased removal of CO₂
 - a. Hypoventilation
 - b. Airway obstruction
 - c. Increased dead space
 3. Rebreathing of CO₂ due to mechanical malfunction
 - a. Faulty valves

- b. Exhausted CO₂ absorber
 - c. Low fresh gas flows
4. Iatrogenic
 - a. Sodium bicarbonate administration
 - b. Increased CO₂ during laparoscopic procedures

BIBLIOGRAPHY

1. Mason RJ, Murray JF, Broaddus VC, et al. *Murray & Nadel's Textbook of Respiratory Medicine*, 4th ed. Philadelphia: Elsevier Saunders, 2005, chapter 85.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 714–8.

17. Indications for Intubation and Extubation^{1,2,3}

Jessica A. Lovich-Sapola, MD

INDICATIONS FOR INTUBATION

SAMPLE CASE

You are called to the medical intensive care unit to intubate a patient. He weighs 520 pounds. He has obstructive sleep apnea and pneumonia. He has no allergies. He is on bi-pap and his oxygen saturation is 87%. The patient is refusing to be intubated.

Labs: pH 7.18, PCO₂ 70 mm Hg, PO₂ 60 mm Hg, oxygen saturation is 93.7%, base excess 8.4 mmol/L, and bicarbonate 43.6 mmol/L

CLINICAL ISSUES

Indications for Intubation

1. Mechanical function
 - a. Respiratory rate (RR) >35
 - b. Vital capacity (VC) <15 mL/kg (adult)
 - c. Vital capacity (VC) <10 mL/kg (child)
 - d. Negative inspiratory force (NIF) less than 20–25 cm H₂O
2. Gas exchange function
 - a. PaO₂ <60 on a FiO₂ of 50%
 - b. A-a gradient ≥ 350 on FiO₂ of 100%
 - c. PaCO₂ ≥ 55 (unless chronically elevated CO₂)
 - d. Dead space ventilation/tidal volume (Vd/Vt) ratio ≥0.6 (normal = 0.3)

3. Unstable vital signs
4. Inability of the patient to protect his airway secondary to agitation, airway burns, facial trauma, or neurologic injury.



KO TREATMENT PLAN

Despite the fact that this patient is refusing to be intubated, he is clearly hypercarbic and cannot be expected to make appropriate decisions for his medical care. An awake fiber optic intubation, which would be my first choice, is out of the question secondary to the fact that he is refusing the intubation and is not cooperative. He will have to be intubated with some sedation. Call the ear, nose, and throat (ENT) physicians for backup, in case he requires an emergent tracheotomy. Verify suction and a free-flowing intravenous (IV) line. Position the patient in the bed, ensuring a good sniffing position. Make sure all emergency airway equipment is immediately available, including the fiber optic scope. Use 4% aerosolized lidocaine spray. Minimal IV sedation should be given, using midazolam and etomidate. (Again, any drug can be used, just be able to justify its use.) Sedation should be done slowly, since he is probably very tired from the work of breathing. An apneic patient could be a dead patient. Try to maintain spontaneous respirations. A Glidescope is a good option for this patient. With the 4% lidocaine topicalization, the Glidescope is less stimulating to the patient than the usual direct laryngoscopy. Once the endotracheal tube is in place, verify placement with auscultation and end-tidal CO₂.

INDICATIONS FOR TRACHEAL EXTUBATION

CLINICAL ISSUES

Indication for Extubation

1. Subjective criteria
 - a. Resolution of acute disease
 - b. Adequate cough
 - c. Patient should be awake, alert, and following commands.
 - d. Cooperative
 - e. Glasgow Coma Scale (GCS) ≥ 13
 - f. No continuous sedation infusions
 - g. Sustained hand grip
 - h. Sustained head lift > 5 seconds
 - i. Able to tolerate spontaneous ventilation without excessive tachypnea, tachycardia, or obvious respiratory distress.
 - j. Acceptable electrolytes
 - k. Appropriate gag reflex and cough
 - l. Able to protect his airway from aspiration.
 - m. Clear oropharynx: no active bleeding or secretions
 - n. Adequate pain control
 - o. Minimal end-expiratory concentration of inhaled anesthetics
2. Objective criteria
 - a. Vital signs
 - i. Respiratory rate < 30 – 35 breaths/min
 - ii. Stable blood pressure with minimal or no inotropic support
 - iii. Heart rate ≤ 140 beats/min
 - iv. Afebrile (temperature < 38 °C)
 - b. Gas exchange function
 - i. Arterial blood gas (ABG) on 40% FiO₂ and PEEP ≤ 5 – 10 cm H₂O
 - (1) PaO₂ ≥ 60 mm Hg
 - (2) PaCO₂ < 55
 - ii. PaO₂/FiO₂ (P/F ratio) ≥ 150 – 300 mm Hg
 - iii. Alveolar-arterial PaO₂ gradient < 350 mm Hg on 100% oxygen
 - iv. Maintenance of a normal pH (> 7.30)
 - c. Mechanical function
 - i. Forced vital capacity (FVC) > 10 – 15 mL/kg
 - ii. Forced expiratory volume in 1 second (FEV₁) > 10 mL/kg
 - iii. Tidal volume (V_T) ≥ 4 – 6 mL/kg
 - iv. Negative inspiratory force (NIF) > 20 cm H₂O
 - v. Vital capacity (VC) ≥ 15 mL/kg
 - vi. Deadspace ventilation/tidal volume (V_D/V_T) ratio < 0.6
 - vii. Rapid shallow breathing index (RSBI) (f/Vt) ≤ 60 – 100 breaths/min
 - d. Adequate hemoglobin (≥ 8 – 10 g/dL)
 - e. No significant respiratory acidosis

BIBLIOGRAPHY

1. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease* 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 218–9.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1469–71, 2483.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 617.

18. The Difficult Airway^{1,2,3,4,5,6}

Jeffrey A. Brown, DO and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 53-year-old male is emergently brought to the operating room for repair of a bleeding gastric ulcer. The patient is sedated from a previous upper endoscopy performed to diagnose the bleeding. The patient weighs 133 kg, has a bull neck, and has a known difficult airway. The patient's heart rate is 133 and his blood pressure is 90/60 mm Hg. He is breathing spontaneously and has an oxygen saturation of 94% on room air. The surgeon is waiting. What is your plan?

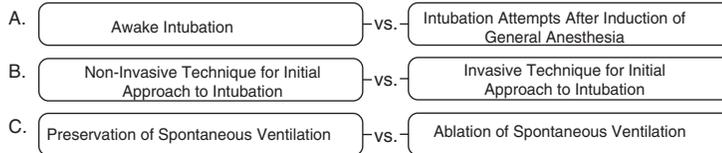
CLINICAL ISSUES

Definition of a Difficult Airway

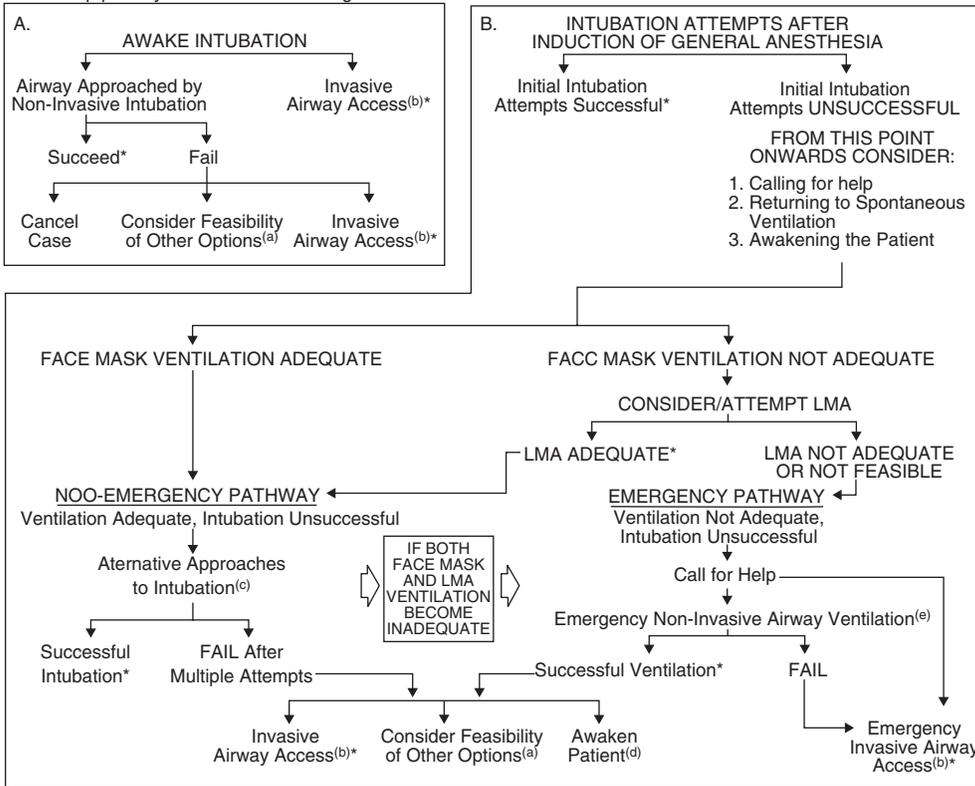
1. An airway in which a conventionally trained anesthesiologist experiences difficulty with direct laryngoscopy (DL) or mask ventilation or both
2. The difficult airway represents an interaction between the skill of the practitioner, the clinical setting, and patient factors.¹

DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
 - A. Difficult Ventilation
 - B. Difficult Intubation
 - C. Difficult with Patient Cooperation or Consent
 - D. Difficult Tracheostomy
2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management
3. Consider the relative merits and feasibility of basic management choices:



4. Develop primary and alternative strategies:



* Confirmed ventilation, tracheal intubation, LMA placement with exhaled CO₂

a. Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

b. Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy.

c. Alternative non-invasive approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

c. Consider re-preparation of the patient for awake intubation or canceling surgery.

e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal jet ventilation.

Figure 18.1. ASA: difficult airway algorithm.
 Source: Reprinted from the American Society of Anesthesiologists Task Force Practice Guidelines for Management of the Difficult Airway: An Updated Report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology*, 2003; 98(5), 1273. With permission from the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

18. The Difficult Airway

3. There are two key branch points regarding the ASA algorithm for difficult airways: anticipated and unanticipated.

Incidence

1. "Cannot ventilate, cannot intubate" situations occur in as many as 1 in 5,000 anesthetics.³
2. Difficult direct laryngoscopy occurs in about 2–9% of general anesthetics.³
3. Failed intubations occur in 0.13–0.3% of all general anesthetics.³

Conditions That Predispose a Patient to Difficult Mask Ventilation

1. Beard
2. Obesity
3. Lack of teeth
4. Obstructive sleep apnea
5. Piercing of the tongue, lip, cheek, and chin.

Pathological States That Predispose Patients to Difficult Intubations

1. Congenital
 - a. Pierre-Robin syndrome: micrognathia, macroglossia, cleft palate
 - b. Treacher-Collins syndrome: mandibular hypoplasia
 - c. Down's syndrome: macroglossia, atlantoaxial instability
 - d. Kippel-Feil syndrome: restricted neck movement secondary to cervical vertebrae fusion.
2. Infection
 - a. Croup
 - b. Ludwig's angina
 - c. Abscess
3. Arthritis
 - a. Rheumatoid
 - b. Ankylosing spondylitis
4. Benign tumors
 - a. Lipoma
 - b. Adenoma
 - c. Goiter
5. Malignancy of the airway
6. Injury
 - a. Facial
 - b. Cervical
 - c. Laryngeal/tracheal
 - d. Burns
7. Diabetes
8. Scleroderma
9. Obesity
10. Pregnancy
11. Acromegaly
12. Anatomic abnormalities
 - a. Micrognathia
 - b. Limited jaw motion

Criteria Commonly Used to Predict a Difficult Airway

1. History
 - a. History of a previous difficult intubation
 - b. Burns
 - c. Edema
 - d. Bleeding
 - e. Airway stenosis
 - f. Gastroesophageal reflux
 - g. Poor dentition
 - h. Radiation treatments
2. Physical Exam
 - a. General
 - i. Massive obesity
 - ii. Cervical collar
 - iii. Traction device
 - iv. External trauma
 - v. Current respiratory difficulty/stridor
 - b. Patency of nares
 - i. Masses
 - ii. Deviated nasal septum
 - c. Mouth opening: temporomandibular joint
 - i. Less than two finger breadths (3–4 cm)
 - d. Teeth
 - i. Prominent incisors
 - ii. Overbite
 - iii. Loose teeth
 - e. Palate
 - i. High arch
 - ii. Narrow mouth
 - f. Tongue
 - i. Macroglossia
 - g. Prognathism
 - i. Increased risk of a difficult intubation if the patient cannot protrude the lower jaw beyond the upper incisors.
 - h. Thyromental distance less than 6 cm
 - i. Neck
 - i. Short and thick
 - ii. Limited neck extension, less than 35 degrees
 - iii. Flexion less than 80 degrees

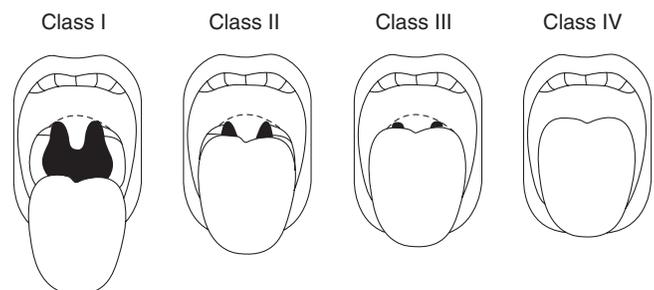


Figure 18.2. Mallampati score.

Source: This figure was published in *Evidence-Based Practice of Anesthesiology, 1st Edition*. Lee A. Fleisher, Chapter 8, Figure 8.2. Copyright Elsevier (2004). Reprinted with permission.

18. The Difficult Airway

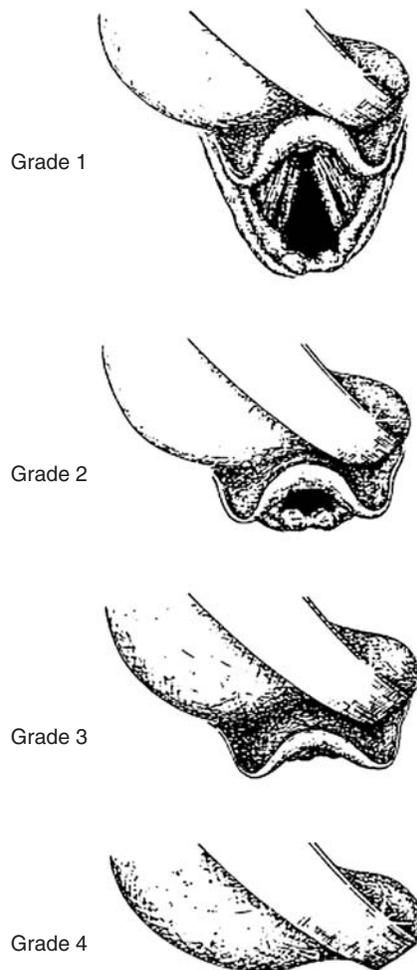


Figure 18.3. Laryngoscopic grades.

Source: This figure was published in *Benumoff's Airway Management, 2nd Edition*. Carin A. Hagberg, Chapter 8, Figure 8.3. Copyright Elsevier (2007). Reprinted with permission.

3. Specific Tests

- a. Mallampati score of 3 or higher
- b. Laryngoscopic grades III or IV
- c. Radiographic assessment
 - i. Lateral cervical films
 - ii. CT scan: trachea and mediastinal tumors
 - iii. Flow-volume loop test to evaluate intra- and extra-thoracic obstructive lesions
- d. Diabetic predictors
 - i. Positive prayer sign: gap between palms

TKO: No single test can provide a high index of sensitivity and specificity. You must use a combination of tests. However, a difficult airway may not always be predicted. You must always be prepared for an unexpected difficult airway.

Treatment Techniques for a Difficult Airway

1. Laryngeal manipulation
 - a. Backward pressure

- b. Upward pressure
 - c. Rightward pressure
2. Stylet
3. Reposition the patient.
4. Change laryngoscope blades.
5. Gum elastic bougie
6. Intubating laryngeal mask airway (LMA)
 - a. The LMA has been found to provide rescue ventilation in about 90% of unanticipated difficult intubation/ventilation scenarios.³
7. Flexible or rigid fiberscope
8. Nasotracheal intubation
9. Blind nasal intubation: successful in only 50% of cases, and has an increased risk of bleeding and trauma.
10. Light wand
11. Combitube: risk of esophageal rupture
12. Transtracheal jet ventilation (TTJV)
13. Tracheostomy



KO TREATMENT PLAN

Anticipated Difficult Airways

1. Keep the patient breathing spontaneously.
2. Most practitioners would choose an awake fiber optic intubation.

Unanticipated Difficult Airway

1. The algorithm path branches at two key points: either mask ventilation is successful or it is not.
2. If mask ventilation is successful, the airway may still be urgent but is no longer emergent and options for securing it can be quickly implemented.
3. If both DL and mask ventilation are not successful, the ASA's algorithm is very clear that the next step is placement of an LMA.
4. If this provides adequate ventilation then go back on the urgent, non-emergent pathway.
5. If the LMA placement is not successful, alternative means for ventilation should quickly be instituted, or if possible, the patient should be awakened.
6. If waking the patient up is not possible, a non-invasive airway must be established via transtracheal jet ventilation, cricothyroidotomy, combitube, or other means.
7. If these methods fail, an invasive airway must be obtained emergently.
8. There are rare extreme circumstances where one might consider invasive methods first, such as severe craniofacial abnormalities from a trauma patient with blood and edema obscuring the airway.
 - a. In these instances, a surgical airway might be the first option.
9. Some practitioners would choose retrograde intubation over a guide wire at this point, but Barash is quick to point out that most anesthesiologists are not skilled in this technique.

It takes a few minutes, and should therefore not be attempted in a hypoxic patient.

10. Some texts state that secretions and blood in the airway may prevent successful fiber optic intubation.

- a. If these secretions are too great and visualization is not possible, then the key is whether the patient is hypoxic or not.
- b. If there is time, a retrograde intubation may be performed. If not, then an invasive airway must be obtained.



SAMPLE CASE KO TREATMENT PLAN

In the sample case above there are several issues to consider. The patient has a known difficult airway, is at risk for aspiration, and may not be cooperative with an awake fiber optic intubation. The patient is breathing spontaneously. He should remain breathing spontaneously until his airway is secured. If the sedation given for the endoscopy is not too great, and the secretions and blood in the airway are easily dealt with, then an awake fiber optic intubation should be the first choice. An awake intubation after topicalization with the glidescope may be another option. If these methods cannot be performed, the choices are limited to invasive means to secure the airway, (i.e., awake tracheostomy). In any case, backup methods such as an LMA should be immediately available.

TKO

1. You will almost always have a difficult airway scenario on your oral board examination: be prepared!!!!

2. The LMA has revolutionized airway management, both difficult and elective. You can bet that the board expects you to know its role in the difficult airway.

3. Fiber optic intubations are most useful in an elective setting, not as an emergency backup!

4. Airway trumps everything else! Remember the ABC's! Whatever methods you choose, once you are on the difficult airway path, keep the patient breathing spontaneously until the airway is secured.

5. Call for help early. A wise anesthesiologist recognizes that a colleague who is skilled in the performance of a surgical airway is the one best suited to do it. However, if pushed, do not allow life-threatening or brain-threatening hypoxia to stand between you and the knife.

BIBLIOGRAPHY

1. Practice guidelines for management of the difficult airway: An updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology* 2003;98(5):1269-77.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 632.
3. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, chapters 43 and 62.
4. Gupta S, Sharma R, Jain D. Airway assessment: Predictors of difficult airway. *Indian J Anesth.* 2005; 49(4): 257-62.
5. Fleisher LE. *Evidence-Based Practice of Anesthesiology*. Philadelphia: Saunders, 2004, chapter 8.
6. Hagberg CA. *Benumof's Airway Management*, 2nd ed. Philadelphia: Mosby Elsevier, 2007, chapters 8, 31, and 37.

19. Dyspnea and Tachypnea^{1,2}

Jessica A. Lovich-Sapola, MD

DYSYPNEA¹

CLINICAL ISSUES

Definition of Dyspnea

1. The clinical term for the symptom of breathlessness¹
2. It is a symptom that alerts individuals when they are in danger of receiving inadequate ventilation.¹
3. It is the subjective experience of breathing discomfort that comprises qualitatively distinct sensations that vary in intensity.¹

4. The experience derives from interactions among physical, psychological, social, and environmental factors.¹

Mechanism of Dyspnea

1. Urge to breathe
2. Sense of excessive effort
3. Increased central respiratory drive secondary to hypoxia or hypercapnia
4. Altered central perception
5. Augmented requirement for the respiratory drive to overcome mechanical weakness

19. Dyspnea and Tachypnea

Differential Diagnosis of Dyspnea

1. Mechanical interference with ventilation
 - a. Obstruction to flow
 - i. Asthma
 - ii. Emphysema
 - iii. Bronchitis
 - iv. Tracheal or laryngeal stenosis
 - v. Laryngomalacia
 - vi. Bronchomalacia
 - b. Resistance to expansion of the lung
 - i. Interstitial fibrosis
 - ii. Restrictive lung disease
 - c. Resistance to chest wall expansion
 - i. Pleural thickening
 - ii. Kyphoscoliosis
 - iii. Obesity
 - iv. Abdominal mass (tumor/pregnancy)
2. Weakness of the respiratory pump
 - a. Absolute
 - i. Poliomyelitis
 - ii. Neuromuscular disease (Guillain-Barré syndrome, muscular dystrophy, systemic lupus, and hyperthyroidism)
 - b. Relative
 - i. Hyperinflation (asthma, emphysema)
 - ii. Pleural effusion
 - iii. Pneumothorax
3. Increased respiratory drive
 - a. Hypoxemia
 - b. Metabolic acidosis
 - c. Stimulation of the intrapulmonary receptors: infiltrative lung disease, pulmonary hypertension, and edema)
4. Psychological dysfunction
 - a. Anxiety
 - b. Depression



KO TREATMENT PLAN

1. Reduce the sense of effort and improve respiratory muscle function.
2. Decrease respiratory drive.
3. Alter central nervous system function.
4. Exercise training and pulmonary rehabilitation

TACHYPNEA²

CLINICAL ISSUES

Definition of Tachypnea

1. Increase in respiratory rate

2. It is characterized by rapid breathing and is not identical with hyperventilation.

3. Tachypnea may be necessary for sufficient gas-exchange within the body - for example, after exercise, in which case it is not hyperventilation.

Definition of Hyperventilation

1. Breathing faster or deeper than necessary
2. Not precipitated by a lack of oxygen
3. Often results in a decrease in the blood carbon dioxide to levels below normal
4. An example would be fast breathing during a panic attack.

Causes of Tachypnea

1. Airway obstruction: extra- or intra-thoracic
2. Anxiety/pain
3. Acute circulatory failure: congestive heart failure, cardiomyopathy
4. Intrapulmonary problems
 - a. Chronic obstructive pulmonary disease (COPD)
 - b. Restrictive lung disease/fibrosis/sarcoidosis
 - c. Asthma
 - d. Aspiration
 - e. Atelectasis
 - f. Pulmonary edema, pneumothorax, pulmonary embolus, and pulmonary hypertension
5. Disease of chest wall or respiratory muscles
 - a. Poliomyelitis
 - b. Myasthenia gravis
6. Systemic disease
 - a. Sepsis
 - b. Acidosis
 - c. Hypoxia
 - d. Shock
 - e. Fever
 - f. Malignant hyperthermia
 - g. Infection
 - h. Hypophosphatemia
7. Excess post-exercise oxygen consumption
8. Hypermetabolic state
9. Hyperthyroid

BIBLIOGRAPHY

1. Mason RJ, Murray JF, Broaddus VC, et al. *Murray & Nadel's Textbook of Respiratory Medicine*, 4th ed. Philadelphia: Elsevier Saunders, 2005, chapter 28.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, p 1087.

20. Wheezing^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 15-year-old female presents emergently to the operating room after falling off a third-story balcony. She is scheduled to have an open reduction and internal fixation of her femur fracture. She has a past medical history significant for poorly controlled asthma. She is currently wheezing. She also complains of significant chest pain from the fall. What tests would you like? How should you treat her wheezing? How does her wheezing affect your anesthetic plan?

CLINICAL ISSUES

Definitions

1. Wheezing
 - a. Expiratory sound produced by turbulent gas flow through narrowed airways
 - b. Continuous, coarse, whistling sound
 - c. Due to narrowing or obstruction in the respiratory tree
2. Acute wheezing
 - a. Medical emergency
 - b. Postpone elective surgery.
3. Chronic wheezing
 - a. Often seen in patients with chronic obstructive pulmonary disease (COPD)
 - b. Gas flow obstruction secondary to smooth muscle contraction, accumulation of secretions, and mucosal edema²
4. Bronchospasm
 - a. Sudden constriction of the bronchioles, triggered by an irritating stimulus
 - b. Causes difficulty breathing.
 - c. The resulting bronchiole constriction and inflammation causes narrowing of the airways and an increase in mucus production.
 - d. This is a serious potential complication of placing an endotracheal tube (ETT) during general anesthesia as the ETT acts as an irritating stimulus.
5. Asthma
 - a. Characterized by recurrent episodes of airway obstruction and chronic inflammation
 - b. There is an exaggerated bronchoconstrictor response to stimuli that would have little or no effect on a nonasthmatic patient.
 - c. Inflammation of the airways

Differential Diagnosis of Wheezing

1. Bronchospasm
2. Asthma
3. Chronic obstructive pulmonary disease (COPD): chronic bronchitis
4. Tracheobronchitis
5. Restrictive pulmonary disease: sarcoidosis
6. Rheumatoid arthritis-associated bronchiolitis
7. Extrinsic compression: thoracic aneurysm, mediastinal neoplasm
8. Intrinsic compression: epiglottitis, croup
9. Congestive heart failure (CHF)
10. Pulmonary embolism
11. Mechanical obstruction of the tracheal tube: kinking, secretions, over-inflation of the cuff
12. Inadequate depth of anesthesia
13. Endobronchial intubation
14. Pulmonary aspiration and edema
15. Pneumothorax

Upper Airway Causes of Wheezing

1. Allergic rhinitis/sinusitis
2. Vocal cord paralysis/dysfunction
3. Laryngeal edema
4. Laryngospasm
5. Laryngotracheomalacia
6. Infection/enlarged lymph nodes
7. Tumor
8. Polyps
9. Obstruction
10. Vascular rings or laryngeal webs
11. Kinked, misplaced, or obstructed ETT

Lower Airway Causes of Wheezing

1. Tracheal stenosis
2. Tracheal tumors
3. Bronchial stenosis
4. Foreign body/aspiration
5. Endobronchial intubation
6. Mucus plug
7. Bronchospasm
8. Bronchopulmonary dysplasia
9. Bronchiolitis: viral or obliterative

20. Wheezing

10. Asthma
11. COPD
12. Pneumonia
13. Pulmonary embolus/pneumothorax/edema
14. Cystic fibrosis

Other Causes of Wheezing

1. Anaphylaxis
2. Transfusion reaction
3. Aspiration/gastroesophageal reflux disease (GERD)
4. Foreign body aspiration into the trachea or bronchus: coins, peanuts
5. Light anesthesia
6. CHF
7. Carcinoid syndrome
8. Descending aortic aneurysm
9. Allergens
10. Chemicals
11. Exercise
12. Smoke
13. Immunologic response
14. Aspirin

Differential Diagnosis of Bronchospasm

1. Kinked ETT
2. Solidified secretion or blood
3. Pulmonary edema
4. Tension pneumothorax
5. Aspiration pneumonitis
6. Pulmonary embolism
7. Endobronchial intubation
8. Persistent cough or strain
9. Negative pressure expiration

Symptoms of Intra-operative Bronchospasm

1. Wheezing
2. Increasing peak airway pressures
3. Decreasing exhaled tidal volumes
4. Slowly rising waveform on the capnograph



KO TREATMENT PLAN

Pre-operative

First, tell the examiner that you want to talk to the surgeon to determine if this surgery is truly an emergent case. If not, you have time to optimize the patient.

1. **History: the goal in obtaining further historical information is to determine the severity of the patient's asthma.**
 - a. Onset of wheezing
 - b. Last asthma attack
 - c. Determine if the patient has ever been hospitalized for her asthma.

- d. If she was hospitalized, did she ever require endotracheal intubation?
 - e. Frequency of rescue inhaler use versus daily inhaler control
2. **Physical Exam**
 - a. Tachypnea
 - b. Tachycardia
 - c. Pulsus paradoxus: sign that is indicative of several conditions including cardiac tamponade, pericarditis, chronic sleep apnea, croup, asthma, and COPD.
 - d. Pulse oximetry
 - e. Listen for bilateral breath sounds.
 - f. Look for signs of rib fractures, flail chest, and other obvious signs of chest trauma.
 3. **A chest X-ray can be done to rule out pneumonia, congestive heart failure, pneumothorax, rib fractures, chest trauma, and aspiration of a foreign body.**
 4. **CT scan to rule out traumatic injury from the fall; pulmonary contusion, rib fractures, or hemothorax**
 5. **Arterial blood gas (ABG)**
 - a. Gives you a baseline reading.
 - b. Determine the patient's level of hypoxia and hypercarbia.
 - c. Used to guide future treatments
 6. **EKG to rule out acute right heart failure**
 7. **Pulmonary function tests (PFTs)**
 - a. Determine bronchodilator responsiveness.
 - b. Determine the severity of the asthma attack.
 - i. Mild: forced expiratory volume in the first second (FEV₁) >70% of the predicted value.
 - ii. Moderate: FEV₁ at 50–70% of the predicted value.
 - iii. Severe: FEV₁ <50% of the predicted value.
 - c. These tests are usually reserved for an elective case since they are time-consuming.
 - d. On your exam, it is good to have PFT results, but you would rarely delay any case, even an elective one, for a PFT. The delay is rarely justifiable for the answers you would get from the test.
 8. **Trial of medications**
 - a. **Rescue treatments/bronchodilator therapy**
 - i. β -adrenergic agents
 - (1) Epinephrine: intravenous (IV), intramuscular (IM), subcutaneous (SQ), or inhaled
 - (2) Albuterol: inhaled
 - (a) Also good for the acute treatment of bronchospasm
 - ii. Anticholinergics
 - (1) Ipratropium bromide: inhaled
 - (2) Produce bronchodilation by blocking the muscarinic receptors in airway smooth muscles.
 - b. **Controller treatments/anti-inflammatory therapy**
 - i. Corticosteroids
 - (1) Although not true bronchodilators, they decrease mucosal edema and may prevent the release of bronchoconstricting substances.
 - (2) Decrease airway hyperresponsiveness.

20. Wheezing

(3) Most effective for controlling chronic symptoms as the effects of the corticosteroids are not evident until hours after administration

(4) Systemic or inhaled

(5) Can still be given in the acute situation; the anti-inflammatory effects will be helpful in the recovery period.

ii. Cromolyn sodium

(1) Inhibitor of inflammation

(2) Stabilizes mast cells and inhibits degranulation and histamine release.

(3) Inhaled

(4) Not effective once bronchospasm is present.

iii. Phosphodiesterase inhibitors

(1) Block one or more of the subtypes of the enzyme phosphodiesterase.

(2) Prevent inactivation of the intracellular second messengers, cAMP and cGMP.

(a) Theophylline

(i) Rarely used secondary to the availability of aminophylline.

(ii) Relaxes bronchial smooth muscles.

(iii) Has some anti-inflammatory effects.

(iv) Central nervous system stimulatory effects on the medullary respiratory center

(b) Aminophylline

(i) Bronchodilator drug combination of theophylline and ethylenediamine in a 2:1 ratio

(ii) Less potent and shorter acting than theophylline; therefore less lasting side effects

1. Side effects range from nausea, vomiting, and insomnia to tremors, cardiac arrhythmias, and intractable seizures.

(iii) Causes bronchodilation

(c) Caffeine

iv. Antileukotrienes

v. Long-acting β -agonists

(1) Salmeterol

(2) Formoterol

c. Antibiotics as needed

Intra-operative

1. If this is considered an emergent surgery, general anesthesia is recommended in this patient. It is likely that she has sustained multiple other injuries from her three-story fall.

a. Associated head injuries could lead to agitation during the surgery.

b. The sedation required with a regional anesthetic could lead to depressed oxygenation secondary to her already compromised pulmonary status.

2. The goal of the induction of anesthesia is to depress airway reflexes to avoid bronchospasm in response to mechanical stimulation.

a. The sympathetic response to direct laryngoscopy can be blunted by IV narcotics and lidocaine 1.5 mg/kg.

3. Propofol as an induction agent has a decreased incidence of wheezing compared to thiopental. Therefore, it is the preferred induction agent in hemodynamically stable, wheezing patients.

4. Ketamine has a sympathomimetic effect that may produce smooth muscle relaxation and decreased wheezing. This is a good choice if the patient is actively wheezing or is hemodynamically unstable. Ketamine is also a good choice if the case is emergent.

5. Sevoflurane is a good choice for maintenance because it is less irritating to the airways than isoflurane and desflurane.

Intra-operative Treatment of Bronchospasm

1. 100% oxygen

2. Deepen the anesthetic: IV propofol.

3. Albuterol

4. IV or SQ epinephrine

5. Consider IV magnesium.

Post-operative

1. Determine the patient's ability to be extubated based on the extubation criteria.

2. The patient may require prolonged intubation based on her mechanism of injury.

BIBLIOGRAPHY

1. Marx JA. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. Philadelphia: Mosby, 2006, chapter 167.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 818-9.
3. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 193-204.
4. Goldman L. *Cecil Medicine*, 23rd ed. Philadelphia: Saunders, 2007, chapter 87.

21. Laryngospasm^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 4-year-old female is scheduled for a tonsillectomy. During the inhalational mask induction, your resident decides to work on the IV. You are suddenly not able to ventilate. How do you treat this? What is the cause?

CLINICAL ISSUES

Definition of Laryngospasm

1. Reflex closure of the upper airway secondary to glottic musculature spasm
2. The false cords and the epiglottic body close firmly together.
3. Hypoxia and hypercarbia result in a less vigorous glottic closure; therefore, the unsuccessful treatment of hypoxia will usually result in a spontaneous reversal of the laryngospasm.

Risk Factors for Laryngospasm

1. Stimulation of the patient while in a light plane of anesthesia: the likely cause for this sample case.
 - a. Can occur at any point during the case.
 - b. Most common during the induction of anesthesia and the wake up
2. Foreign objects: oral and nasal airway
3. Secretions, blood, and/or vomitus irritating the vocal cords.
4. Children are at an increased risk compared to adults.
5. Recent upper respiratory tract infection (URI). This is especially relevant in children.
6. Airway surgery
7. Airway pathology

Associated Complications of Laryngospasm

1. Hypoxia
2. Noncardiogenic pulmonary edema can result if there is continued spontaneous ventilation against the closed vocal cords.
3. Cardiac arrest and death



KO TREATMENT PLAN

1. Place the patient on 100% oxygen. Turn off the sevoflurane and nitrous oxide.
2. Remove the irritating factor; in this case, stop the attempt at an IV placement.
3. Apply a jaw thrust.
4. Apply continuous positive pressure ventilation via a tight mask fit.
5. Increase the depth of anesthesia with an IV agent, such as fentanyl or propofol.
6. Give intravenous or topical lidocaine.
7. Call for help if these first few techniques do not immediately break the laryngospasm.
8. Give a rapid-acting muscle relaxant such as succinylcholine. A dose of 10–50 mg IV, IM, or sublingual (SL) is usually appropriate depending on the patient's weight.
9. Attempt to intubate if the above techniques do not break the laryngospasm!!
10. As discussed above, hypoxia usually results in less vigorous glottic closure and therefore reversal of the laryngospasm.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2538–9.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 602.

22. Stridor^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

You are called to the post-anesthesia care unit (PACU) to see an 18-year-old male who has been recently extubated after a 20 hour long finger re-implantation case. He had accidentally cut off four fingers with his father's chain saw. The patient has no prior medical history. He was intubated with a #8 endotracheal tube (ETT). The anesthesia records showed that the intubation required three attempts before the patient was successfully intubated. The patient met all extubation criteria prior to extubation. He was transported to the PACU on 2 L of oxygen via nasal cannula. On arrival his oxygen saturations were hovering in the low 90s, and the nurse reported stridor. What is stridor? Are you concerned? How will you treat the patient?

CLINICAL ISSUES

Definition of Stridor

1. Noisy breathing as a result of turbulent upper airway gas flow
2. Harsh, high-pitched, vibrating sound heard with respiratory tract obstruction

Triggers

1. Obstruction
2. Airway edema

Clinical Signs

1. Noisy inspiration
2. Wheezing
3. Cyanosis
4. Chest retractions
5. Nasal flaring

Causes

1. Vocal cord paralysis
2. Laryngomalacia
3. Laryngocoele
4. Infection: tonsillitis, peritonsillar abscess, croup, and epiglottitis
5. Large tonsils and/or adenoids
6. Craniofacial abnormalities

7. Foreign body in the airway
8. Cyst or mass in the airway
9. Choanal atresia
10. Tracheomalacia
11. Vascular ring

Differential Diagnosis

1. Inspiratory stridor: upper airway obstruction
2. Expiratory stridor: lower airway obstruction
3. Biphasic stridor: mid-tracheal lesions



KO TREATMENT PLAN

1. Go to evaluate the patient immediately; stridor can be a sign of a serious problem.
2. Rule out the need for an emergent intubation!
3. Obtain a thorough history, including the time of onset and positions that make the stridor better or worse.
4. Perform a physical exam to evaluate the general condition of the patient and the degree of airway compromise. Pay special attention to the respiratory rate, heart rate, and the patient's level of consciousness.
5. Chest X-ray
6. Arterial blood gas
7. Flexible bronchoscopy can be done to directly visualize the airway.
8. Consider an ear, nose, and throat (ENT) consult.

If the intubation can be delayed, treatment includes

1. Oxygen by facemask
2. Head-up positioning: 45 to 90 degrees
3. Nebulized racemic epinephrine
4. Intravenous dexamethasone 4–8 mg given every 8–12 hours.
5. Heliox (70% helium, 30% oxygen)
 - a. One-third as dense as air or oxygen
 - b. It can lead to a dramatic decrease in airway resistance and therefore improved ventilation.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, p 2539.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 1003.

23. Obstructive Sleep Apnea (OSA)¹⁻⁹

Norman Bolden, MD

SAMPLE CASE

A 54-year-old obese male whose surgical history includes gastric bypass is scheduled for a left knee arthroscopy for ACL reconstruction at the ambulatory center. The patient has lost 65 lbs since his gastric bypass surgery two years ago and his current BMI is 45. The patient reports that a tooth was dislodged during the previous gastric bypass procedure. The patient has a past medical history significant for severe OSA (apnea-hypopnea index [AHI] of 65 three years ago) and was previously on a continuous positive airway pressure (CPAP) of 12. The patient states that his sleep and breathing is much improved following his weight loss, and that currently he does not use his CPAP as he believes his OSA is no longer a problem. The patient also has diabetes and was previously on oral agents, but his condition is currently diet controlled. He has a blood pressure of 155/84 mm Hg, heart rate of 105, respiratory rate of 20, and a temperature of 35.6°C. His hematocrit is 49%. Is this patient a candidate for an outpatient surgery? How would you induce anesthesia? Would you prefer to do a regional or general anesthetic? Are you concerned that he is a difficult intubation? How would you manage his post-operative pain? Will he need special post-operative monitoring?

CLINICAL ISSUES

OSA Clinical Definitions

1. OSA: medical condition characterized by complete airway obstruction (apnea) or partial airway obstruction (hypopnea) despite respiratory effort during periods of sleep
 - a. Symptoms
 - i. Heavy snoring
 - ii. Sudden awakenings with a choking sensation
 - iii. Witnessed apneas by bed partner
 - iv. Excessive daytime sleepiness
2. Polysomnogram (PSG): a formal sleep study. This is the standard for the definitive diagnosis of OSA.
3. Apnea-Hypopnea Index (AHI): average number of apneas and hypopneas per hour during the formal sleep study. This is used to determine the severity of OSA.
 - a. Mild OSA = AHI 5-15
 - b. Moderate OSA = AHI 16-30
 - c. Severe OSA = AHI > 30

Medical Management of OSA

1. CPAP or bi-level positive airway pressure (BiPAP)
 - a. Most common form of treatment
 - b. Improves oxygenation and limits apneic episodes by stenting the airway open.
 - c. Patient compliance with CPAP and BiPAP is often problematic.
2. Oral airway devices

Surgical Management of OSA

1. Tonsillectomy is often performed for the treatment of children with OSA.
2. Uvulopalatopharyngoplasty (UP3) is often performed in adults.
 - a. "Success" occurs in only about 50% of surgical procedures performed.
 - b. "Surgical success" is defined as a 50% improvement in symptoms.
 - c. The patient can have a "successful" UP3 procedure, *yet still have severe OSA!*
3. Maxillomandibular advancement
4. Tracheostomy

Peri-operative Concerns

1. Increased risk for difficult intubation
2. Post-operative hypoxemia
3. Post-operative airway obstruction requiring the need for reintubation
4. Myocardial ischemia
5. Arrhythmia
6. Death

Pathophysiology of OSA during the Peri-operative Period

1. All central nervous system (CNS) depressant drugs (inhaled agents, hypnotics, narcotics) and neuromuscular blocking agents cause (to varying degrees) relaxation of pharyngeal muscles.
2. Relaxation of pharyngeal muscles predisposes the OSA patient to airway collapse and obstruction.
3. CNS depressants also impair the arousal state, which terminates apneic episodes, which predisposes the patient to hypoxemia and hypercarbia.



KO TREATMENT PLAN

Pre-operative

1. One must not assume that because this patient has lost weight, severe OSA no longer exists. A formal PSG must be obtained to ensure that the patient no longer has OSA. Therefore, this patient must be treated as if severe OSA remains. Consequently, one must pre-operatively make the determination of whether it is reasonable to perform this procedure on an outpatient basis. Patients with OSA are known to be at risk for difficult intubation, and the fact that a tooth was dislodged during the previous surgical procedure suggests that this patient may have been a difficult intubation. One cannot guarantee that regional anesthesia can be successfully performed, nor can it be assumed that a peripheral nerve block will be successful; therefore one must have contingency plans for a general anesthetic. Given this patient's BMI, history of severe OSA, and probable history of difficult intubation, one can make a strong argument that it would be most prudent to perform this procedure on an inpatient basis.
2. One must do a thorough cardiac and pulmonary exam. The elevated hematocrit may be a sign of chronic hypoxemia or may be due to dehydration. Electrolytes (specifically BUN/creatinine may indicate whether the patient is prerenal) and glucose levels should be assessed pre-operatively.
3. Have the patient bring his CPAP machine to hospital even though he hasn't been using it.
4. Arrange to have a monitored bed available for the patient post-operatively.

Intra-operative

1. Utilize regional anesthesia whenever possible for OSA patients. Use neuraxial anesthesia (spinal or epidural) as the primary anesthetic plan. Patients with OSA are still at risk for airway obstruction and apnea following neuraxial opioids. Consider a femoral block following the procedure rather than neuraxial opioids.
2. Your contingency plan should be a general anesthetic (GA) if the regional anesthetic is unsuccessful or a high spinal develops. You should always be prepared for a difficult intubation by having a fiber optic bronchoscope (FOB), Glidescope, Bougie, LMA, and/or intubating LMA all available. If GA is required, propofol, dexmedetomidine, remifentanyl, sevoflurane/desflurane are all good choices, since they promote a rapid recovery.
3. Monitor glucose levels intra-operatively and treat accordingly.

4. Ensure that the patient's muscle relaxation is fully reversed and the patient is awake at the time of extubation.
5. Discuss the possible use of nonsteroidal anti-inflammatory drugs (NSAIDs) with the surgeon to avoid narcotics.

Post-operative

1. Discuss a possible femoral block with the patient. If successful, this may eliminate the need for intravenous narcotics.
2. If the nerve block is unsuccessful or the patient refuses, treat his pain with narcotics.
3. Monitor the patient closely in the post-anesthesia care unit (PACU). If the patient is stable during narcotic administration in the PACU, without evidence of apneas/hypoxemia, he can be admitted to a monitored bed with continuous pulse-oximetry monitoring following the procedure. If the patient has profound episodes of apnea and associated desaturation, he likely needs intensive care unit (ICU) observation. OSA patients should not have pain medications withheld just because they have OSA. Rather, these patients require monitoring when receiving narcotics to address their pain.
4. Patients should be encouraged to wear their CPAP during all periods of sleep. If they are sleeping during the day, they should be wearing their CPAP.

BIBLIOGRAPHY

1. Gupta R, Parvizi J, Hanssen A, Gay P. Postoperative complications in patients with obstructive sleep apnea syndrome undergoing hip or knee replacement: A case-control study. *Mayo Clin Proc.* 2001;76:897-905.
2. Auckley D, Bolden N, Smith CE. Perioperative considerations in patients with obstructive sleep apnea. *Current Respiratory Medicine Reviews* 2007;3:245-53.
3. McMains KC, Terris DJ. Evidence-based medicine in sleep apnea surgery. *Otolaryngologic Clinics of North America.* 2003;36(3):539-61.
4. Partinen M, Jamieson A, Guilleminault C. Long-term outcome for obstructive sleep apnea syndrome patients. Mortality. *Chest.* 1988;94 (6) 1200-1204.
5. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med* 2005;353(19):2034-41.
6. Benumof J. Obstructive sleep apnea in the adult obese patient: implications for airway management. *Journal of Clinical Anesthesia.* 2001;13(2):144-56.
7. Bell RL, Rosenbaum SH. Postoperative considerations for patients with obesity and sleep apnea. *Anesthesiology Clinics of North America.* 2005;23(3):493-500.
8. Couch ME, Senior B. Nonsurgical and surgical treatments for sleep apnea. *Anesthesiology Clinics of North America.* 2005;23(3):525-34.
9. Harbison J, O'Reilly P, McNicholas WT. Cardiac rhythm disturbances in the obstructive sleep apnea syndrome. Effects of nasal continuous positive airway pressure therapy. *Chest* 2000;118(3):591-5.

24. Post-Operative Mechanical Ventilation^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

CLINICAL ISSUES

Controlled Mechanical Ventilation (CMV)

1. The ventilator cycles from expiration to inspiration after a fixed time interval.
2. The interval determines the respiratory rate.
3. Fixed tidal volume and fixed rate; therefore, fixed minute ventilation
4. Works independently of the patient's effort.
 - a. The patient cannot breathe spontaneously.
 - b. Best used for patients with little or no respiratory effort.
 - i. Sedated
 - ii. Paralyzed

Assist-Control Ventilation (AC)

1. Positive-pressure mechanical ventilation
2. Volume-cycled lung inflation
 - a. Each machine breath delivered has a pre-selected inflation volume.
 - b. This pre-selected volume is delivered if the breath is initiated by the patient or the machine.
3. The patient can initiate each mechanical breath: assisted ventilation.
4. The ventilator provides machine breaths at a pre-selected rate: controlled ventilation.
 - a. If no spontaneous effort is detected, the machine functions as if it is in the control mode.
5. Adverse effects
 - a. Occur primarily in patients who are spontaneously breathing rapidly.
 - i. Increased frequency of machine breaths can lead to overventilation, hyperventilation, and respiratory alkalosis.
 - (1) Auto-PEEP

Intermittent Mandatory Ventilation (IMV)

1. Delivers periodic volume-cycled breaths at a pre-selected rate.
2. Allows spontaneous ventilation between machine breaths.
 - a. Each spontaneous breath does not trigger a machine breath.

3. If the spontaneous breath triggers the scheduled machine breath, it is called synchronized IMV.
 - a. The machine breaths are synchronized to coincide with spontaneous lung inflations.
4. Disadvantages
 - a. Increases the work of breathing.
 - i. Spontaneous ventilation occurs against a high resistance circuit.
 - ii. Could lead to respiratory muscle fatigue.
 - b. Reduced cardiac output in patients with left-ventricular dysfunction

Pressure-Controlled Ventilation (PCV)

1. Pressure-cycled ventilation
 - a. Peak airway pressure is set.
 - b. Mandatory rate is set.
2. Lower peak airway pressure compared to volume-cycled ventilation
3. The ventilation is completely controlled by the machine and not the patient.
4. The inspiratory flow rate decreases exponentially during lung inflation.
 - a. Reduces peak airway pressures.
 - b. May improve gas exchange and oxygenation.
5. Advantage
 - a. Decreased risk of barotraumas and volutrauma when compared with volume set breaths
6. Disadvantages
 - a. Inflation volumes vary.
 - b. The patient must be heavily sedated or paralyzed to tolerate this mode of ventilation.

Pressure-Support Ventilation (PSV)

1. Pressure-augmented breathing
 - a. Peak airway pressure is set.
2. Allows the patient to determine the inflation volume and respiratory cycle duration.
3. Used to augment the tidal volume of spontaneously breathing patients and overcome any increased inspiratory resistance from the endotracheal tube, breathing circuit, and ventilator.
4. Should not be used to provide full ventilatory support.

24. Post-Operative Mechanical Ventilation

5. At the onset of each spontaneous breath, the negative pressure generated by the patient opens a valve that delivers the inspired gas at a pre-selected pressure (usually 5–10 cm H₂O).
6. When the patient's inspiratory flow rate falls below 25% of the peak inspiratory flow, the augmented breath is terminated.
 - a. This allows the patient to dictate the duration of lung inflation and the inflation volume.
7. Indications
 - a. Augment inflation volumes during spontaneous respiration.
 - b. Used to overcome the resistance of breathing through the ventilator
 - c. Used for weaning patient from mechanical ventilation
 - d. Increase patient comfort
 - e. Can be used in combination with IMV to decrease the work of breathing imposed by the breathing circuit and machine.
8. Can be used non-invasively through a specialized face or nasal mask.
 - a. Use pressures of 20 cm H₂O.

Positive End-Expiratory Pressure (PEEP)

1. The alveolar pressure at end-expiration is above the atmospheric pressure.
2. A pressure-limiting valve is placed in the expiratory limb of the ventilator circuit.
 - a. Positive pressure is applied during expiration.
3. Without PEEP, the distal airspaces would tend to collapse at the end of expiration.
 - a. This alveolar collapse impairs gas exchange.
 - b. PEEP prevents the alveoli from collapsing at end-expiration and can open alveoli that have already collapsed.
4. Improves gas exchange (decreases intrapulmonary shunt) and makes the lungs less stiff (increases lung compliance).
5. Physiologic effect
 - a. Cardiac filling and cardiac output are hampered due to pressure in the thoracic cavity having a negative effect on venous return.
 - b. Increased alveolar pressure causes higher partial pressure of oxygen at the alveoli.
6. Indications
 - a. Toxic levels of inhaled oxygen are required.
 - i. The addition of PEEP can increase arterial and systemic oxygenation and allow the reduction of the inhaled oxygen to be reduced to less toxic levels.

- b. Low volume ventilation
 - c. Obstructive lung disease
7. Benefits
 - a. Increase in functional residual capacity (FRC)
 - b. Improved lung compliance
 - c. Correction of ventilation-perfusion abnormalities
 - d. Improved oxygenation
8. Adverse effects
 - a. Barotrauma
 - i. Pneumothorax
 - ii. Pneumomediastinum
 - iii. Pneumopericardium
 - iv. Spontaneous emphysema
 - b. Decreased venous return
 - c. Decreased cardiac output

Continuous Positive Airway Pressure (CPAP)

1. Spontaneous breathing in which positive pressure is maintained throughout the respiratory cycle
 - a. Positive pressure is applied during inspiration and expiration during spontaneous breathing.
2. The patient does not have to generate a negative airway pressure to receive the inhaled gas.
 - a. This eliminates the extra work involved in generating a negative airway pressure to inhale.
3. Indications
 - a. Non-intubated patients
 - i. Nasal and face masks
 - ii. The patient should be awake and alert.
 - iii. Pressures should not exceed 15 cm H₂O.
 - b. Obstructive sleep apnea
 - c. Acute respiratory failure (to postpone intubation)
 - d. Acute exacerbation of chronic obstructive pulmonary disease

BIBLIOGRAPHY

1. Marino PL. *The ICU Book*, 2nd ed. Baltimore: Williams & Wilkins, 1998, pp 434–46.
2. Duke J. *Anesthesia Secrets*, 2nd ed. Philadelphia: Hanley & Belfus, 2000, p 106.
3. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 3rd ed. New York: McGraw-Hill, 2002, pp 959–65.
4. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2821–8.

25. High-Frequency Jet Ventilation^{1,2,3,4}

Ryan J. Gunselman, MD and Donald M. Voltz, MD

SAMPLE CASE

A famous vocalist presents to the operating room for removal of a vocal cord lesion. The surgeon is concerned about further damage to this patient's vocal cords and requests that the patient not be intubated. Given the shared nature of the airway and assuming the patient is otherwise healthy, how would you manage the anesthesia and the airway for this case? One suggestion by the surgeon is to use high-frequency jet ventilation. How is this managed and what are your concerns with this form of ventilation? Are there any patients for whom this type of ventilation is not appropriate? What additional monitoring is required to perform this type of ventilation?

CLINICAL ISSUES

Definition of High-Frequency Jet Ventilation (HFJV)

1. HFJV is a method of ventilation in patients requiring surgery of the larynx when an endotracheal intubation may obscure or interfere with the intended procedure.
2. HFJV has also been used as
 - a. Mode of ventilation in patients when a conventional endotracheal tube could not be placed
 - b. In neonatal patients with significant pulmonary disease when conventional ventilation is not successful
3. This technique is usually performed using some type of high-flow oxygen source, tubing, and a narrow conduit (e.g., needle, bronchoscope) to deliver oxygen flows into the patient's trachea.
4. HFJV is most commonly used in procedures in which access to the larynx or trachea is required by the surgeon (i.e., excision/laser of tumors, polyps).
5. This technique stems from the clinical application of *Bernoulli's principle*, which is elaborated on below.
6. In addition to patients undergoing laryngeal surgery, HFJV has been utilized as a bridge to definite, surgical airway control in patients who cannot be ventilated or intubated due to an anatomic or pathologic airway condition.
 - a. Although principles are the same in these two situations, the specialized ventilators typically used in airway surgery are often not present for emergency airway control necessitating higher vigilance to avoid complications or injury to the patient.

Principles behind the HFJV Technique

1. *Bernoulli's principle*: as the velocity of a fluid (liquid or gas) increases, the pressure decreases.
2. *Venturi effect*: as a fluid flows through a narrowed orifice, the velocity of the fluid increases with a concomitant decrease in the pressure.
 - a. This change in fluid velocity and pressure stems from *Bernoulli's principle*.

TKO: The clinical application of these two mechanisms using specialized ventilators and endotracheal delivery devices allows for HFJV.

Mechanism of HFJV

1. With the jet delivery of oxygen through specialized cannulae placed in the patient's airway (i.e., larynx, trachea, distal airways) at a very high rate, one is able to deliver oxygen into the trachea.
 - a. HFJV provides a tidal volume less than the anatomic dead space at rates significantly more rapid than normal.
 - i. Delivery of a pulse of gas from a high-pressure source (50 psi)
 - b. Frequencies are stated as cycles per minute or second (Hz).
 - i. Goal rate is 100–400 breaths per minute.
 - ii. Inspiration accounts for 20–30% of the cycle.
2. Since, unlike conventional ventilation, the delivery of oxygen is at a lower pressure and therefore does not allow for traditional tidal volume excursion of the lungs and thoracic cage, oxygen is distributed throughout the alveolar space by diffusion.
 - a. HFJV delivers a small tidal volume with high gas flow rates and therefore lower maximum, mean, and transpulmonary airway pressures than with conventional positive pressure ventilation.
3. Despite the name "jet ventilation," removal of carbon dioxide is not as efficient as with conventional ventilation and occurs by the passive recoil of the chest and lungs with some assistance from intra-abdominal pressure.
 - a. When the jet is active, a volume of oxygen is entrained allowing for the chest wall expansion, followed by elastic recoil of the chest wall when the jet flow is terminated.

25. High-Frequency Jet Ventilation

b. Invasive catheters

i. Most commonly placed through the cricothyroid membrane

ii. Often placed in emergent situations (cannot ventilate, cannot intubate patient); can be set up using basic supplies on hand in most operating rooms (see Figure 25.1).

iii. More complications with this technique

iv. Displacement of the catheter can result in significant subcutaneous emphysema making it more difficult to replace the cannula or perform a definitive surgical airway.

v. Delivery of high pressure for extended periods of time can result in tension pneumothorax and/or cardiovascular collapse.

vi. If the patient does not have a patent glottic or supraglottic area (i.e., patients with obstructing airway tumors or redundant pharyngeal tissue), this technique is of limited utility and results in air trapping and hyper-expansion of the lungs.

vii. Insertion technique

(1) Identify the cricothyroid membrane by palpation.

(2) Attach a 10 mL or 20 mL syringe partially filled with saline to a large-bore angiocatheter.

(3) Insert through the cricothyroid membrane at a 30 degree angle, pointing the needle caudally (down the trachea).

(4) Aspirate while inserting; air will bubble up through the syringe.

(5) Thread the angiocatheter off the needle into the trachea; once the angiocatheter is inserted completely, the position should be reconfirmed by withdrawing air.

(6) Attach the jet ventilation source to the angiocatheter.

viii. Complications (in order of incidence)

(1) Subcutaneous emphysema (most common)

(2) Bleeding

a. Often this is minor.

b. If an anterior jugular vein, thyroid isthmus, or innominate artery is encountered, the bleeding can be significant.

(3) Barotrauma

(4) Pneumothorax

(5) Esophageal injury

(6) Arterial laceration

(7) Cardiovascular compromise from air trapping

a. Driving pressure is the residual oxygen pressure in the ventilator that is available to deliver a breath to the patient.

b. Maximum recommended driving pressure is 50 psi.

5. Peak inspiratory pressure (PIP)

a. Measured from the pressure line

b. Detects high pressures in the system.

c. The alarm will disable breath delivery until elevated PIP is corrected.

d. Recommended initial pressure settings for the PIP is 28 cm H₂O in adults and 12 cm H₂O in children.

6. Pause pressure (PP)

a. Measured in the jet line 10 milliseconds prior to the delivery of each breath

b. The purpose of the PP is to prevent breath stacking and barotraumas.

c. Recommended pressure limit is 24 cm H₂O.

7. Humidification level

Ventilation Technique, General Recommendations

1. Adults will require a pressure of at least 30 psi for ventilation to be effective (flow through common gas outlet is approx. 50–55 psi).

2. For children, the recommendation is 0.4 psi/kg.

3. Inspiration should be less than one second followed by 2–3 seconds of expiration time.

4. Keep in mind that in cases of severe airway obstruction the only outlet for expiration may be the angiocatheter, and this is not a large enough orifice to allow for exhalation of the delivered volume.

a. Establishing a patent proximal airway or placement if a larger diameter catheter may be required to allow for adequate exhalation.



Figure 25.1. Transtracheal jet ventilator setup to the common gas outlet: Remember that this set-up does not allow for a way to monitor airway pressures or sample exhaled gas. Photo credit: R. Günselman.

Jet Ventilator Parameters

1. Frequency

2. Percent inspiratory time

a. Recommended initial setting is 40%.

3. Inspiratory oxygen content

4. Driving pressure



Figure 25.2. Monsoon High Frequency Jet Ventilator. Photo Credit: D. Voltz.

- b. Monitoring airway pressures is important to prevent complications; however, in an emergency situation this is not always possible or available.
- c. Intermittent disconnection may be needed to allow exhalation and to avoid barotrauma.
- 5. CO₂ monitoring should be performed.
 - a. Side channel sampling of CO₂ is often unreliable.
 - b. Arterial blood gas sampling may be needed.



KO TREATMENT PLAN

Pre-operative

1. Complete history and physical examination
2. If HFJV is the anesthetic plan, discuss the goals for the patient's ventilation, and your plan if these goals are not met.
 - a. Intubation
 - b. Aborting the procedure

Intra-operative

1. Apply standard ASA monitors.
2. Consider the placement of an arterial line for frequent CO₂ sampling.
3. Intravenous induction using lidocaine, fentanyl, propofol, and rocuronium
3. Maintenance anesthesia: propofol and remifentanyl infusion
 - a. Good for rapid onset and termination
 - b. Volatile anesthetics cannot be used with the HFJV technique.
4. The HFJV catheter should be placed beyond the vocal cords using direct laryngoscopy.
5. Use the HFJV technique that you are the most comfortable with.
 - a. Microcomputer controlled jet ventilator
 - b. Subglottic catheter
6. Insist on CO₂ monitoring with side channel sampling.

BIBLIOGRAPHY

1. Miller RD, Cucchiara RF, Miller ED, et al. *Anesthesia*, 5th ed. New York: Churchill Livingstone, 2000, pp 2418-9.
2. Yao FSF, Fontes ML, Malhotra V. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 77-80.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 843.
4. Morgan GE, Mikhail MS, Murray M. *Clinical Anesthesiology*, 4th ed. New York: Lange, 2006, pp 985, 1034-5.

26. One Lung Ventilation^{1,2}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 60-year-old, 120 kg male is scheduled for a right middle lobectomy for carcinoma of the lung. He has a 50 pack-year smoking history. He suffers from chronic obstructive pulmonary disease (COPD) and asthma. His pulse is 70 and blood pressure (BP) is 145/90 mm Hg. What are your concerns? How will you secure this patient's airway? What are your intra-operative concerns? If the intubation was difficult, how would you change the double-lumen tube (DLT) to a single-lumen tube?

CLINICAL ISSUES

Indications for Separation of the Two Lungs/ One Lung Ventilation¹

1. Absolute
 - a. Isolation to prevent spillage or contamination
 - i. Infection
 - ii. Massive hemorrhage

26. One Lung Ventilation

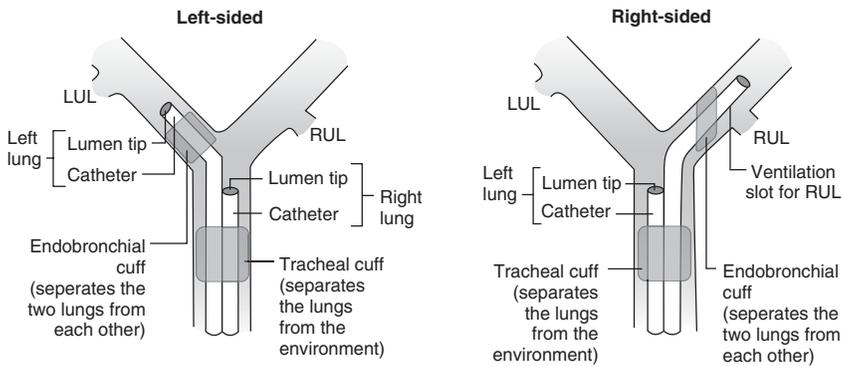


Figure 26.1. Placement of a DLT.

Source: This figure was published in *Anesthesia, 6th Edition*, RD Miller, LA Fleisher, RA Johns, et al., Page 1874, Copyright Elsevier (2005). Reprinted with permission.

- b. Control of ventilation**
 - i.** Bronchopleural fistula
 - ii.** Bronchopleural cutaneous fistula
 - iii.** Surgical opening of a major conducting airway
 - iv.** Giant unilateral lung cyst or bulla
 - v.** Tracheobronchial tree disruption
 - vi.** Life-threatening hypoxemia from unilateral lung disease
- c. Unilateral bronchopulmonary lavage**
 - i.** Pulmonary alveolar proteinosis
- 2. Relative**
 - a. Surgical exposure**
 - i.** Thoracic aortic aneurysm
 - ii.** Pneumonectomy
 - iii.** Upper lobectomy
 - iv.** Mediastinal exposure
 - v.** Thoracoscopy
 - vi.** Middle and lower lobectomies
 - vii.** Esophageal resection
 - viii.** Thoracic spine surgery
 - b. Severe hypoxemia from unilateral lung disease**

Techniques for Lung Separation

- 1. Bronchial blocker**
 - a.** Physiologically the same effect as that produced by clamping one lumen of a DLT
 - b. Clinical indications**
 - i.** Critically ill patients in whom it may not be feasible to place a DLT
 - ii.** Intubated patients
 - iii.** Patients with a known difficult airway
 - iv.** Need for post-operative ventilation
 - (1)** May avoid a risky post-operative change from a DLT to single-lumen tube.
 - c. Limitations**
 - i.** Slow lung deflation time
 - ii.** Slow lung reinflation time
 - iii.** Difficult suctioning of the operative lung
- 2. Endobronchial placement of a regular endotracheal tube (ETT)**
 - a. Clinical indications**
 - i.** Rapid, easy way of effectively separating two lungs, especially in cases of massive hemoptysis

- ii.** In most situations, the tube will guide to the right mainstem.

(1) If the left mainstem must be intubated, the patient's head is turned to the right, and the tube is rotated 180 degrees.

(2) If there is time, and good visualization, a fiber optic bronchoscope may be used to guide the tube into place.

- b. Limitations**
 - i.** If entering the right main stem bronchus, there is a high probability that the right upper lobe bronchus may be blocked off, leading to serious hypoxemia.
 - ii.** Unable to suction the operative lung

- 3. DLT**
 - a.** Placement of a DLT (see [Figure 26.1](#))
 - b.** Verification of DLT placement by auscultation (see [Figure 26.2](#))
 - c.** Verification of DLT placement by fiber optic bronchoscopy
 - i.** In a left-sided DLT, placing the fiber optic scope through the tracheal lumen will reveal a view of the tracheal carina, with the upper surface of the blue left endobronchial balloon just visible below the tracheal carina, off to the left.
 - ii.** The cuff should not be herniated over the carina.

TKO: Major malpositions are frequently tested!! Know the signs of malposition both by auscultation and fiber optic bronchoscopy! Know that fiber optic bronchoscopy will reveal a malpositioning incidence as high as 78%. Therefore, even after appropriate auscultation, verification of precise placement with a fiber optic scope is critical.



KO TREATMENT PLAN

Pre-operative

- 1. Patients presenting for thoracic surgeries often have an associated history of chronic obstructive pulmonary disease and a history of cigarette smoking.**
 - a.** Evaluate the respiratory and cardiovascular functions of these patients appropriately.

26. One Lung Ventilation

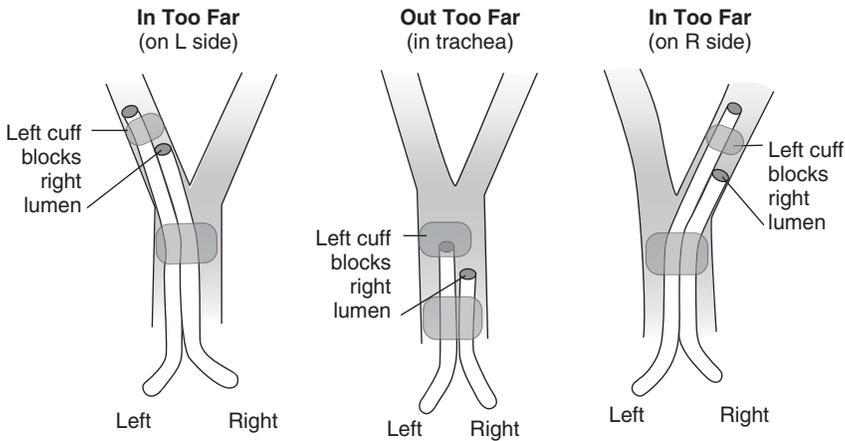


Figure 26.2. Verification of DLT placement by auscultation.

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Table 26.1. Procedure and Breath Sounds Heard

Procedure	Breath Sounds Heard		
	In Too Far Left	Out Too Far	In Too Far Right
Clamp tracheal lumen, both cuffs inflated	Left	Left and right	Right
Clamp bronchial lumen, both cuffs inflated	None or very decreased	None or very decreased	None or very decreased
Clamp tracheal lumen, deflate bronchial cuff	Left	Left and right	Right

Adapted from Miller RD, ed. *Miller's Anesthesia, 6th ed.* Philadelphia: Churchill Livingstone, 2005, p. 1879.

i. Pre-operative pulmonary function tests may be indicated to establish a baseline and identify patients unable to tolerate the planned procedure.

(1) Tests also indicate if improvement in respiratory function occurs following bronchodilator therapy.

ii. EKG and other studies may be indicated from the patient's history and physical examination.

(1) This is considered an intermediate risk surgery per the ACC/AHA guidelines.

iii. Evaluate for hematologic abnormalities and follow baseline blood counts with hematocrit and coagulation studies.

(1) Coagulation studies are especially important if thoracic epidural anesthesia is planned for post-operative pain relief.

2. Evaluate for appropriate post-operative pain control.

a. Ensures patient comfort.

b. Minimizes pulmonary complications.

c. Thoracic epidural analgesia is the gold standard for post-thoracotomy analgesia with a combination of low-dose narcotic and dilute local anesthetic solution.

Intra-operative

1. Assuming the patient has a normal airway, place a DLT after the induction of general anesthesia.

a. Verify the correct placement using auscultation and direct visualization with the fiber optic scope.

2. Verify tube placement again once the patient is placed in the lateral position.

3. Maintain two-lung ventilation as long as possible.

4. Use 100% FiO₂.

5. Begin one-lung ventilation without changing tidal volumes, unless peak pressures are prohibitive of this maneuver.

a. Keep the plateau airway pressures <25 cm H₂O.

6. Keep the PaCO₂ at approximately 40 mm Hg by adjusting the respiratory rate.

7. Treatment of hypoxemia

a. If severe, switch to two-lung ventilation immediately!

b. Check the position of the DLT with fiber optic bronchoscopy.

c. Apply continuous positive airway pressure (CPAP), 5–10 cm H₂O to the nondependent lung.

i. Overcomes the atelectasis in the nonventilated lung, decreasing the shunt fraction.

d. Apply positive end-expiratory pressure (PEEP), 5–10 cm H₂O to the dependent lung.

e. Adjust the CPAP and PEEP in an attempt to find the optimal end-expiratory pressure for each lung.

f. Intermittently ventilate both lungs.

g. In an emergency, have the surgeon clamp the pulmonary artery.

Post-operative

1. The post-operative course should be guided by the patient's pre-operative cardiopulmonary condition and intra-operative course.
2. If the trachea is to be extubated, the patient must meet extubation criteria as defined in this review book.
3. If the patient is to remain on mechanical ventilation, the DLT must be changed to a single-lumen tube and the position of the tube should be verified with end-tidal CO₂ monitoring and auscultation.
 - a. In the setting of a difficult initial intubation, the problem of maintaining adequate post-operative ventilation exists. There are a number of possible solutions, all of which may be appropriate in a given set of circumstances.
 - i. Leaving the double-lumen tube *in situ* and withdrawing the bronchial lumen into the trachea
 - (1) This avoids any further manipulation of the airway but is bulky and should not be used if there is a suspicion that post-operative ventilation will last for an extended period of time.
 - (2) In addition, few intensive care units are comfortable with caring for a DLT, and as a member of the patient care team, it is important to maintain such consideration.
 - ii. Exchange of DLT for single-lumen tube via an extended tube changer

- (1) This would be the author's method of choice.
- (2) It requires an assistant to remove the DLT over a tube changer while direct laryngoscopy is maintained by the anesthesiologist. The single-lumen tube is then threaded over the tube changer by the assistant.

iii. In the setting of a known pre-operative difficult airway, the DLT may be avoided, and a bronchial blocker placed instead.

- (1) This avoids the post-operative problems of changing the tube, and allows for maintenance of mechanical ventilation.
- (2) This is also a successful option in patients who are intubated pre-operatively, as the change from a single-lumen to a DLT may lead to loss of airway control.

4. If the patient remains mechanically ventilated, an aggressive respiratory care regimen is necessary to remove secretions, diagnose/prevent/treat infections, and dilate the airways.

BIBLIOGRAPHY

1. Miller RD, ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Churchill Livingstone, 2005, pp 1873-1906.
2. Jaffe RA, Samuels SI, eds. *Anesthesiologist's Manual of Surgical Procedures*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2004, pp 206-17.

27. Hypotension^{1,2}

Robert C. Lee, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 58-year-old female sustained a right femur fracture. She had a prior right total hip arthroplasty and will need a revision. Her past medical history includes hypertension, mild obstructive sleep apnea, hiatal hernia, and obesity. The patient is terrified of needles and states she had a bad experience with a prior cesarean section after multiple attempts at a spinal anesthetic. She is taken to the operating room and general anesthesia is induced with IV esmolol, lidocaine, fentanyl, and propofol. The tracheal intubation is uneventful. The femoral prosthesis with bone cement is placed. Two minutes later, you notice the end-tidal CO₂ decrease to 25 mm Hg when it had been between 35–40 mm Hg during the entire case. The minute ventilation has remained the same and you cycle the blood pressure (BP) cuff. The BP is 78/45 mm Hg. A repeat BP is 67/38 mm Hg and the end-tidal CO₂ is now 12 mm Hg. What would you do next? What is your differential diagnosis?

CLINICAL ISSUES

Definition of Hypotension

1. The blood pressure is low enough to cause signs and symptoms of inadequate blood flow to the vital organs.
2. A blood pressure less than 90/60 mm Hg is sometimes considered the cutoff for hypotension, but this may be a perfectly acceptable value in some people.

Signs and Symptoms of Hypotension

1. An alert and awake patient may start complaining of light-headedness, dizziness, nausea, fatigue, shortness of breath, chest pain, lack of concentration, and blurred vision.
2. The patient may have a syncopal episode and/or have cold, clammy, and pale skin.
3. A patient under general anesthesia will have a drop in blood pressure greater than 20% of his or her normal baseline.
4. You may see changes in the EKG (ischemic; such as ST segment depressions or elevations), pulse oximeter (hypoxia), heart rate (reflex tachycardia), and end-tidal CO₂ (decreased secondary to a decreased cardiac output state).

Differential Diagnosis

TKO: This list is extremely important for the oral board exam. In almost every exam, at some point, the patient will become hypotensive. You should have this list memorized, so that you can easily determine the possible causes of the patient's hypotension and be able to quickly treat the problem.

1. Pulmonary: hypoxia, hypercarbia, tension pneumothorax
2. Hypovolemia: fluid deficit, acute blood loss
3. Cardiac: rate/rhythm abnormality, inotropic failure, myocardial ischemia, contusion, tamponade, rupture, congestive heart failure (CHF), cardiomyopathy, valvular injury, or lesion
4. Shock: hypovolemia, cardiogenic, septic
5. Surgical compression of the heart, aorta, inferior vena cava (IVC), or abdominal contents
6. Embolus: pulmonary, air, fat, amniotic
7. Electrolyte and hormonal abnormalities: hypoglycemia, hypocalcemia, adrenal insufficiency, anti-diuretic hormone (ADH) suppression, and hypermagnesemia
8. Anaphylaxis: latex, transfusion, drugs such as antibiotics (penicillin, cephalosporin, sulfa, vancomycin), local anesthetic (usually an ester), muscle relaxants (atracurium, mivacurium, d-tubocurarine, succinylcholine), opioids (morphine, meperidine), protamine, colloids, iodine, and IV contrast dye
9. Deep anesthesia, drug overdose, medications such as angiotensin converting enzyme inhibitors (ACEIs) and angiotension receptor blockers (ARBs)
10. Hypothermia
11. Sympathetic blockade, neuraxial block
12. Venodilation
13. Laparoscopy
 - a. Hypercarbia
 - b. Dysrhythmia
 - c. Increased vagal tone from excessive stretching of the peritoneum
 - d. Compression of the inferior vena cava causing a decrease in cardiac output
 - e. Venous gas embolism



KO TREATMENT PLAN

Pre-operative

1. Validate the blood pressure by re-checking it. Make sure the cuff is the appropriate size and/or that the arterial line transducer is at the correct level.
2. Evaluate the patient and inquire about specific symptoms such as lightheadedness, dizziness, nausea, blurred vision, and chest pain.
3. Perform a physical exam.
 - a. Auscultate the heart and lungs.
 - b. Palpate for a carotid or femoral pulse.
 - c. Check the mucous membranes and extremities to help assist in a rough estimate of the patient's volume status.
 - d. Assess the ABCs (airway, breathing, and circulation).
 - e. Check the patient's temperature.
 - f. Look for signs of bleeding.
4. Provide supplemental oxygen.
5. Increase IV fluid administration until the cause of the hypotension is determined.
6. Review the patient's medical history.
7. Review the patient's medication list. Determine when her medications were last given.
8. Attempt to determine the patient's baseline blood pressure.
9. Consider getting a 12-lead EKG to evaluate the patient's cardiac rate and rhythm.
10. Order an arterial blood gas, check electrolytes, and get a chest radiograph.
11. Initiate therapy directed at the etiology of the hypotension, if indicated, and re-confirm the diagnosis.
12. Consider postponement of an elective procedure, especially if the patient is experiencing symptoms from the hypotension, unless the cause of the hypotension can be treated surgically.

Intra-operative

1. Confirm the blood pressure measurement. Check the transducer and/or blood pressure cuff.
2. Evaluate the ABCs, vital signs, temperature, and end-tidal CO₂. Pay particular attention to the patient's cardiac rate and rhythm.
3. Place the patient on 100% oxygen.
4. Decrease the volatile inhalational agent, if tolerated.
5. Increase IV fluid administration until the etiology is determined with consideration for colloid solution or blood products.
6. Evaluate the surgical field and consult with the surgeon to evaluate bleeding and IVC compression.
7. Review the history and physical exam.
8. Consider placement of invasive hemodynamic monitors and transesophageal echocardiography, if available.
9. Stop infusions that may cause vasodilation.

- a. Stop any anti-hypertensive agents such as beta-blockers, calcium channel blockers, nitroglycerin, nitropruside, isoproterenol, and fenoldopam.
10. Start inotropic therapy (bolus and/or infusion).
 - a. Phenylephrine
 - i. α_1 receptor agonist
 - ii. Increases systemic vascular resistance (SVR) and causes a reflex decrease in the heart rate.
 - iii. Can be given either as a bolus or an infusion.
 - b. Ephedrine
 - i. α_1, β_1 , and β_2 receptor agonist
 - ii. Increases the blood pressure, heart rate, contractility, and cardiac output.
 - iii. Usually used as a bolus agent.
 - c. Epinephrine
 - i. $\alpha_1, \alpha_2, \beta_1$, and β_2 receptor agonist
 - ii. Increases blood pressure, heart rate, contractility, and myocardial oxygen demand.
 - iii. Used as a bolus and infusion.
 - d. Norepinephrine
 - i. α_1, α_2 and β_1 receptor agonist
 - ii. Causes an increase in SVR, contractility, and afterload.
 - iii. Reflex decrease in heart rate
 - iv. Used as bolus and infusion.
 - e. Dopamine
 - i. Moderate doses (2–10 mcg/kg/min) cause β_1 receptor stimulation.
 - ii. Higher doses (10–20 mcg/kg/min) cause a more prominent α_1 receptor effect.
 - iii. Used as an infusion.
 - f. Dobutamine
 - i. A relatively selective β_1 agonist.
 - ii. Mainly increases blood pressure and cardiac output by increasing myocardial contractility.
 - g. Milrinone
 - i. Phosphodiesterate inhibitor
 - ii. Leads to an increase in cyclic-AMP.
 - iii. This results in an increase in myocardial contractility and cardiac output.
 - iv. There is a decrease in SVR.
11. Placing the patient in head-down tilt or Trendelenburg position is controversial. It does increase the central blood volume and transiently increases the cardiac output but ultimately activates baroreceptors and can worsen peripheral vasodilation.¹

Case Discussion

The case presented is classic for an embolism from the cement used for the femoral prosthesis. The patient also may have suffered from a fat embolism secondary to the femur fracture. The treatment for this patient would be supportive care. The first mode of action should be to immediately call for help. Inform the surgeon of the likely diagnosis. The blood pressure

in this scenario had been re-checked. Provide 100% oxygen. Decrease or stop all volatile anesthetics. Palpate for a carotid pulse. If there is no palpable pulse, call for the crash cart and begin ACLS protocol. Intravenous fluids should be given “wide open.” Place an arterial line and obtain an arterial blood gas. If the arterial line is difficult, the arterial blood gas should be drawn first. Consider placing a central line to monitor the central venous pressure (CVP). The patient should be taken to the intensive care unit (ICU) immediately post-operatively with continued mechanical ventilation.

Post-operative

1. Confirm the blood pressure measurement. Check the transducer and/or blood pressure cuff.
2. Evaluate the patient, perform a physical exam, and check ABCs.
3. Provide supplemental oxygen.
4. Increase IV fluid administration as hypovolemia is the most common etiology of hypotension in the post-operative period.

5. Check the cardiac rate and rhythm.
6. Investigate any recent drug administration and the anesthesia record.
7. Stop any infusion that may cause vasodilatation.
8. Consider a 12-lead EKG, arterial blood gas, complete blood count, and chest radiograph.
9. Aim therapy at the etiology. Perform any maneuver that may help with venous return or help alleviate symptoms if present.
10. If crystalloid solution is not sufficient, consider plasma expanders or blood products.
11. Inotropic therapy
12. If fluid administration does not improve the blood pressure, myocardial dysfunction may be a possibility. Monitor for myocardial ischemia and end-organ damage.²

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 644.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, p 2716.

28. Hypertension (HTN)^{1,2,3,4,5,6}

Robert C. Lee, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 48-year-old female is scheduled for a laparoscopic cholecystectomy. She has history of hypertension, diabetes mellitus type II, and hyperlipidemia. She is taking lisinopril, metoprolol, metformin, and atorvastatin. She states that she did not take any of her medications this morning. Her pre-operative blood pressure is 181/112 mm Hg. A repeat blood pressure is 184/117 mm Hg. She states she is a little nervous and denies any other symptoms. General endotracheal anesthesia is performed uneventfully using propofol, fentanyl, and vecuronium. An arterial line is placed. Shortly after skin incision, her blood pressure increases from 130/80 mm Hg to 170/100 mm Hg. What is your differential diagnosis? What steps would you take to determine the cause of the intraoperative hypertension? Would you be concerned with the patient's pre-operative blood pressure? Is there a specific blood pressure reading that would cause you to postpone the surgery?

CLINICAL ISSUES

Definition of Hypertension

1. An adult whose systemic blood pressure is greater than 140/90 mm Hg on at least two different occasions measured more than 1 week apart is considered to be hypertensive.¹
2. Hypertension can be further broken down into stages.
 - a. Normal: less than 120/80 mm Hg
 - b. Pre-hypertension: 120–130/80–89 mm Hg
 - c. Stage 1: 140–159/90–99 mm Hg
 - d. Stage 2: 160–179/100–109 mm Hg
 - e. Stage 3: greater than or equal to 180/110 mm Hg
3. A hypertensive crisis occurs when the blood pressure rises to greater than 180/120 mm Hg.²
4. Hypertensive urgency: the patient is not showing evidence of target organ damage.²
5. Hypertensive emergency: the patient develops signs or symptoms of target organ damage.²

28. Hypertension

Types of Target End-Organ Damage

The highest risk of death is seen in patients with systolic arterial pressures greater than 180 mm Hg.⁵

1. Ischemic heart disease
2. Congestive heart failure (CHF)
3. Cerebrovascular accident (CVA)
4. Arterial aneurysm
5. End-stage renal disease

Differential Diagnosis

TKO: This list is extremely important for the oral board exam. In almost every exam, at some point, the patient will become hypertensive. You should have this list memorized so that you can easily determine the possible causes of the patient's HTN and be able to quickly treat the problem.

1. Pre-existing HTN and end-organ dysfunction of the brain, heart, and kidneys
2. "White coat" hypertension
3. Pulmonary: hypoxia, hypercarbia, pulmonary edema, obstructive sleep apnea
4. Renal: renovascular disease, renal parenchymal disease, renin-secreting tumor, polycystic kidney disease
5. Neurologic: elevated intracranial pressure (ICP), spinal cord injury, Guillain-Barré syndrome, dysautonomia
6. Cardiac: ischemia, stiff vessels, aortic coarctation, fluid overload
7. Endocrine: Cushing's syndrome, pheochromocytoma, thyrotoxicosis, hyperaldosteronism, hyperparathyroidism
8. Vascular: coarctation of the aorta, vasculitis, collagen vascular disease
9. Drugs: vasopressors, cocaine, monoamine oxidase inhibitors +/- tyramine, tricyclic antidepressants, naloxone, glucocorticoids, mineralocorticoids, oral contraceptives, withdrawal from anti-hypertensive therapy, and withdrawal from drugs of abuse
10. Pain, anxiety, inadequate anesthesia
11. Bladder distension
12. Malignant hyperthermia
13. Hypothermia
14. Electrolyte abnormalities: hypercalcemia, hypoglycemia
15. Autonomic instability

Risk Factors for HTN

1. African American race
2. HTN in father and/or mother
3. Excess alcohol intake
4. Obesity
5. Dyslipidemia



KO TREATMENT PLAN

Pre-operative

1. Confirm the blood pressure measurement. Check the blood pressure cuff to ensure it is the appropriate size.
2. Evaluate the patient, assess patient's ABCs (airway, breathing, and circulation), and look for evidence of end-organ damage.
3. Review the history and pharmacology of the drugs that the patient is on for blood pressure control. Check to see what drugs the patient has been taking during the peri-operative period, including the day of surgery.
4. The decision of whether to cancel a case is highly debatable. Delaying the surgery for the purpose of blood pressure control may not be necessary, especially in mild to moderate hypertension. It is recommended that an elective case should be postponed if the hypertensive patient exhibits signs of target organ damage that can be improved with a delay, or if the damage requires further evaluation prior to the surgery.⁴ Strict care should be taken to ensure peri-operative hemodynamic stability because labile hemodynamics, rather than pre-operative hypertension, appear to be more closely related with adverse cardiovascular complications.⁴
 - a. Review blood pressure readings over the past few weeks and look for a trend.
 - b. Is the patient compliant with her medications?
 - c. Is her blood pressure elevated today only because she is nervous and also did not take her medications?
 - d. Counsel the patient on the importance of good blood pressure control with respect to the risks of anesthesia, including heart attack, stroke, arrhythmias, and death.
 - e. Many patients will choose to delay the surgery to establish better blood pressure control.
 - f. If the case is an emergency, consider the placement of a pre-induction arterial line to help titrate in anti-hypertensive agents.
 - g. A very old study conducted in the 1970s recommended canceling all elective cases with a diastolic blood pressure >110 mm Hg. Current research, medications, and anesthetic techniques have made this cutoff obsolete. Each case must be evaluated on an individual basis.
 - h. According to the American College of Cardiology/American Heart Association (ACC/AHA), it is recommended that for a systolic blood pressure of >180 mm Hg and a diastolic blood pressure of >110 mm Hg the potential benefits of delaying the surgery to optimize the effects of the anti-hypertensive medications should be weighed against the risk of delaying the surgical procedure.⁶ With rapid-acting intravenous agents, the blood pressure can be controlled within hours.⁶ An attempt should be made to treat these blood pressures with anti-hypertensive medications prior to going into the operating room.

28. Hypertension

i. The intra-operative arterial blood pressure should be maintained within 20% of the best estimate of pre-operative arterial pressure, especially in patients with markedly elevated pre-operative pressures.⁵

Induction

1. Expect an exaggerated blood pressure (both hypo- and hypertensive) response to the anesthetic drugs and increased cardiovascular lability.

a. A patient with chronic hypertension will have an associated increase in systemic vascular resistance (SVR) and a relative hypovolemia.

b. The anesthetics will cause systemic vasodilatation and associated hypotension.

c. Patients with chronic hypertension also have a more vigorous response to laryngoscopy and intubation than normotensive or controlled hypertensive patients.⁵

2. Consider maneuvers to suppress tracheal reflexes and blunt the sympathetic responses to tracheal intubation by using lidocaine, opioids, β -blockers, or vasodilators.

3. Attempt to limit the duration of tracheal manipulation.

4. Consider pre-induction invasive hemodynamic monitors, such as an arterial line.

Intra-operative

1. Confirm the blood pressure measurement. Check the transducer and/or blood pressure cuff.

2. Evaluate ABCs, heart rate, and end-tidal CO₂. Check all of the vital signs.

3. Verify adequate oxygenation and ventilation. Place the patient on 100% oxygen.

4. Review the history and physical exam to guide your differential diagnosis.

5. Consider placement of invasive hemodynamic monitors.

6. Anti-hypertensive agents

a. β_1 -adrenergic receptor blocker

i. Esmolol, metoprolol, labetalol

ii. Decreases heart rate and peripheral vascular resistance

iii. Avoid if heart rate is <60 beats per minute

iv. Use with caution in asthmatics.

(1) It can induce bronchospasm in severe asthmatics.

(2) Usually safe for mild to moderate asthmatics.

b. α_1 -receptor blocker

i. Hydralazine has a slower onset (10–20 minutes) and longer duration (2–4 hours) compared to beta-blockers.

ii. The associated baroreceptor reflex may increase heart rate and cardiac output.

c. α_2 receptor agonist

i. Clonidine and dexmedetomidine

ii. Decreases SVR and heart rate.

iii. Some sedative properties

d. Vasodilators

i. Nitroglycerin is a direct vasodilator that acts on the venous system more than the arterial system. It reduces preload, myocardial oxygen demand, and platelet aggregation.

ii. Sodium nitroprusside is a balanced arteriolar and venous dilator that results in a decreased SVR. Reflex tachycardia and possible intracoronary steal of blood from ischemic areas of the heart can be seen.

7. Monitor for myocardial ischemia.

a. Pay close attention to the EKG and look for ST segment depressions or elevations.

b. Transesophageal echocardiography (TEE) is helpful to evaluate for intra-operative wall motion abnormalities.

Post-operative

1. Confirm the blood pressure measurement. Check the transducer and/or blood pressure cuff.

2. Evaluate ABCs.

3. Review the history and physical exam.

4. Rule out the most common etiologies such as pain, hypercapnia, hypoxemia, urinary retention, or excessive intravascular fluid volume.³

5. Anti-hypertensive therapy should be given with the goal to gradually resume the patient's regimen of oral anti-hypertensive drugs.

6. Monitor for signs of end-organ damage.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 475–501.
2. Hines, Marschall. *Stoelting's Anesthesia and Co-Existing Disease*, 5th ed. Philadelphia: Churchill Livingstone, 2008, p 92.
3. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, p. 2717.
4. Hanada S, Kawakami H, Goto T, et al. Hypertension and anesthesia. *Current Opinion in Anaesthesiology* 19(3):315–9.
5. Howell SJ, Sear JW, and Foex P. Hypertension, hypertensive heart disease, and perioperative cardiac risk. *British Journal of Anaesthesia (BJA)*, 2004, 92(4):570–83.
6. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 Guidelines on the Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery: Executive Summary; *Circulation* 2007; 116:000–000.

29. Arrhythmias^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

An 80-year-old male just woke up after an emergency pinning of his left hip. You are called to the recovery room because the patient's heart rate suddenly increased from 80 to 160 beats/minute. How would you evaluate? What would you do if his blood pressure (BP) was 120/80 mm Hg? What if his BP was 60/30 mm Hg?

CLINICAL ISSUES

Diagnosis of Arrhythmias

1. Classified by heart rate (HR).
 - a. Bradyarrhythmia (HR < 60 beats/minute)
 - b. Tachyarrhythmia (HR > 100 beats/minute)
 - c. Conduction blocks (HR at any rate)
2. Classified by the anatomic origin within the heart
 - a. Ventricular
 - b. Supraventricular
 - c. Junctional
 - d. Elsewhere

Intra-operative Factors Contributing to Arrhythmias

1. General anesthetics
 - a. Volatile anesthetics can produce an arrhythmia through a re-entrant mechanism.
 - b. Halothane sensitizes the myocardium to endogenous catecholamines.
 - c. Cocaine and ketamine block the reuptake of norepinephrine and can facilitate the development of epinephrine-induced arrhythmias.
2. Local anesthetics
 - a. Pharmacological sympathectomy leading to a parasympathetic dominance and bradycardia
3. Abnormal arterial blood gases and electrolytes
 - a. Abnormalities in pH
 - b. Hypoxia/hypercarbia
4. Sympathetic response to endotracheal intubation
5. Reflexes
 - a. Vagal: sinus bradycardia, atrioventricular block, or asystole
 - b. Carotid sinus stimulation: bradycardia
 - c. Oculocardiac reflex: bradycardia or asystole

6. Central nervous system stimulation
7. Dysfunction of the autonomic nervous system
8. Pre-existing cardiac disease
 - a. Myocardial ischemia/infarction
 - b. Congestive heart failure (CHF)
 - c. Cardiomyopathy
 - d. Valvular disease
 - e. Conduction system abnormalities
9. Central venous cannulation
 - a. Insertion of the wire or catheter into central circulation
10. Surgical manipulation of the cardiac structures
 - a. Atrial sutures
 - b. Venous bypass cannulas
11. Location of the surgery
 - a. Dental
 - i. Profound stimulation of the sympathetic and parasympathetic nervous system.
 - ii. Trigeminal (5th cranial) nerve stimulation leading to stimulation of the autonomic nervous system
12. Pain
13. Hypovolemia
14. Hypotension
15. Anemia
16. Endocrine abnormalities
 - a. Hyperthyroidism
 - b. Pheochromocytoma
17. Temperature abnormalities
 - a. Hyperthermia
 - b. Hypothermia



KO TREATMENT PLAN

Continued Assessment of an Arrhythmia

1. What is the arrhythmia?
 - a. Usually best evaluated in limb lead II because it has the largest P wave.
 - b. What is the heart rate?
 - c. Is the rhythm regular?
 - d. Is there one P wave for each QRS complex?
 - e. Is the QRS complex normal?
 - f. Is the rhythm dangerous?
2. Does it produce a hemodynamic disturbance?
3. What treatment is required?

29. Arrhythmias

4. How urgently does it need to be treated?
 - a. Treatment should be initiated immediately if the arrhythmia is associated with marked hemodynamic impairment.
 - b. Treatment should be started promptly if the arrhythmia is a precursor to a more severe arrhythmia.
 - c. Treatment should also be initiated if the arrhythmia is detrimental to the patient's underlying cardiac disease.
 - i. Tachycardia in a patient with mitral stenosis

ASYSTOLE AND PULSELESS ELECTRICAL ACTIVITY (PEA)

CLINICAL ISSUES

Definitions

1. Asystole
 - a. No discernible electrical activity on the EKG
 - b. Always confirm by checking the monitor for loose leads, no power, too low a signal gain, or operator error.
 - c. Very poor prognosis
 - d. Asystole is the end point rhythm for patients initially in ventricular fibrillation or ventricular tachycardia.
2. PEA
 - a. Heterogeneous group of rhythms that are organized or semi-organized but lack a palpable pulse
 - b. It is any organized rhythm without a pulse except for ventricular fibrillation, ventricular tachycardia, or asystole.
 - c. A poor outcome is likely unless diagnosed and treated quickly and aggressively.

Causes

1. Asystole and PEA²
 - a. Hypovolemia
 - b. Hypoxia
 - c. Acidosis
 - d. Hyper- and hypokalemia
 - e. Hypoglycemia
 - f. Hypothermia
 - g. Toxins/tablets
 - h. Tamponade (cardiac)
 - i. Tension pneumothorax
 - j. Thrombosis
 - k. Trauma



KO TREATMENT PLAN

1. Call for help.
2. Cardiopulmonary resuscitation (CPR)
3. Oxygen

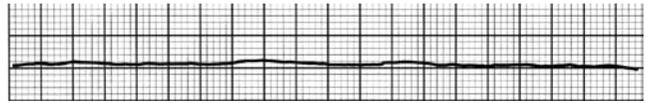


Figure 29.1. Asystole. Drawing credit: J. Lovich-Sapola, MD.

4. Attach the monitor/defibrillator.
5. Resume CPR.
6. Epinephrine 1 mg intravenous or intraosseous (IV/IO) (may repeat every 3–5 minutes) or one dose of vasopressin 40 units IV/IO
7. Atropine 1 mg IV/IO (may repeat every 3–5 minutes) if the PEA rate is slow
8. Look for and treat the underlying cause.
9. There is no evidence that attempting to defibrillate or transcutaneously pace asystole or PEA is helpful.

SINUS BRADYCARDIA

CLINICAL ISSUES

Definition

1. Heart rate <60 beats/minute.
2. Heart rate <50 beats/minute in patients with chronic β -blocker therapy.
3. Regular rhythm
4. Normal QRS complex

Side Effects of Bradycardia

1. Decreased cardiac output
2. Decreased blood pressure
3. Syncope, vertigo, lightheadedness, and dizziness

Causes

1. Hypoxia/hypercarbia
2. Drug effects
 - a. Opioids
 - b. β -blockers
 - c. Succinylcholine
 - d. Anticholinesterase inhibitors
 - e. Anesthetic overdose
3. Acute inferior myocardial infarct
4. Vagal stimulation
 - a. Oculo-cardiac reflex
 - b. Visceral stimulation
5. High sympathetic blockade
 - a. High spinal
 - b. Spinal shock
6. Acidosis
7. Allergic reaction
8. Hypertension



Figure 29.2. Sinus bradycardia. Drawing credit: J. Lovich-Sapola, MD.

- 9. Increased intracranial pressure (ICP): Cushing’s response
- 10. Baseline slow heart rate: well-trained athletes may have large stroke volumes and large cardiac output reserve that allow for normal resting bradycardia.



KO TREATMENT PLAN

1. Check all vital signs.
2. Check the baseline heart rate.
3. Ensure a secure airway with adequate oxygenation and ventilation.
4. Obtain a 12-lead EKG.
5. Heart rates <40 beats/minute are poorly tolerated even in healthy patients.
6. Treatment is recommended if hypotension, ventricular arrhythmias, or signs of poor peripheral perfusion are observed.
7. Atropine 0.5–1.0 mg IV bolus repeated every 3–5 minutes, up to 0.04 mg/kg
8. Ephedrine 5–10 mg IV bolus
9. Dopamine 5–20 mcg/kg/minute IV infusion
10. Isoproterenol 2–10 mcg/minute IV infusion
11. Temporary transcutaneous pacing or transvenous pacemaker: this should be done immediately if the patient is symptomatic.

SINUS TACHYCARDIA

CLINICAL ISSUES

Definition

1. Most commonly occurring arrhythmia in the peri-operative period.
2. Heart rate 100–160 beats/minute.
3. Regular rhythm
4. Normal QRS complex
5. Normal P wave

Causes

1. Hypoxia/hypercapnia
2. Pain/anxiety
3. Inadequate anesthesia
4. Hypovolemia/anemia

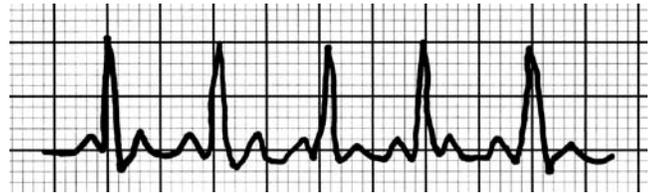


Figure 29.3. Sinus tachycardia. Drawing credit: J. Lovich-Sapola, MD.

5. Fever/malignant hyperthermia/sepsis
6. Congestive heart failure
7. Drug effect
 - a. Catecholamines
 - b. Pancuronium
 - c. Anticholinergics
 - d. Vasodilators
8. Endocrine abnormalities
 - a. Hyperthyroidism
 - b. Thyrotoxicosis
 - c. Pheochromocytoma
9. Electrolyte abnormalities
 - a. Hypoglycemia
10. Surgery
11. Pulmonary embolism
12. Pneumothorax
13. Pacemaker malfunction
14. Drug withdrawal
15. Bladder distension



KO TREATMENT PLAN

Stable Tachycardia

1. Check all vital signs.
2. Check an EKG.
3. Check the baseline heart rate.
4. Check the oxygenation and ventilation.
5. Prolonged tachycardia can precipitate congestive heart failure.
6. Tachycardia decreases coronary perfusion time.
7. Treat the underlying disorder.
8. β-blockers in patients with underlying ischemic heart disease

Unstable Tachycardia with a Pulse

1. A patient has unstable tachycardia if he or she
 - a. Is hypotensive
 - b. Shows signs of shock
 - c. Has an altered mental status
 - d. Has ongoing chest discomfort
 - e. Has shortness of breath
 - f. Has syncope

29. Arrhythmias

2. Support the airway, breathing, and circulation.
3. Give oxygen.
4. Check all vital signs.
5. Establish IV access.
6. Synchronized cardioversion: 100, 200, 300, then 360 joules

PREMATURE ATRIAL CONTRACTIONS (PAC)

CLINICAL ISSUES

Definition

1. An ectopic pacemaker site in the left or right atrium initiates the atrial premature beat.
2. The P wave will have a different shape from the usual.
3. PR interval will vary depending on the site of the ectopic focus.
4. It has a normal sinus cycle with no compensatory pause.
5. The heart rate is variable, but usually less than 100 beats/minute.
6. The rhythm is irregular.



KO TREATMENT PLAN

1. Rarely needed
2. Digitalis, β -blockers, or verapamil may be considered if hemodynamic function is impaired.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

CLINICAL ISSUES

Definition

1. Rapid, regular rhythm
2. Narrow QRS complex
3. Lacks the normal sinoatrial node P wave.
4. Usually abrupt in onset and termination.
5. Heart rate is 130–270 beats/minute.

Associated with the Following

1. Seen in 5% of normal adults and in patients with Wolff-Parkinson-White Syndrome.
2. Intrinsic heart disease
3. Systemic illness
4. Thyrotoxicosis
5. Digitalis toxicity
6. Pulmonary embolism
7. Pregnancy
8. Changes in the autonomic nervous system
9. Drug effect
10. Intravascular volume shifts



Figure 29.4. PAC. Drawing credit: J. Lovich-Sapola, MD.

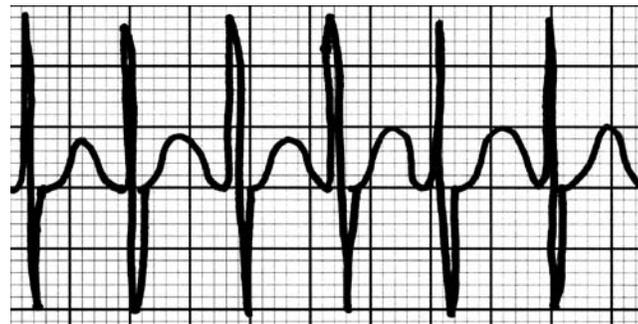


Figure 29.5. PSVT. Drawing credit: J. Lovich-Sapola, MD.



KO TREATMENT PLAN

1. Often must be treated because of its rapid rate and associated poor hemodynamic function.
2. Vagal maneuvers
3. Give adenosine 6 mg rapid IV bolus, followed by a second and third dose if necessary at 12–18 mg per bolus.
4. Verapamil 2.5–10 mg IV
5. Amiodarone 150 mg IV infusion over 10 minutes
6. Esmolol 1mg/kg bolus IV and 50–200 mg/kg/minute infusion
7. Edrophonium 5–10 mg IV bolus
8. Give phenylephrine 100 mcg IV if the patient is hypotensive.
9. Digoxin 0.5–1.0 mg IV
10. Rapid overt pacing in an attempt to capture the ectopic focus
11. Synchronized cardioversion in incremental doses of 50, 100, 200, 300, and 360 joules: this should be done immediately if the patient is hemodynamically unstable.

ATRIAL FLUTTER

CLINICAL ISSUES

Definition

1. Classic sawtooth flutter waves
2. Atrial rate is 250–350 beats/minute.
3. Ventricular rate is usually about 100–150 beats/minute.
4. Rhythm is regular.
5. The QRS complex is normal.
6. Macro-reentrant arrhythmia



Figure 29.6. Atrial flutter. Drawing credit: J. Lovich-Sapola, MD.

Causes

1. Severe heart disease
2. Coronary artery disease
3. Mitral valve disease
4. Pulmonary embolism
5. Hyperthyroidism
6. Cardiac trauma
7. Cancers of the heart
8. Myocarditis



KO TREATMENT PLAN

Hemodynamically Stable

1. Start pharmacologic or synchronized cardioversion after ruling out the risk of a thromboembolic event.
2. Control the ventricular response rate by slowing the conduction through the atrioventricular node.
3. β -blockers such as esmolol and propranolol
4. Calcium channel blockers such as verapamil or diltiazem

Hemodynamically Unstable

1. Start synchronized DC cardioversion with 100 joules, gradually increasing to 360 joules if needed.
2. Procainamide 5–10 mg/kg IV loading dose with a 0.5 mg/kg/minute infusion
3. Rapid atrial pacing from within the atrium

ATRIAL FIBRILLATION

CLINICAL ISSUES

Definition

1. Excessively rapid and irregular atrial focus with no P waves on the EKG
2. Irregularly irregular rhythm
3. Atrial rate 350–500 beats/minute
4. Ventricular rate 60–170 beats/minute
5. P waves are absent.
6. QRS complex is normal.



Figure 29.7. Atrial fibrillation. Drawing credit: J. Lovich-Sapola, MD.

Causes

1. Cardiac disease
2. Other causes are similar to those associated with atrial flutter.

Clinical Significance

1. Loss of atrial “kick” may reduce the ventricular filling and significantly compromise cardiac output by 10–20%.
2. May be associated with atrial thrombi after 24 hours, which can result in pulmonary and systemic embolization.



KO TREATMENT PLAN

Acute Atrial Fibrillation

1. IV diltiazem or esmolol
2. Start synchronized cardioversion in patients with pronounced hemodynamic instability. Start with 100–200 joules, then 300 joules, then 360 joules.
3. If atrial fibrillation has been present for over 48 hours, there is an increased risk of thromboembolism.
 - a. Consider a transesophageal echocardiogram (TEE) to rule out an atrial thrombus.
 - b. Adequate anticoagulation for 3–4 weeks should be considered prior to cardioversion if a thrombus is present.

Long-Term Therapy

1. Anticoagulation with warfarin
2. β -blockers, calcium channel blockers, and digitalis can be used to control the heart rate if it is >100 beats/minute.
3. Electrode catheter ablation of the atrioventricular junction and permanent pacemaker insertion
4. Implanted atrial defibrillators are indicated for patients who go in and out of atrial fibrillation.

Prevention of Recurrence

1. Quinidine
2. Flecainide
3. Sotalol
4. Amiodarone
5. Dofetilide

JUNCTIONAL RHYTHMS

CLINICAL ISSUES

Definition

1. Cells in the atrioventricular (AV) node are not able to act as pacemakers.
2. The ectopic activity is initiated by sites just superior or inferior to the AV node.
3. P waves are abnormal.
4. Variable heart rate of 40–180 beats/minute
5. Regular rhythm
6. QRS complex is normal.

Causes

1. Junctional rhythms are common under general anesthesia, especially with halogenated anesthetic agents.

Significance

1. Frequently decrease blood pressure and cardiac output by about 15%.



KO TREATMENT PLAN

1. Usually requires no treatment
2. If the patient becomes hypotensive and has poor perfusion, treatment is indicated.
3. Amiodarone is the drug of choice.
4. IV atropine, ephedrine, or isoproterenol can be used in an effort to increase the activity of the sinoatrial (SA) node.
5. A small 10 mg dose of succinylcholine while under general anesthesia may revert a nodal rhythm to a sinus rhythm.
6. Dual chamber pacing at a rate faster than a slow nodal rhythm.

PREMATURE VENTRICULAR CONTRACTIONS (PVC)

CLINICAL ISSUES

Definition

1. Ectopic pacemaker activity arising inferior to the AV junction.
2. Wide QRS complex (>0.12 seconds)

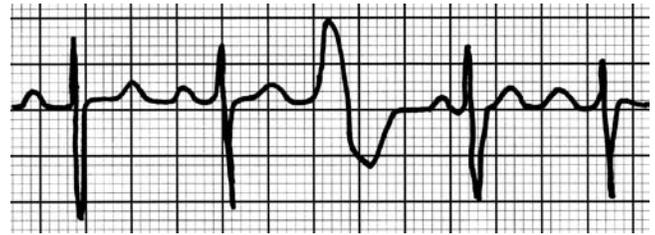


Figure 29.8. PVC. Drawing credit: J. Lovich-Sapola, MD.

3. No P wave
4. Rhythm is irregular.

Causes

1. Common during anesthesia, especially in patients with pre-existing cardiac disease.
2. Electrolyte and blood gas abnormalities
3. Drug interactions
4. Brainstem stimulation
5. Trauma to the heart

Significance

1. May be a life-threatening event.
2. The arrhythmia may progress to ventricular tachycardia or fibrillation.



KO TREATMENT PLAN

1. Correct the underlying abnormality, such as decreased serum potassium or low arterial oxygen tension.
 - a. Check arterial blood gas, electrolytes, EKG, and chest X-ray.
2. If greater than 6 PVCs per minute, treat with a lidocaine 1.5 mg/kg IV bolus.
3. Recurrent ventricular premature beats can be treated with an IV infusion of lidocaine at 1–4 mg/minute, esmolol, propranolol, procainamide, quinidine, atropine, verapamil, or overdrive pacing.

VENTRICULAR TACHYCARDIA (VT) WITH A PULSE

CLINICAL ISSUES

Definition

1. The presence of 3 or more PVCs.
2. The presence of a fusion beat, capture beat, and atrioventricular dissociation.
3. Heart rate of 100–250 beats/minute.



Figure 29.9. Ventricular tachycardia. Drawing credit: J. Lovich-Sapola, MD.

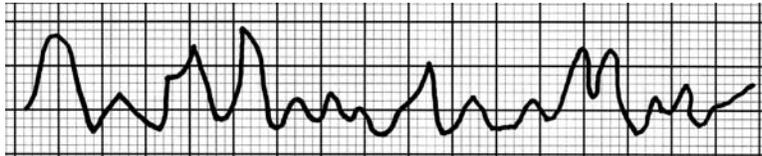


Figure 29.10. Ventricular fibrillation. Drawing credit: J. Lovich-Sapola, MD.



Figure 29.11. Torsades de pointes. Drawing credit: J. Lovich-Sapola, MD.

- 4. Usually a regular rhythm but can be irregular.
- 5. Wide QRS complex greater than 0.12 seconds.

- 4. P waves are not visible.
- 5. Rapid, grossly disorganized rhythm
- 6. QRS is not seen.

Significance

- 1. Acute onset is life threatening and requires immediate treatment.

Causes

- 1. Myocardial ischemia
- 2. Hypoxia
- 3. Hypothermia
- 4. Electric shock
- 5. Electrolyte imbalance
- 6. Drug effect



KO TREATMENT PLAN

- 1. Amiodarone 150 mg IV infusion over 10 minutes
- 2. Synchronized cardioversion: start with 100-200 joules, then 300 joules, then 360 joules.

Significance

- 1. No effective cardiac output.
- 2. Life must be sustained by artificial means.

VENTRICULAR FIBRILLATION (VF)

CLINICAL ISSUES

Definition

- 1. Irregular rhythm
- 2. Erratic ventricular contractions
- 3. The EKG has a bizarre pattern of various sizes



KO TREATMENT PLAN FOR VF AND PULSELESS VT

- 1. CPR
- 2. Oxygen
- 3. Attach the monitors/defibrillator.

4. Asynchronous defibrillation (one shock with 120–200 joules for biphasic defibrillator or 360 joules for a monophasic defibrillator)
5. Resume CPR.
6. Continue to alternate one shock and CPR as long as the patient maintains a shockable rhythm.
7. Once the patient has an IV, give epinephrine 1 mg IV/IO (may repeat every 3–5 minutes) or vasopressin 40 units IV/IO.
8. Consider an anti-arrhythmic such as
 - a. Amiodarone 300 mg IV/IO once, then consider an additional dose of 150 mg IV/IO.
 - b. Lidocaine 1–1.5 mg/kg first dose, then 0.5–0.75 mg/kg IV/IO, with a maximum of 3 doses or 3 mg/kg.
 - c. Magnesium 1–2 grams IV/IO for torsades de pointes.
9. After 5 cycles or CPR/shock, reevaluate the rhythm.

TORSADES DE POINTES

CLINICAL ISSUES

Definition

1. A ventricular tachycardia with a prolonged QT.
2. Rapid, polymorphic ventricular tachycardia with a characteristic twist of the QRS complex around the isoelectric baseline.
3. May mimic VF.
4. It is a life-threatening arrhythmia.

5. Rate is usually 150–250 beats/minute.
6. No atrial component.
7. P wave is buried in the QRS.
8. Ventricular rhythm is regular or irregular.

Causes

1. Occurs in the presence of disturbed repolarization: prolonged QT.
2. Seen also with electrolyte disturbances such as hypokalemia, hypocalcemia, and hypomagnesemia.



KO TREATMENT PLAN

1. Discontinue the drugs that led to the prolonged QT and correct any electrolyte abnormality.
2. Asynchronous defibrillation
3. 1–2 grams IV magnesium sulfate, IV amiodarone, and/or IV isoproterenol
4. Overdrive pacing

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1397–1409.
2. Field JM. *American Heart Association 2005 Guidelines for Advanced Cardiovascular Life Support*. American Heart Association, 2006.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1540–5.

30. Cardiac Conduction Blocks^{1,2,3,4,5,6,7}

Cristian M. Prada, MD

SAMPLE CASE

A 15-year-old female is scheduled for a mastoidectomy. Her mother reports that she was 8 weeks premature and has significant developmental delays. She is followed closely at an outside hospital for her primary care. Her mother also tells you that she was recently diagnosed with a second-degree heart block (Mobitz type II). She hands you a copy of the cardiologist's note. He recommends a follow up appointment and EKG in 3 months, but no medications or pacemaker. The patient is currently asymptomatic. Are you concerned? How will you treat this patient? Would you place external pacemaker pads? Would you like another cardiology consult?

CLINICAL ISSUES

Definitions

1. Conduction systems of the heart
 - a. Sinoatrial (SA) node
 - b. Atrioventricular (AV) node
 - c. His-Purkinje system (AV bundle [of His], Purkinje fibers of the right and left bundle branches)
2. Heart block above the AV node is transient and benign; below the AV node is more progressive and permanent.
3. Elderly patients
 - a. Increase in the incidence of bradydysrhythmias and conduction abnormalities

30. Cardiac Conduction Blocks

- i. Caused by a progressive fibrosis in both the SA and the AV conduction system¹
- ii. For the SA node: fat accumulation, which separates it from the atria musculature¹
- b. SA pacemaker cells decrease progressively from 60 years of age: at 75 years old, approximately 10% of the original numbers of cells are present.¹
- c. Bradydysrhythmias may be present pre-operatively but can have an unexpected onset as heart block under general anesthesia, mainly in the elderly population.

SINUS NODE BLOCK

- 1. Failure of the normal pacemaker of the SA node
- 2. This rarely causes symptoms, because if the patient has a complete block at the SA node, the secondary pacemaker of the heart would be the AV node, which depolarizes at 40–60 beats/minute.
 - a. Sinus node arrest
 - i. Absence of spontaneous depolarization
 - ii. Prolonged sinus pauses caused by failure of sinus impulse formation
 - b. Sinus node exit block
 - i. No electrical propagation
 - ii. Block of conduction of sinus impulses from the SA node to the surrounding atrial tissue
 - c. Atrial tissue failure
 - i. Depolarization does not reach the AV node.
 - d. Sick sinus syndrome
 - i. A combination of symptoms caused by SA node dysfunction and manifested by marked bradycardia, sino-atrial block, or sinus arrest
 - ii. Can have associated episodes of supraventricular tachycardia; often called bradycardia-tachycardia syndrome
 - (1) Dizziness
 - (2) Confusion
 - (3) Fatigue
 - (4) Syncope
 - (5) Congestive heart failure (CHF)
- iii. Treatment
 - (1) Atrial or dual-chamber pacemaker
 - (2) These patients are at a high risk of developing a pulmonary embolism and should be started on anti-coagulation therapy.³

ATRIOVENTRICULAR NODE BLOCK

- 1. Presence of P waves without ventricular activation
- 2. Temporary or permanent; anatomic or functional impairment of conduction
- 3. Classification: first-, second-, or third- (complete) degree block
 - a. First-degree block:
 - i. Prolonged PR interval >200 msec on a 12-lead EKG
 - ii. Benign

- (1) Sometimes significant bradycardia can occur during spinal anesthesia.
- (2) Rare progression to higher degrees of block; reported only with spinal or general anesthesia²
- (3) If the PR interval is >400 msec, then an apparent AV dyssynchrony and higher grade AV block can develop if the atrial rate increases (exercise, trauma, anemia), because not all of the atrial impulses are conducted to the ventricles.

iii. Causes

- (1) AV nodal disease
- (2) Enhanced vagal tone (athlete)
- (3) Myocarditis
- (4) Acute myocardial infarction
- (5) Electrolyte disturbances
- (6) Medication
 - (a) Calcium channel blockers
 - (b) β -blockers
 - (c) Cardiac glycosides
 - (d) Cholinesterase inhibitors
 - (e) Digitalis

iv. Treatment depends on symptomatology but can include

- (1) Correction of any electrolyte imbalances
- (2) Withholding of any offending medications
- (3) Admit the patient to the coronary care unit if it is associated with a myocardial infarction; otherwise, continue with outpatient follow-up.

b. Second-degree block

- i. Some impulses are blocked.
 - (1) Mobitz I (Wenckebach): the PR interval progressively lengthens until the ventricles fail to activate (P wave not followed by QRS).
 - (a) Disease of the AV node
 - (b) If the patient is hemodynamically stable, the treatment is observation only.
 - (c) This is usually a benign condition.
 - (2) Mobitz II: intermittently non-conducted P waves are not preceded by PR prolongation.
 - (a) Disease along the His-Purkinje system
 - (b) May progress rapidly to a complete heart block.
 - (i) Cardiac arrest and sudden death
 - (c) May occur with an antero-septal infarct.
 - (d) Treatment: pacemaker

c. Third-degree block:

- i. No atrial impulses are conducted to the ventricles.
- ii. Complete heart block
- iii. P wave without relation to the QRS complexes
- iv. Causes
 - (1) Coronary ischemia
 - (2) Congenital: lupus
- v. Treatment
 - (1) Dual chamber pacemaker
- vi. Treatment if the patient is acutely hemodynamically unstable:

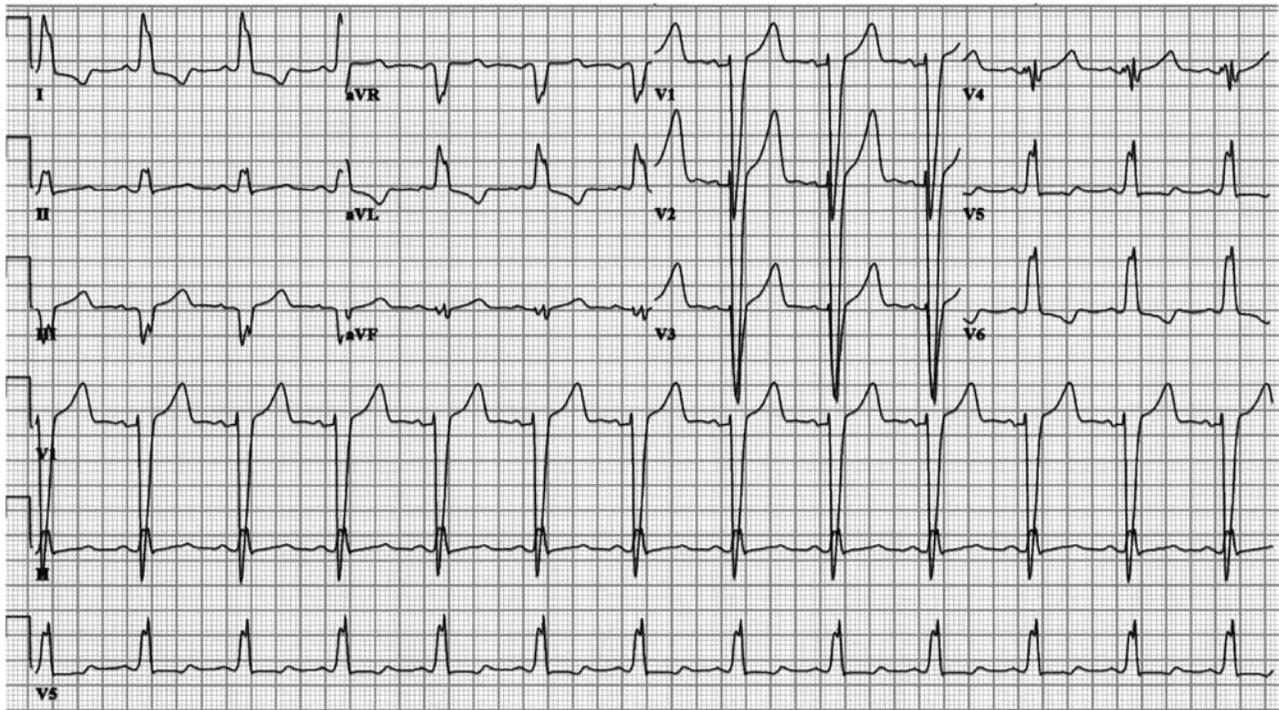


Figure 30.1. Left bundle branch block: EKG.

- (1) Pharmacologic: continuous infusion of isoproterenol; possibly atropine, ephedrine, epinephrine
 - (a) Glycopyrrolate is indicated only for vagally induced bradycardia.
 - (b) These treatments can sometimes cause uncontrolled sinus tachycardia.
- (2) Pacing: transcutaneous, transvenous, transthoracic, or transesophageal

BUNDLE BRANCH BLOCKS (FASCICULAR BLOCKS)

1. Left bundle branch block (see Figure 30.1)

- a. Activation of the left ventricle is delayed, which results in the left ventricle contracting later than the right.
 - i. Usually indicates an underlying cardiac pathology
 - (1) Dilated or hypertrophic cardiomyopathy
 - (2) Hypertension
 - (3) Aortic valve disease
 - (4) Coronary artery disease: myocardial infarction
- b. Causes
 - i. Hypertension
 - ii. Myocardial infarction
 - iii. Excessive coronary artery disease
 - iv. Primary conduction defect
- c. Diagnosis
 - i. Heart rhythm must be supraventricular in origin.
 - ii. The QRS duration must be ≥ 120 msec
 - iii. There should be a QS or RS complex in lead V1.
 - iv. There should be a monophasic R wave in leads I and V6.

- (1) R, R' in the left chest leads (V5 or V6)

v. The T wave should be deflected opposite the terminal deflection of the QRS complex.

d. Treatment

- i. Requires a complete cardiac evaluation.
- ii. Patients with syncope, CHF, and prolonged QRS may require a pacemaker.

2. Right bundle branch block (see Figure 30.2)

- a. The right ventricle is not directly activated by impulses traveling through the right bundle branch.
 - i. First the left ventricle receives the electrical impulse, then the right ventricle.
 - ii. The left ventricle is normally activated by the left bundle branch.
 - iii. These impulses travel through the myocardium of the left ventricle and activate the right ventricle.
 - iv. New onset with symptoms
 - (1) Screen for pulmonary embolism, chronic lung disease, cardiomyopathy, congestive heart failure, atrial and ventricular septal defects, Ebstein anomaly, blunt chest trauma, and polymyositis.
- b. Causes
 - i. Central line placement (right side)
 - ii. Increased prevalence with increased age
 - (1) Usually a marker of slow progressive degenerative disease
 - iii. Can be a normal variant in healthy patients
- c. Diagnosis
 - i. The heart rhythm must be supraventricular in origin

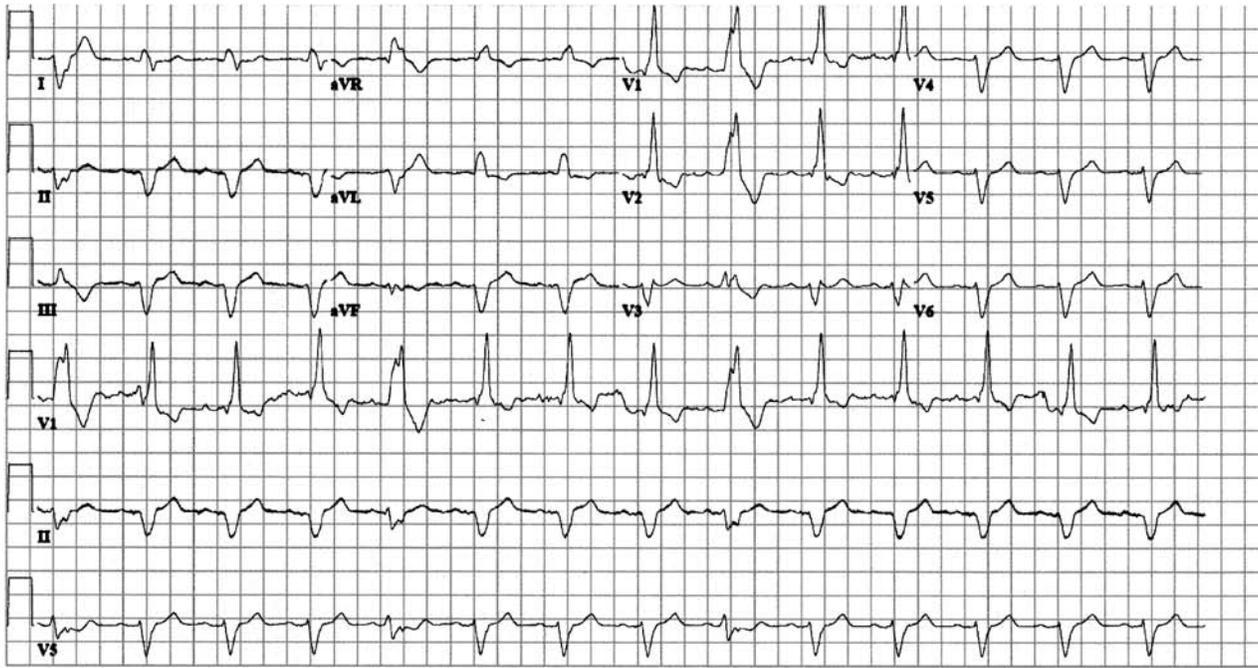


Figure 30.2. Right bundle branch block: EKG.

- ii. The QRS duration must be ≥ 120 msec
- iii. There should be a terminal R wave in lead V1.
 - (1) R, R' in the right chest leads (V1 or V2)
- iv. There should be a slurred S wave in leads I and V6.
- v. The T wave should be deflected opposite the terminal deflection of the QRS complex.
- d. Treatment
 - i. Generally asymptomatic and does not require treatment.
 - ii. Pacemaker if syncope occurs



KO TREATMENT PLAN

Pre-operative

1. The patient was diagnosed with Mobitz II second-degree heart block.
 - a. May progress to complete heart block.
 - b. Usually treated with a pacemaker
2. Since the surgery is elective, I would postpone the surgery to get a second opinion cardiology consult.
3. If the surgery suddenly became an emergency
 - a. Place external pacer pads.
 - b. Have the pacer/defibrillator in the operating room.

Intra-operative

1. Standard ASA monitoring
2. Begin general anesthesia with an endotracheal tube so that the patient already has a secure airway if she goes into complete heart block and requires cardiac pacing.

Post-operative

1. Maintain the pacer pads in the post-anesthesia care unit.
2. Close observation on a telemetry or coronary care unit floor
3. Order a cardiology consult for close follow-up management.

BIBLIOGRAPHY

1. Longnecker DE, Brown D, Newman M, et al. *Anesthesiology*. New York: McGraw Hill. 2008, pp 349.
2. Lobato EB, Gravenstein N, Kirby RR. *Complications in Anesthesiology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 256-9.
3. Miller RD, Cucchiara RF, Miller ED, et al. *Anesthesia*, 5th ed. New York: Churchill Livingstone. 2000, pp 271-5.
4. Yao FSF, Fontes ML, Malhotra V. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*, 6th ed. Philadelphia: Lippincott Williams & Wilkins. 2008, pp 231-2.
5. Management of symptomatic bradycardia and tachycardia. *Circulation* 2005;112:67-77.
6. Suzuki M, et al. Association between right bundle branch block and impaired myocardial tissue-level reperfusion in patients with acute myocardial infarction. *J Am Coll Cardiol*, 2006; 47:2122-4.
7. Dubin D. *Rapid Interpretation of EKG's 6th ed*. Tampa: Cover, 2000, pp 174-94.

31. Acute Coronary Syndrome^{1,2,3,4,5}

Cristian M. Prada, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 68-year-old female, 231 lbs and 5'1" tall, with a history of hypertension (HTN), diabetes mellitus, and large joint chronic arthritis, is scheduled for a laparoscopic cholecystectomy. The patient is non-compliant with her treatment: atenolol and glyburide. On the morning of the surgery, the patient's blood pressure (BP) was 145/86 mm Hg, heart rate (HR) 88, respiratory rate (RR) 20, oxygen saturation 97% on room air, and temperature 36.8 °C. Her blood glucose was 186 mg/dL. Physical examination revealed no abnormalities and the airway was assessed as a Mallampati class II. After a smooth induction of general anesthesia with midazolam, fentanyl, propofol, and rocuronium, a #7.0 endotracheal tube was placed atraumatically. Anesthesia was maintained with mechanical ventilation, isoflurane, oxygen, air, fentanyl boluses, and rocuronium. About 30 minutes after the incision, the patient's HR increased to 112 beats/minute and her BP became 184/99 mm Hg. The anesthesiologist also noticed a depression of the ST segment in the monitored V5 cardiac lead.

What would you do? What treatment would you give? Could this event have been prevented? Would you extubate this patient? What is your plan for the post-operative care of this patient?

CLINICAL ISSUES

Definitions⁴

1. Acute coronary syndrome
 - a. Patient with coronary atherosclerosis
 - b. Spectrum of clinical syndromes
 - i. Unstable angina
 - ii. Non-ST-segment elevation myocardial infarction (NSTEMI)
 - iii. ST-segment elevation myocardial infarction (STEMI)
 - iv. Sudden cardiac death
 - c. Varying degree of coronary occlusion
2. Unstable angina
 - a. Prolonged episodes of substernal chest pain or stable angina that has been increasing in frequency or severity
 - b. A type of acute coronary syndrome
 - c. Partially occluding thrombus

- d. Produces symptoms of ischemia.
- e. Symptoms may occur at rest.
- f. Treatment: antiplatelet agents
 - i. Aspirin
 - ii. Clopidogrel (Plavix)
 - iii. Glycoprotein IIb/IIIa receptor inhibitors
 - (1) Fibrinolytic therapy is not effective.
- g. Additional treatment
 - i. Oxygen
 - ii. Nitroglycerine (sublingual or intravenous)
 - iii. β -blockers
 - iv. Heparin

Mechanism of Cardiac Ischemia¹

1. Imbalance between the supply and demand of oxygen to the myocardium
 - a. Supply of oxygen to the myocardium: determined by coronary perfusion pressure and the duration of the diastole
 - i. Coronary perfusion pressure is maintained by
 - (1) Normal to high diastolic arterial pressure
 - (2) Normal to low left ventricular end-diastolic pressure (LVEDP)
 - ii. Increased length of diastole increases the supply of oxygen.
 - b. Demand of oxygen by the myocardium is determined by
 - i. Heart rate
 - (1) Decreasing heart rate
 - (a) Increases oxygen supply by prolonging diastole
 - (b) Decreases oxygen demand: less work
 - (c) Avoid severe bradycardia: causes decreased diastolic arterial pressure and increased LVEDP.
 - (d) Use β -blockers.
 - ii. Contractility: difficult to measure; even by cardiac output or left ventricular ejection fraction
 - (1) Decreased contractility may be beneficial in coronary artery disease (CAD) as long as coronary perfusion pressure is maintained.
 - (2) Potent volatile anesthetic agents: isoflurane and halothane

31. Acute Coronary Syndrome

(a) Myocardial depressants are useful for patients with CAD, as long as the coronary perfusion pressure is maintained.

iii. Myocardial wall tension

(1) Increased during systole against high afterloads, resulting in higher oxygen demand, despite the increase in coronary perfusion pressure

Factors That Influence Peri-operative Myocardial Ischemia and Infarction²

1. Increased myocardial oxygen demand
 - a. Heart rate
 - i. Maintain a normal rate and rhythm.
 - b. Blood pressure
 - i. Maintain a normal blood pressure within 20% of the peri-operative mean.
 - c. Surgical stress and post-operative pain can lead to a hyperadrenergic state that can result in tachycardia and increased oxygen demand.
2. Decreased myocardial oxygen supply occurs with improper ventilation of the patient.
3. Coronary artery spasm
4. Anesthetic effects: see below
5. Rupture of atherosclerotic plaque: coronary thrombosis
6. Coagulation abnormalities: surgically induced
 - a. Prothrombotic state secondary to the stress response and direct activation of the coagulation cascade by the surgical injury
 - b. Platelet activation

Cardiac Cascade²

1. Progressive sequence of patho-physiologic events triggered by myocardial ischemia: coronary artery occlusion
2. Diastolic abnormalities: increase in LVEDP
3. Systolic abnormalities: regional wall motion abnormalities
4. Hemodynamic abnormalities
5. EKG changes
6. Angina: symptoms may not occur (silent MI).

TKO: Reversal of the ischemia signs is not in the same temporal order: the stunned myocardium may have wall motion abnormalities despite EKG signs of resolution.²

1. High likelihood: if the patient has any of the findings below
 - a. History
 - i. Chest or left arm pain or discomfort plus
 - (1) Current pain is similar to prior angina
 - (2) Known CAD, MI
 - b. Physical exam
 - i. Transient mitral regurgitation
 - ii. Hypotension

iii. Diaphoresis

iv. Pulmonary edema or rales

c. EKG

i. New transient ST deviation (≥ 0.5 mm)

ii. T-wave inversion (≥ 2 mm) with symptoms

d. Cardiac markers

i. Elevated troponin I or T

ii. Elevated CK-MB

2. Intermediate likelihood: if the patient has no high likelihood findings and has any of the findings below

a. History

i. Chest or left arm pain or discomfort plus

(1) Age >70 years

(2) Male sex

(3) Diabetes mellitus

b. Physical exam

i. Extracardiac vascular disease

c. EKG

i. Fixed Q waves

ii. Abnormal ST segments or T waves that are not new

d. Cardiac markers

i. Any finding in the intermediate likelihood category plus normal cardiac enzymes

3. Low likelihood: if the patient has no high or intermediate risk findings and has any of the findings below

a. History

i. Probable ischemic symptoms

ii. Recent cocaine use

b. Physical exam

i. Chest discomfort reproduced by palpitation (15% of patients with musculoskeletal pain will have an MI)

c. EKG

i. Normal EKG

ii. T-wave flattening

iii. T-wave inversion in leads with dominant R waves

d. Cardiac markers

i. Normal

Clinical Tools to Detect Ischemia^{2,4}

1. EKG

a. Most useful

b. Non-invasive

c. Continuous monitor

i. Lead changes and the associated artery and area of damage

(1) V_1 - V_2

(a) Left coronary artery

(i) Left anterior descending artery (LAD) and septal branch

(b) Septum, AV bundle, and bundle branches

(c) Complications: infranodal block and bundle branch blocks (BBB)

(2) V_3 - V_4

(a) Left coronary artery

(i) LAD and diagonal branch

31. Acute Coronary Syndrome

- (b) Anterior wall of the left ventricle (LV)
 - (c) Complications: LV dysfunction, congestive heart failure (CHF), BBBs, complete heart block, and premature ventricular complex (PVC)
 - (d) Associated with a poor prognosis and sudden death
 - (3) V_5 - V_6 plus I and aVL
 - (a) Left coronary artery
 - (i) Circumflex branch
 - (b) High lateral wall LV
 - (c) Complications: LV dysfunction and atrioventricular (AV) nodal block
 - (4) II, III, aVF
 - (a) Right coronary artery
 - (i) Posterior descending branch
 - (b) Inferior wall LV and posterior wall LV
 - (c) Complications: hypotension and sensitivity to nitroglycerine and morphine sulfate
 - (5) V_4 R
 - (a) Right coronary artery
 - (i) Proximal branches
 - (b) Right ventricular (RV), inferior wall LV, and posterior wall LV
 - (c) Complications: hypotension, supranodal and AV-nodal blocks, atrial fibrillation/flutter, premature atrial complex (PAC), and adverse medical reactions
 - (6) V_1 - V_4 (marked depression)
 - (a) Either left coronary artery (circumflex) or right coronary artery (posterior descending branch)
 - (b) Posterior wall LV
 - (c) Complication: LV dysfunction
2. Transesophageal echocardiography (TEE)
- a. Most sensitive
 - i. Reduction of the normal degree of thickening across the ventricular wall during systole (systolic wall thickening): the most sensitive marker of ischemia
 - b. Most recognizable sign: reduction in movement of the ventricular wall toward the center of the ventricle (endocardial excursion)
 - c. Wall motion abnormalities: not always specific – e.g., hypovolemic or hypervolemic states
3. Pulmonary artery (PA) catheterization
- a. Information about systolic dysfunction, diastolic dysfunction, and mitral regurgitation
 - b. Questionable sensitivity and safety of catheterization

Initial Management of an Acute Coronary Syndrome⁴

1. Immediate assessment
- a. Vital signs
 - b. Oxygen saturation
 - c. Obtain intravenous (IV) access
 - d. 12-lead EKG
 - e. Brief targeted history
 - f. Fibrinolytic checklist
- g. Cardiac markers
- h. Electrolytes and coagulation studies
- i. Portable chest X-ray
- j. Assess for
- i. $HR \geq 100$
 - ii. Systolic blood pressure (SBP) ≤ 100 mm Hg
 - iii. Pulmonary edema (rales)
 - iv. Signs of shock
2. Immediate treatment
- a. Oxygen
- i. May limit ischemic myocardial injury, reducing the amount of ST-segment elevation.
- b. Nitroglycerin
- i. Dilates the coronary arteries and vascular smooth muscle in veins, arteries, and arterioles.
 - ii. Reduces ischemic pain.
 - (1) Routes
 - (a) Sublingual (SL) tablet: 0.4 mg, may repeat 2 times every 3–5 minutes.
 - (b) Sublingual Spray: 1 or 2 sprays repeated times 2 at 3–5 minute intervals
 - (c) Intravenous (IV): 12.5–25 ug bolus, 0.5–10 ug/kg/minute infusion
 - (2) Treatment goals
 - (a) Relief of ischemic discomfort
 - (b) Limit the blood pressure drop
 - (3) Contraindications/cautions
 - (a) SBP < 90 mm Hg
 - (b) Severe bradycardia ($HR < 50$)
 - (c) Tachycardia ($HR > 100$)
 - (d) RV infarction
 - (e) Recent use (within 24–48 hours) of phosphodiesterase inhibitor for erectile dysfunction
- c. Aspirin
- i. Inhibits thromboxane A_2 platelet aggregation to reduce coronary reocclusion and recurrent events after fibrinolytic therapy.
 - ii. Effective for unstable angina
 - iii. Recommendations
 - (1) For all patients with acute coronary syndrome unless they have a true aspirin allergy
 - (a) Aspirin allergy: consider clopidogrel.
 - (2) 160–325 mg of nonenteric coated aspirin orally: crushed or chewed
 - (a) Rectal suppository if the patient has nausea/vomiting or peptic ulcer disease
 - iv. Cautions
 - (1) Active peptic ulcer disease
 - (2) True allergy
 - (3) Bleeding disorders
 - (4) Severe hepatic disease
- d. Morphine
- i. Dilates the arteries and veins so it redistributes blood volume, reduces ventricular preload and afterload, and can reduce pulmonary edema.
 - ii. The analgesic effects reduce chest pain.

31. Acute Coronary Syndrome

- iii. It decreases oxygen requirements.
- iv. Recommendations
 - (1) 2–4 mg IV
 - (2) May give an additional dose of 2–8 mg IV at 5 to 15 minute intervals.
- v. Indicated for patients with ischemic pain not relieved by nitroglycerin and acute coronary syndrome without hypotension
- vi. May be useful to redistribute blood volume in patients with pulmonary edema.
- vii. Cautions
 - (1) Do not use in patients with hypotension or suspected hypovolemia.
 - (2) If hypotension develops without pulmonary edema, elevate the patient's legs and give a 250–500 ml bolus of IV normal saline.

Triage⁴

1. ST-segment elevation or a new left bundle branch block (LBBB)
 - a. High specificity for an evolving STEMI
 - b. Treat with β -blockers, clopidogrel, and heparin.
 - c. Evaluate for reperfusion candidacy if \leq to 12 hours from the onset of symptoms.
 - d. Admit to a monitored bed, assess risk status, and consider cardiac catheterization if it has been >12 hours from the onset of symptoms.
 - i. Start statin and ACE inhibitor/ARB therapy.
2. ST-segment depression or dynamic T-wave inversion
 - a. Strongly suggestive of ischemia
 - b. High risk subset of patients with unstable angina/NSTEMI
 - c. Start treatment with nitroglycerin, β -blockers, clopidogrel, heparin, and glycoprotein IIb/IIIa receptor inhibitor.
 - d. Admit to a monitored bed.
 - e. Assess risk status.
 - i. Consider cardiac catheterization.
 - ii. Start statin and ACE inhibitor/ARB therapy.
3. Nondiagnostic or normal EKG
 - a. If the patient continues to have chest pain
 - i. Repeat the EKG.
 - ii. Order serial cardiac markers.
 - iii. Myocardial imaging/2-d echocardiography
 - iv. Continue medical observation.

Fibrinolytic Therapy⁴

1. Goals
 - a. Limit infarct size.
 - b. Preserve LV function.
 - c. Reduce mortality.
 - d. Door-to-drug time ≤ 30 minutes
2. Most effective in the following patients
 - a. ST-segment elevation or a new LBBB
 - b. Early presentation

- c. Larger infarction
 - d. Younger patients with a lower risk of intracerebral hemorrhage
3. Can be harmful
 - a. ST-segment depression
 - b. Patients >24 hours after the onset of pain
 - c. Presence of high blood pressure (SBP >175 mm Hg) on presentation denotes an increased risk of stroke after fibrinolytic therapy.
 4. Absolute contraindications
 - a. Any prior intracranial hemorrhage
 - b. Known structural cerebral vascular lesion
 - c. Known malignant intracranial neoplasm
 - d. Ischemic stroke within 3 months (unless it is acute, within 3 hours)
 - e. Suspected aortic dissection
 - f. Active bleeding
 - g. Significant closed head trauma within 3 months
 5. Relative contraindications
 - a. History of chronic, severe, poorly controlled hypertension
 - b. Traumatic or prolonged CPR (>10 minutes)
 - c. Recent internal bleeding (2–4 weeks)
 - d. Noncompressible vascular puncture
 - e. Pregnancy
 - f. Active peptic ulcer disease
 - g. Current anticoagulant use



KO TREATMENT PLAN

Pre-operative

1. Complete history and physical
 - a. Functional capacity
2. Cardiac history
 - a. Chest pain
 - b. Shortness of breath
 - c. Peripheral edema
3. ACC/AHA guidelines for the sample case
 - a. Intermediate risk surgery
 - b. Minor clinical risk secondary to her uncontrolled hypertension
 - i. These guideline require no further cardiac work-up unless patient had symptoms of cardiac ischemia (i.e., chest pain).
4. Pre-operative medications
 - a. β -blockade
 - i. Clinical evidence shows that patients who are appropriately beta-blocked before the surgery have less ischemia than those who are not or who receive only calcium channel blockers; this appears to be related to less tachycardia in β -blocked patients.²
 - b. Short acting calcium channel blockers
 - i. Patients who are on short-acting calcium channel blockers in the pre-operative period have resulted in worse outcomes after cardiac ischemia.

31. Acute Coronary Syndrome

(1) Possibly due to the resulting negative inotropic effect

(2) However, they may be beneficial in preventing stunned myocardium.²

c. Nitrates

i. There are no good data available for the use of pre-operative nitrate treatment in the prevention of myocardial ischemia.

(1) Rarely used, except in patients with high risk: patients with acute unstable angina requiring non-cardiac surgery²

Intra-operative

1. Rapid assessment of which determinants of the myocardial O₂ balance have been compromised

a. In most instances: tachycardia

b. This patient became tachycardic and hypertensive.

2. Improve the patient's hemodynamics.

a. This may require deepening of patient's anesthesia: light anesthesia could lead to hypertension, tachycardia, and cardiac ischemia.

3. Evaluate her IV access.

4. 100% oxygen

5. Medications

a. Nitroglycerine

b. Morphine

c. B-blockers

i. Initially give short-acting esmolol.

ii. Effects

(1) Decreased heart rate

6. 12-lead EKG

7. Labs

a. Cardiac markers

b. Complete blood count

c. Electrolytes

d. Coagulation panel

8. Chest X-ray

9. Inform the surgeon: cancel or finish the case ASAP.

10. Look for resolution or worsening of the ST-depression.

11. Consult cardiology.

12. If the patient becomes hemodynamically unstable, consider TEE for further cardiac evaluation.

13. This situation may have been prevented with better hemodynamic maintenance during the surgery. According to the ACC/AHA guidelines, the patient did not require any further pre-operative workup.

Preferred Anesthetic Technique for a High-Cardiac Risk Patient

1. There is no evidence that a specific anesthetic technique is associated with a better cardiac outcome.²

2. Regional versus general anesthesia for the at-risk (myocardial ischemia) patients in the non-cardiac surgical setting

a. Regional anesthesia

i. Advantages

(1) Stress response is attenuated or blocked.

(a) Thoracic epidurals may suppress the stress response to surgery and reduce myocardial oxygen demand better than lumbar epidural anesthesia.²

(2) Vasodilatation

(a) During vascular surgery, the vasodilatation can facilitate surgical anastomoses and graft perfusion.

(3) Decreased risk of deep vein thrombosis

ii. Disadvantages

(1) Spontaneous ventilation during lengthy surgery in a compromised patient

(2) Hypotension and bradycardia from a high sympathectomy

b. Inhalational anesthetic effects²

i. Common potent volatile anesthetic effects

(1) Dose-related decrease in arterial blood pressure

(2) Decreased oxygen consumption

(3) Increase in coronary blood flow

(4) Studies have shown the volatile anesthetics to all be essentially similar in terms of myocardial ischemia and adverse cardiac outcomes, assuming the patients received opioids during the anesthetic.

ii. Halothane

(1) Decrease in myocardial contractility

(a) Increase in right atrial pressure

(2) Decrease in cardiac output

(a) Decrease in blood pressure

(b) Decreases myocardial and peripheral O₂ demand

(c) Reduces the risk of ischemia, unless there is a severe decrease in cardiac output.

(3) Stable heart rate

(4) Decreased contractility

(a) Greater decrease in contractility than the new volatile anesthetics

(5) Can predispose patients to ventricular arrhythmias.

(6) Sensitizes the heart to the arrhythmogenic effects of epinephrine.

ii. Isoflurane

(1) Maintains cardiac output.

(2) Increase in heart rate by 10–20% at 1 MAC

(3) Less negative inotropic effects than halothane except in the elderly

(4) Potent peripheral and coronary vasodilator

(5) No effect on cardiac conduction system

(6) Theoretical concerns about “coronary steal syndrome”

iii. Desflurane

(1) Maintains cardiac output.

(2) Increased heart rate

(a) Increase of 10–20% at greater than 1 MAC

- (b) The increase in heart rate is specifically associated with rapid increases in the inspired concentration.
- (c) The associated tachycardia can be lessened by pretreatment with narcotics or clonidine.
- (3) Rapid increase in inhaled concentration also results in hypertension.
 - (a) During induction, a 1.0 to 1.5 MAC (end-tidal 7.5–10.9%) can lead to sympathetic hyperactivity and myocardial ischemia in patients with coronary artery disease.²
 - (b) Lower concentrations, such as 0.55–0.83 MAC (end-tidal 4–6%) often result in no clinically significant sympathetic stimulation.²
 - (c) If the sympathetic stimulation is decreased with opioids, propofol, esmolol, or clonidine, then desflurane can be used in patients at risk for myocardial ischemia.
- iv. Sevoflurane
 - (1) Maintains cardiac output and heart rate.
 - (2) Heart rate
 - (a) Does not increase heart rate, BP, or plasma norepinephrine concentrations at 1.5–2.7 MAC.²
 - (3) Cardiac contractility: same effects as isoflurane and desflurane
 - (4) The same incidence of myocardial ischemia in patients with or at high risk for coronary artery disease as isoflurane
 - (5) No sympathetic stimulation: some prefer it for patients with HTN and normal left ventricular function.²
- v. Nitrous oxide
 - (1) Increases sympathetic nervous system activity.
 - (2) Increases vascular resistance at 40% concentration.
- c. Narcotics
 - i. Lack of intrinsic myocardial depression
 - ii. Vagotonic effects

- iii. Minor decrease in blood pressure
- iv. Dose-response relationship between plasma levels of opioid and suppression of adrenergic responses is extremely variable: risk of breakthrough hypertension and tachycardia.
- d. Benzodiazepines: significant hypotension because of peripheral vasodilation

Post-operative

1. Hemodynamically stable
 - a. Resolution of patient's ST-depression
 - b. Extubate
 - i. Closely monitor the patient and treat any hypertension and tachycardia.
 - ii. Be prepared to abort the extubation if the patient develops ST-changes.
 - c. Maintain hemodynamics.
 - d. Send the patient to a cardiac-monitored floor.
 - e. Cardiology consult
2. Hemodynamically unstable
 - a. Keep intubated.
 - b. Coronary care unit (CCU)
 - c. Cardiology consult

BIBLIOGRAPHY

1. Reed AP, Yudkowitz FS. *Clinical Cases in Anesthesia*, 3rd ed. New York: Churchill Livingstone, 2005, pp 11–20.
2. Kirby RR, Gravenstein N, Lobato E, et al. *Clinical Anesthesia Practice*. 2nd ed. Elsevier, 2002, pp 810–26.
3. Stabilization of the patient with acute coronary syndromes. *Circulation*, 2005; 112:IV-89-IV-110. Online version: http://circ.ahajournals.org/cgi/content/full/112/24_suppl/IV-89.
4. Field JM. American Heart Association. Advanced Cardiac Life Support Guidelines. Handbook of Emergency Cardiovascular Care for Healthcare Providers. 2006, pp 22–42.
5. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins. 2006, pp 401–4.

32. Embolism^{1,2,3,4,5}

Cristian M. Prada, MD

SAMPLE CASE

A 73-year-old female underwent a repair of an abdominal aortic aneurysm. She has a history of heavy smoking, severe chronic obstructive pulmonary disease (COPD),

hypertension (HTN), diabetes mellitus, and is non-compliant with her insulin treatment. Toward the end of the surgery, the patient was transferred to another anesthesia provider, who took her to the post-anesthesia care

32. Embolism

unit (PACU) intubated, sedated, and with the arterial line and central venous pressure (CVP) monitors in place. Approximately 15 minutes after the patient's arrival in the PACU, the anesthesiologist was called emergently to see the patient. The patient had become severely hypotensive. After a quick examination, the anesthesiologist noticed that one of the peripheral infusions was placed on an inflated pressure bag and there was no fluid in the bag or in the intravenous (IV) line.

What do you think happened? How would you further examine the patient? What is your treatment?

VENOUS THROMBOEMBOLISM

CLINICAL ISSUES

Definition of Pulmonary Embolism (PE)

1. A thrombotic embolism that originates in the deep veins of the legs or pelvis and embolizes to the pulmonary artery.
2. Presents as asymptomatic to fulminate cardiovascular collapse depending on the amount of clot and the patient's physiologic reserve.
3. A large clot can obstruct the pulmonary artery (PA) causing right heart strain/failure, severe V/Q mismatch, and release of thromboxane and serotonin, which also increase the PA pressure.
4. Unusual to occur intra-operatively – more frequent in the post-operative period.

Physiologic Causes of Venous Thromboembolism

1. Venous stasis, vessel wall damage, and hypercoagulability (Virchow's triad)
2. During general anesthesia
 - a. Vasodilatory effects of the anesthetic drugs
 - b. Immobility of the lower extremities
 - c. Hypercoagulability
 - d. Stress response to surgery
 - e. Direct damage to the venous endothelium during surgery

Signs and Symptoms of Pulmonary Embolism

1. Dyspnea
2. Chest pain
3. Cough
4. Blood-tinged sputum
5. Fever
6. Tachycardia
7. Tachypnea
8. Coarse breath sounds
9. New S4 heart sound
10. Accentuation of the pulmonic component of the S2 heart sound

Diagnosis of Pulmonary Embolism

1. Gold standard is the pulmonary angiogram
2. Laboratory
 - a. Not typically used (including arterial blood gas [ABGs]) for diagnosing or ruling out PE
 - b. Even patients with a normal PaO₂ can still have a PE.²
 - c. A low PaO₂/FiO₂ ratio (less than 200), or D-dimer presence are non-specific.
3. Imaging studies
 - a. V/Q scan for patients with renal failure (avoids the IV contrast used in a pulmonary angiogram)
 - i. Algorithm elaborated by PIOPED (Prospective Investigation of Pulmonary Embolism Diagnosis) study
 - ii. First, use clinical signs and symptoms to give the probability of PE (low, medium, high).
 - iii. Then, factor the symptoms with the results of the V/Q scan (low to high probability of PE).
 - b. Computerized tomography (CT): spiral (helical) CT often replaces the V/Q scans secondary to the speed of the study and the ability to evaluate other potential embolic sources (i.e., legs, pelvis).
 - i. The sensitivities can be up to 90% with a single detector scan.
 - c. CT angiogram combined with CT venography has a higher sensitivity for venous thromboembolism (VTE).
 - i. A negative CT angiogram does not rule out a subsegmental PE.
 - ii. According to the PIOPED II study, the pretest clinical probability of all post-surgical patients is considered to be at least intermediate risk.
 - iii. Only patients with a high pre-test (clinical) probability and a negative CT scan should undergo compression ultrasonography of the extremities to rule out VTE.

Etiology

1. Upper extremity deep venous thrombosis (DVT)
 - a. There is a 9% incidence of symptomatic PE arising from the upper extremities.
 - b. This type of DVT results from
 - i. Central venous catheters
 - ii. Patients with cancer
 - iii. Paget-Schroetter syndrome
 - c. Rarely spontaneous
 - d. It presents clinically with swelling, erythema, and arm pain.
2. Pulmonary emboli
 - a. There is a 90% incidence of a PE arising from extremities (lower or upper) and 10% from pelvic veins, renal veins, inferior vena cava (IVC), or heart.
3. Surgical procedures related to the risk of VTE
 - a. Highest for (in order) total hip arthroplasty, excision/destruction/biopsy of the brain, total knee arthroplasty, abdominal aortic surgery, splenectomy, and coronary artery bypass graft (CABG) surgery.

32. Embolism

Risk Factors for DVT¹

1. Patient-related
 - a. Elderly
 - b. Obesity
 - c. Bedridden patients
 - d. Limb paralysis
 - e. Prolonged immobilization (including long-distance travel by plane or car)
 - f. Pregnancy
 - g. Varicose veins
 - h. Congenital venous malformations
2. Hypercoagulable and inflammatory states
 - a. Factor V Leiden deficiency
 - b. Activated protein C resistance
 - c. Antiphospholipid syndrome
 - d. Dyslipoproteinemia
 - e. Nephrotic syndrome
 - f. Inflammatory bowel disease
 - g. Infection
 - h. Bechet's syndrome
 - i. Myeloproliferative diseases
 - j. Neoplasms
3. Predisposing factors
 - a. Previous VTE
 - b. Trauma
 - c. Respiratory failure
 - d. Paroxysmal nocturnal hemoglobinuria
 - e. Superficial vein thrombosis
 - f. Central venous catheter
 - g. Vena cava filter
 - h. Intravenous drug abuse
4. Medications
 - a. Oral contraceptives
 - b. Hormone replacement therapy
 - c. Heparin-induced thrombocytopenia
 - d. Chemotherapy
 - e. Tamoxifen
 - f. Thalidomide
 - g. Antipsychotics

- i. DVTs of the proximal (above the knee) lower extremities have a high likelihood of pulmonary embolization.
 - (1) These patients usually require therapeutic anticoagulation.
 - (2) If anticoagulation is contraindicated (recent surgery, continued bleeding), consider the insertion of an IVC filter.
 - ii. Also, prophylactic insertion is recommended in morbidly obese or high-risk trauma patients.
2. Treatment of an acute PE
 - a. Therapeutic anticoagulation
 - b. IVC filter
 - c. Clot thrombolysis with tissue plasminogen activator (TPA) or streptokinase/urokinase.
 - d. Surgical embolectomy

Preventive Therapy Related to the Risk of Developing DVT¹

1. Low risk
 - a. Minor surgery
 - b. Age <40 years old
 - c. No risk factors
 - i. Prevention = early mobilization
2. Moderate risk
 - a. Minor surgery (patient with risk factors)
 - b. Age 40–60 years (patient with no risk factors)
 - c. Major surgery (age <40 years old, no risk factors)
 - i. Prevention = UFH every 2 hours, LMWH, or IPC
3. High risk
 - a. Minor surgery (age >60 years old or with risk factors)
 - b. Major surgery (age >40 or with risk factors)
 - i. Prevention = UFH every 8 hours, LMWH, or IPC
4. Highest risk
 - a. Major surgery (age >40 years old and with major risk factors = prior VTE, malignancy, hip or knee replacement, major trauma, hip fracture surgery, spinal cord injury, or thrombophilia)
 - i. Prevention = LMWH or adjust heparin dose

TKO: UFH = unfractionated heparin

LMWH = low molecular weight heparin

IPC = intermittent pneumatic compression devices



KO TREATMENT PLAN

1. Prevent formation of the DVT
 - a. Low molecular weight heparin (LMWH)
 - b. Intermittent pneumatic compression (IPC) devices
 - c. Elastic stockings
 - d. Regional anesthesia
 - i. Only surgical anesthesia from an epidural, but not post-operative analgesia from an epidural, lowers the peri-operative risk of VTE.
 - ii. Evaluate the patient's coagulation status before neuraxial anesthesia.
 - e. Inferior vena cava filters

VENOUS AIR EMBOLISM

CLINICAL ISSUES

Definition

1. Occurs when air enters venous circulation through an incised or cannulated vein.
2. Frequent IV introduction of small amounts of air during injections or infusions of medications

32. Embolism

- a. 100 ml of air per second can flow into a 14 gauge IV catheter with a 5 cm H₂O pressure gradient.
- b. The amount of air needed to cause a problem can be as low as 20 ml.
 - i. The amount of air that can cause complications is relative to the rate of air infusion.

Surgeries and Procedures at Increased Risk of Venous Air Embolism (VAE)¹

1. TURP (trans-urethral resection of prostate) or radical prostatectomy
2. Sitting craniotomy: 76% risk of VAE
3. Spinal surgery: including laminectomy
4. Laparoscopic surgery
5. Endoscopic bowel procedures
6. Hip replacement
7. Arthroscopic joint procedures
8. VATS: video-assisted thoracoscopic surgeries
9. Chest trauma
10. Any procedure with human- or pump-delivered infusions (e.g., angiography)

Physiology of the VAE

1. Slow entrainment of a large volume of air is well tolerated in a dog model.
2. Small amounts of air are well tolerated in healthy patients if the air is isolated to the right heart and pulmonary circulation.
 - a. Even a small amount of air, if it goes into the coronary or cerebral circulation, can cause an embolic event.
3. Large amounts of air (>50 ml) entering rapidly can cause increased pulmonary pressures, hypotension, and eventual cardiac collapse.
4. In an awake and spontaneously ventilating patient, an air embolus will present with
 - a. Dyspnea
 - b. Cyanosis
 - c. Arrhythmias
 - d. Hypotension
 - e. Increased central venous and pulmonary artery pressures
 - f. Decreased cardiac output
 - g. "Mill wheel" murmur
 - h. Cardiac collapse.

Diagnosis

1. Signs of cardiac ischemia
2. Look for signs of increased pulmonary artery pressure (PAP).
3. Hypotension
4. Acute decrease in end-tidal CO₂
 - a. Increase in alveolar dead space and decrease of cardiac output
5. Increased ETN₂ (end-tidal nitrogen)

6. Precordial (right atrium) Doppler ultrasound: detection of "mill-wheel" murmur
7. Air with aspiration of a right atrium (RA) multi-orifice catheter
8. TEE: right ventricular dilation or hypokinesis
 - a. Sometimes presents as pulmonary artery hypertension: but this symptom is non-specific since it is also present in patients with chronic obstructive lung disease.

Monitoring for Patients at High Risk for VAE¹

1. Observation (surgical field)
 - a. Non-invasive
 - b. Not sensitive
2. ETCO₂
 - a. Non-invasive
 - b. Sensitive
 - c. Non-specific
 - d. Widely used
3. Precordial Doppler ultrasound
 - a. Sensitive
 - b. Easy signal detection
 - c. Difficult placement in the prone position, obese patient, or the patient with a chest deformity
 - d. Non-quantitative
4. End-tidal nitrogen
 - a. Specific
 - b. Hypotension lowers sensitivity.
5. Multiorifice right atrial catheter
 - a. Quantitative
 - b. Not good for continuous screening
6. TEE (transesophageal echocardiography)
 - a. Sensitive
 - b. Expensive
 - c. Not widely available

Patient Outcome with a VAE

- a. The outcome depends on the volume of air that enters the pulmonary circulation and, when present, the amount of air that crosses into the systemic arterial circulation and enters the brain.
- b. Large amounts of venous air create an airlock, increase right heart pressures, and may open a patent foramen ovale.

Complications of the VAE¹

1. Pulmonary
 - a. Hypoxemia
 - b. Pulmonary hypertension
 - c. Pulmonary edema
 - d. Hypercarbia
 - e. V/Q mismatch
2. Cardiovascular
 - a. Hypotension
 - b. Arrhythmias

32. Embolism

- c. Myocardial ischemia
 - d. Right heart failure
 - e. Cardiac arrest
3. Central nervous system
- a. Stroke
 - b. Brain edema

Prevention of VAE

1. When cannulating veins: keep vein elevation below the level of the heart.
 - a. Position the patient in the Trendelenburg position for the placement of central catheters in the internal jugular or subclavian veins.
 - b. Removal of large bore catheters
 - i. Should be done with the patient supine.
 - ii. Apply compression to the vein.
2. Monitor the stopcock position and IV line connection to prevent entry of air into lines.
3. Modern IV pumps should have air detection and alarm systems.
4. Patients who are at a high risk for VAE should have precordial Doppler monitoring during the anesthetic.
5. Minimize the elevation of the surgical site from the heart.
6. Keep the patient euvolemic.
7. Avoid medications that increase venous capacitance: nitroglycerin.



KO TREATMENT PLAN

1. Lower the site of air entry to below the heart.
2. Notify the surgeon.
3. Secure the airway and ensure adequate oxygenation and ventilation.
4. Stop the nitrous oxide and place the patient on 100% O₂.
5. Flood the surgical field with saline.
6. Compression of the proximal vein: internal jugular vein in sitting cases
7. Aspiration of right atrial catheter: if present
8. IV saline bolus
9. Support the circulation by starting a vasopressor, if needed.
 - a. Inotropic support may help clear the air bubble.
10. Position the patient in the left lateral decubitus position in an attempt to keep the intraventricular air from entering the pulmonary artery.
 - a. Air rises to the nondependent portion of the ventricle.
 - i. This may help, but you may not be able to maintain this position because of the need to perform chest compressions.
11. Positive end-expiratory pressure (PEEP) does not decrease the incidence of VAE.

TKO: The presented sample case is likely an example of a venous air embolism, due to the injection of air into a venous line under pressure.

1. IV bolus with saline
2. Support the patient's hemodynamics.
3. Aspirate from her CVP catheter
4. Consider placing her in the left lateral decubitus position if she is stable.
5. If she is hemodynamically unstable
 - a. Intubate
 - b. Pressors
 - c. Chest compression (CPR)
 - d. Advanced cardiac life support (ACLS)

FAT VENOUS EMBOLISM/FAT EMBOLISM SYNDROME (FES)

CLINICAL ISSUES

Definition

1. Fat embolism to the venous and pulmonary circulation is common during orthopedic surgeries and in trauma patients.
2. The presence of fat globules that obstruct circulation, usually the result of fractures of a long bone, burns, parturition, or the fatty degeneration of the liver⁴
3. Mechanism
 - a. Disruption of the venous system at the surgical or injury site allows fat from the bone marrow or adipose tissue to enter the venous system.
 - b. Increased intraosseous pressure (which occurs during the placement of rods or nails in bones) pushes the fat into the venous system.
4. Most commonly occurs in hip replacement, knee replacement, and intramedullary nailings of the diaphysis (bone shaft).
5. The course can vary from benign to fulminate pulmonary failure with cardiovascular collapse.
6. The current theory about the mechanism is that it is not a mechanical obstruction of the pulmonary arterial circulation but rather an immune reaction caused by the breakdown of the embolic fat into free fatty acids causing an inflammatory cascade in the pulmonary and systemic circulation.¹
 - a. There is some degree of fat embolism in up to 90% of all long bone fractures and orthopedic procedures that instrument into bone marrow.

Fat Embolism Syndrome (FES)

1. Described more than 100 years ago.
2. Lack of a gold standard for diagnosis.⁵
3. FES occurs in 0.5–2.0% of long bone fractures and 10% of multiple long bone fractures or concomitant pelvic fractures.
4. The mortality in FES is 1% to 20%, with variability depending on the patient's pre-existing co-morbid conditions.

Diagnosis of FES

1. FES is not clinically recognized in most patients because only a few develop the traditional signs and symptoms.
2. Triad
 - a. Hypoxemia
 - b. Neurological abnormalities: altered mental status
 - c. Petechiae: occur 12 to 72 hours after initial trauma or instrumentation

Signs and Symptoms of FES

1. Hypoxia-associated tachypnea
2. Dyspnea
3. Central nervous system (CNS) depression, including lethargy, confusion, seizures, focal deficits (in paradoxical fat embolism)
4. Petechial rash: head, neck, torso, and axilla
5. Fever
6. Tachycardia
7. Retinal fat emboli
8. Decreased end-tidal CO₂
9. Increased pulmonary artery pressure
10. Bronchospasm
11. Hypotension
12. Acute respiratory distress syndrome (ARDS)
13. Disseminated intravascular coagulation (DIC)

Laboratory Findings of FES

1. Lipiduria
2. Fat in the alveoli: bronchio-alveolar lavage
3. Fat in the blood: aspirate of pulmonary blood or systemic blood
4. Fat in the right ventricle: TEE
5. Ground glass opacity: high-resolution computer tomography



KO TREATMENT PLAN

1. Early fracture fixation may prevent FES.
2. Therapy: supportive
3. 50% of patients will require intubation and mechanical ventilation secondary to severe hypoxemia.
4. ARDS: “low stretch protocol” to minimize alveoli trauma
 - a. Goal: to keep the PaO₂ > 60 mm Hg, without excessive O₂ or volutrauma³
 - b. Tidal volume less than 6 mL/kg and plateau pressures less than 30 cm H₂O³
5. The patient will be at an increased risk for anemia and thrombocytopenia, so monitor for DIC.
6. Epinephrine or milrinone can be given to improve right ventricular function.

AMNIOTIC FLUID EMBOLISM SYNDROME (AFES)

CLINICAL ISSUES

Definition

1. Amniotic fluid (including fetal squamous cells, lanugo hair, and mucin) enter the venous system and eventually the pulmonary circulation.
2. The pathogenesis is unclear. It is not a mechanical outflow obstruction but is postulated to be secondary to an immune response to the amniotic fluid contents, or an immune reaction triggered by leukotrienes or arachidonic acid within the fluid.
3. Profound multi-organ failure can occur
 - a. Respiratory failure: intubation and mechanical ventilation
 - b. Cardiogenic shock: vasopressors
 - c. DIC: 83%
4. AFES can occur as early as 20 weeks gestation but is more likely to occur during labor and delivery or in the 48 hours postpartum.
5. AFES can occur after abortions and an amniocentesis.
6. Pulmonary and systemic hypertension can occur during the initial phase of AFES.
 - a. Pulmonary hypertension can worsen right ventricular failure and contribute to low cardiac output, which may resolve quickly, only to be followed by hypotension due to left ventricular failure.
7. The incidence in the United States is 1 in 20,000 to 1 in 30,000 deliveries.
8. The mortality is 16% to 80%, and is a leading cause of perinatal maternal mortality.
 - a. Survivors have a high morbidity due to cerebral hypoxia.

Risk Factors for AFES

1. Tumultuous labor
2. Trauma
3. Multiparity
4. Advanced maternal age
5. Cesarean section
6. Increased gestational age

Signs and Symptoms of AFES

1. Usually presents with an abrupt onset and rapid clinical deterioration
2. Hypoxia
3. Bronchospasm
4. ARDS
5. Pulmonary edema: capillary leak from embolism, left heart failure, and cardiogenic shock
6. Increased pulmonary artery pressure
7. Increased end-tidal CO₂
8. Hypotension (cardiogenic shock)
9. DIC

10. Altered mental status and seizures
11. Fetal distress
12. Fever
13. Headache
14. Nausea and vomiting

Differential Diagnosis

1. Pulmonary thromboembolism
2. Venous air embolism
3. Hemorrhage
4. Anaphylaxis
5. Transfusion reaction
6. Myocardial infarction

Laboratory Diagnosis

1. Fetal squamous cells in the patient's blood are thought to be pathognomonic for AFES but have been found in women without the syndrome.



KO TREATMENT PLAN

1. There are no known strategies to prevent AFES.
2. The management is supportive.
3. A high percentage of patients require intubation and mechanical ventilation for hypoxia, secondary to the associated pulmonary edema.

- a. Pulmonary edema tends to clear up more rapidly than in patients with typical ARDS.
4. Severe hemodynamic instability of the patient requires invasive monitoring and vasopressors.
5. Laboratory studies for coagulopathies/DIC are required to determine the need for blood, plasma, or platelet transfusion.
6. Parturients who have not delivered will require immediate delivery of the fetus to prevent further hypoxic damage to the fetus and facilitate cardiopulmonary resuscitation of the mother.
7. Surviving AFES patients have a high incidence of neurological deficits due to the associated hypoxia.

BIBLIOGRAPHY

1. Lobato EB, Gravenstein N, Kirby RR. *Complications in Anesthesiology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 193-203.
2. Richard C, Ricome JL, Rimalho A, et al. Fatal pulmonary embolism with a normal PaO₂. *Ann Med Interne (Paris)* 1983;134(6):559-62.
3. Critical Care Medicine Tutorials - http://www.ccmtutorials.com/rs/ali/03_alitreat.htm
4. <http://medical-dictionary.thefreedictionary.com/fat+embolism>.
5. Talbot M, Schemitsch EH. Fat embolism syndrome: history, definition, epidemiology. *Injury*, 2006 Oct;37 Suppl 4;S 3-7.

33. Cardiac Tamponade^{1,2,3,4,5}

Cristian M. Prada, MD

SAMPLE CASE

A 65-year-old female, morbidly obese, smoker (1 pack per day for 35 years), with a history of hypertension (HTN), coronary artery disease (CAD), and diabetes mellitus treated with insulin, underwent an uneventful coronary artery bypass graft (CABG). The patient was taken, sedated and intubated, to the intensive care unit (ICU), with dopamine at 5 mcg/kg/min and insulin 2 I.U./hour as continuous infusions. After three hours in the ICU, the patient suddenly became tachycardic, and her blood pressure decreased dramatically. She had no response to a higher dose of dopamine and norepinephrine infusion. The surgical fellow on call in the ICU performed an emergent needle pericardial aspiration, which removed 100 cc of fresh blood. The patient was taken emergently to the operating room (OR) on a

wide-opened epinephrine drip. After a thoracotomy and pericardiocentesis, the patient's hemodynamic status improved significantly. How would you have prepared this patient for surgery? What would be your choice of induction drugs, and why?

CLINICAL ISSUES

Etiology of Cardiac Tamponade

1. Most often: malignancies and post-cardiectomy
2. Other
 - a. Idiopathic pericarditis
 - b. Uremic pericarditis

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- c. Hemorrhagic
 - i. Post-cardiopulmonary bypass
 - ii. Any cardiac surgery
 - iii. Dissecting aortic aneurysm
 - d. Systemic lupus erythematosus (SLE)
 - e. Rheumatoid arthritis
 - f. Radiation
 - g. Air
3. Acute: occurs within minutes, hours, or less than 5 days post-cardiac surgery.²
4. Delayed: occurs 5–7 days post-cardiac surgery.²

Physiology

1. Tamponade: fluid in the pericardial sac limits filling of the heart
2. Hemodynamic manifestations: mainly due to atrial rather than ventricular compression
3. Initially, diastolic filling is limited, causing a reduced stroke volume that stimulates sympathetic reflexes resulting in increased heart rate and contractility to maintain the cardiac output (CO).
4. Rising right atrial (RA) pressure stimulates reflex tachycardia and peripheral vasoconstriction.
5. Blood pressure is maintained by vasoconstriction, but CO begins to fall as the pericardial fluid increases.
6. The jugular venous pulse has a small Y-descent but a prominent X-descent because the diastolic filling is decreasing.
7. The pericardial pressure-volume curve approaches vertical because any additional fluid greatly restricts cardiac filling and reduces diastolic compliance.
8. RA pressure, pulmonary artery (PA) diastolic pressure, and pulmonary capillary wedge pressure (PCWP) equilibrate to within 5 mm Hg of each other.
9. A precipitous blood pressure drop leads to reduced coronary artery blood flow and subendocardial ischemia.

Diagnostic

1. Triad of acute tamponade
 - a. Decreasing arterial pressure
 - b. Increasing venous pressure
 - c. Small, quiet heart
2. CVP = PAD = PAOP (CVP = central venous pressure; PAD = pulmonary artery diastolic pressure; PAOP = pulmonary artery occlusion pressure)
 - a. This is rarely observed in the post-operative CABG patient because the transected pericardium is left open.²
3. Pulsus paradoxus
 - a. May be seen with cardiac tamponade.
 - b. Defined as a fall in systolic pressure >12 mm Hg during inspiration.
 - c. It is caused by a reduced left ventricular (LV) stroke volume produced by increased filling of the right heart during inspiration.

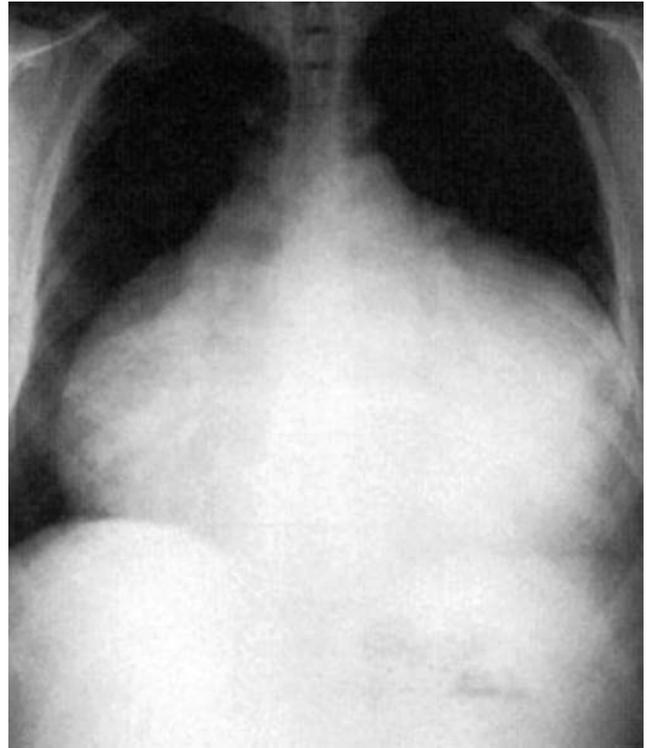


Figure 33.1. Chest X-ray: chronic cardiac tamponade. Source: Used with permission of the American College of Chest Physicians, from *Chest*, Volume 109, Gary S. Kabinoff and Bernard Gitler, Bottle-shaped heart, Page 825, 1996; permission conveyed through Copyright Clearance Center, Inc.

- ii. Pulsus paradoxus may also be present with
 - (1) Obstructive pulmonary disease
 - (2) Right ventricular (RV) infarction
 - (3) Constrictive pericarditis
 - (4) Pulmonary embolism
 - ii. Pulsus paradoxus is absent with
 - (1) LV (left ventricle) dysfunction
 - (2) Positive-pressure ventilation
 - (3) Atrial septal defect (ASD)
 - (4) Severe aortic regurgitation
4. The inspiratory venous pressure remains steady or increases (Kussmaul's sign) rather than decreases.
 5. The EKG can show
 - a. Low-voltage QRS
 - b. Electrical alternans
 - c. T-wave abnormalities
 - d. Sinus rhythm
 6. Echocardiography is the most reliable non-invasive method for the diagnosis of cardiac tamponade.
 - a. Exaggerated motion of the heart within the pericardial sac.
 - b. Diminished left ventricular dimensions and mitral valve excursions during inspiration.²
 - c. Shifting of the interventricular septum toward the left ventricle.²
 - d. Atrial and ventricular collapse.

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- e. Echocardiography can also be used to guide the needle or catheter for aspiration.
- 7. Chest X-ray (anterior-posterior view)
 - a. Normal or extremely enlarged heart, depending on the acuity and chronicity of the tamponade process.²
 - b. A lateral view may also show an enlarged heart.
- 8. Normal pericardial fluid: 15 to 25 mL²
 - a. Tamponade physiology: 150 mL (acute) to 1000 mL (chronic)²
- 9. After cardiac surgery, tamponade due to hemorrhage requires immediate mediastinal exploration.
 - a. It is often difficult to diagnose cardiac tamponade after cardiac surgery because the classic signs are often missing.
 - b. If the patient has a persistently poor CO with increased and equalized RA and LA pressures, strongly consider the possibility of cardiac tamponade.
 - c. An echocardiogram should be performed whenever a patient is not progressing as expected in the post-operative period and has signs of end-organ dysfunction (decrease in urine output, increased blood urea nitrogen [BUN] and creatinine).²



KO TREATMENT PLAN

TKO: Emergency treatment

1. Pericardiocentesis
2. Supportive therapy

Pre-operative Preparation²

1. Complete history, physical examination, and labs
2. Key areas of concern identified and addressed
 - a. Respiratory distress: give supplemental O₂, review the chest X-ray, and order blood gas labs.
 - b. **Positive pressure ventilation: avoid, unless the clinical picture suggests that the patient will develop cardiac arrest.**
 - c. **Oliguria: reflects prerenal or renal (acute tubular necrosis) processes secondary to decreased cardiac output and vasoconstriction.**
 - i. Optimize hemodynamics.
 - ii. Do not give diuretics.
 - iii. The most effective treatment is pericardiocentesis.
 - d. **Elevated prothrombin time (PT) and partial thromboplastin time (PTT)**
 - i. Treat the coagulopathy.
 - ii. Check a complete blood count (CBC).
 - iii. Have 4–6 units of packed red blood cells (PRBCs) in the operating room (OR) before starting surgery.
 - e. Premedication
 - i. Do not give an anxiolytic.

- ii. The most effective and safest technique in this situation to decrease the patient's anxiety is to communicate with the patient.

- f. Sympathoadrenal activation is supporting perfusion of vital organs through selective vasoconstriction and tachycardia.
 - i. Do not give any medications to decrease the patient's tachycardia pre-operatively.
- 3. Communication with the surgical team is very important so that you will be able to follow the surgeon's findings in the operating field and be ready to treat the dramatic hypotension which usually occurs upon opening of the pericardium.

Intra-operative

1. Transport the patient to the OR.
 - a. Monitors
 - i. Standard ASA monitors
 - ii. Pre-induction arterial line
 - b. Oxygen by face mask
 - i. Increase the FiO₂ as needed.
 - c. Have all equipment for intubation immediately available.
 - d. Have a cardioversion unit available.
 - e. Have all emergency drugs available, including
 - i. Phenylephrine (0.1 to 0.5 mg boluses, 25 to 125 mcg/min infusion)
 - ii. Epinephrine (0.01–0.1 mcg/kg/min)
 - iii. Calcium chloride (2–4 mg/kg)
 - iv. Lidocaine (1–2 mg/kg)
 - v. Atropine (0.01 mg/kg)
2. Medications²
 - a. Ketamine: first choice for induction agent in pericardial tamponade
 - i. Induction dose: 1 mg/kg IV
 - ii. Rapid onset
 - iii. Peak plasma concentration is achieved in less than 1 minute.
 - iv. The patient's unconsciousness will last for 10 to 15 minutes, but the analgesic effect (somatic > visceral) will last for an additional 30 minutes or longer.
 - v. Effects: small increases in blood pressure, heart rate, cardiac output, and myocardial oxygen demand
 - vi. Direct central sympathetic stimulation and inhibition of norepinephrine uptake into the sympathetic nerve endings
 - vii. Intrinsic myocardial depressant
 - b. Etomidate: second choice for induction agent in pericardial tamponade
 - i. Preferred when cardiovascular collapse is anticipated
 - ii. Induction dose: 0.2 to 0.3 mg/kg IV
 - iii. No myocardial depression
 - iv. Limiting factors: pain at the injection site, involuntary muscle movements, reduction of the seizure

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threshold, and enhanced duration of the seizure limit its use.

c. Thiopental: not indicated for induction in pericardial tamponade

i. Mild to moderate negative inotropic and vasodilatory effects

ii. 1 to 2 mg/kg does not cause significant hemodynamic disturbance, but do not use it for induction in patients with cardiac tamponade because these patients are close to cardiovascular collapse.

d. Propofol: not indicated for induction in pericardial tamponade

i. Similar effects as thiopental

ii. May cause up to a 30% reduction in systemic blood pressure or even cardiovascular arrest

iii. Antiarrhythmic effect on the AV node

(I) Therefore, use with caution in combination with calcium channel blockers, beta-blockers, or digoxin.

e. Fentanyl

i. Not a depressant on myocardial contractility

ii. Causes a vagotonic decrease in heart rate and mild sympatholytic (central outflow) effects. The resulting decrease in blood pressure may be more profound in shock or pre-shock and when in combination with benzodiazepines, thiopental, or propofol.

iii. Give with caution.

f. Midazolam: do not give to the patient pre-operatively.

i. Mild to moderate systemic vasodilatation, especially when used in combination with narcotics

3. Induction

a. Standard ASA monitors and arterial line

b. Surgical team ready (gowned), with the patient sterilely prepared and draped

c. Induction with IV ketamine or etomidate and a muscle relaxant (depolarizing or non-depolarizing)

i. Keep the patient breathing spontaneously prior to induction.

d. Be prepared to treat circulatory collapse.

e. Management of hypotension

i. Vasoactive drugs

ii. Cardioactive drugs

iii. Fluid challenge

iv. Reduce inspiratory positive pressure (decrease tidal volume, or change to hand-ventilation) to facilitate cardiac filling.

v. Surgical relief of the tamponade is the definitive treatment.

f. Hemodynamic changes upon chest opening

i. Normalizes the pressure relationship between the pericardium and the heart chambers.

ii. Relieves the tamponade and its hemodynamic effects.

iii. Dramatic improvements in blood pressure and stroke volume, unless the myocardium is injured or stunned

iv. The patient can usually wean quickly from most vasoactive and cardioactive drugs.

v. Expect to see improvements in the oxygenation, acid-base status, and urine output.

Post-operative²

1. Extubation criteria

a. Clinically stable with no significant inotropic or vasoactive support

b. Neurologically intact: the patient is alert and follows simple commands.

c. Adequate pulmonary function: acceptable acid-base status and weaning parameters should be fulfilled (tidal volume, respiratory rate, vital capacity, inspiratory effort).

d. Normal body temperature

e. Intact neuromuscular function (reverse paralysis if needed)

f. Normal coagulation factor studies

2. Hypertension in the ICU after the surgical treatment of cardiac tamponade

a. Primary: exclude hypoxia, hypercarbia, acid-base derangements, and pain.

b. First few hours in the ICU: hypertension

i. Uncertain etiology

ii. Possible sympathoadrenal activation including pain, vasoconstriction, or local stress mediators.

c. Treat with

i. IV sodium nitroprusside

ii. Beta-blockers (labetalol, metoprolol, atenolol, esmolol, or propranolol): theoretical concerns of myocardial depression and bradycardia are not justified in practice.

BIBLIOGRAPHY

1. Kaplan JA, Reich DL, Lake CL, et al. *Kaplan's Cardiac Anesthesia*, 5th ed. Philadelphia: Saunders, 2006. pp 800-2.
2. Yao FSF, Fontes ML, Malhotra V. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 322-54.
3. Field JM. American Heart Association 2005 Guidelines for Advance Cardiovascular Life Support. American Heart Association, 2006.
4. Abdullah RS. Restoration of circulation after cessation of positive pressure ventilation in a case of "Lazarus Syndrome." *Anesth Analg* 2001;93:241.
5. Ben-David B, Stonebraker VC, Hershman R, et al. Survival after failed intraoperative resuscitation: a case of "Lazarus Syndrome." *Anesth Analg* 2001;92:690-2.

34. Current ACC/AHA Guidelines for Peri-Operative Cardiac Evaluation for a Non-Cardiac Surgery (Revised in 2007)^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 46-year-old male presents for gastric bypass surgery. He weighs 160 kg. He has obstructive sleep apnea and a functional capacity of less than 4 METs. He denies chest pain. He reports shortness of breath with walking, even a short distance. What further tests would you like to order? Does he need further cardiac workup? EKG? Cardiac stress test?

CLINICAL ISSUES

Risk Predictors

1. Major clinical risk predictors (active cardiac conditions)
 - a. The presence of one or more of these conditions mandates intensive management and may result in delay or cancellation of surgery unless the surgery is emergent.
 - i. Unstable coronary syndromes
 - (1) Unstable or severe angina
 - (2) Recent myocardial infarct (MI)
 - ii. Significant arrhythmias
 - iii. Severe valvular disease
 - iv. Decompensated heart failure
 2. Revised clinical risk predictors (stable cardiac conditions)
 - a. Ischemic heart disease
 - i. History of MI
 - ii. History of a positive treadmill test
 - iii. Use of nitroglycerine
 - iv. Current complaints of chest pain thought to be secondary to coronary ischemia
 - v. EKG with abnormal Q waves
 - b. Congestive heart failure
 - i. History of heart failure
 - ii. Pulmonary edema
 - iii. Paroxysmal nocturnal dyspnea
 - iv. Peripheral edema
 - v. Bilateral rales
 - vi. S3 heart sound
 - vii. Chest radiograph with pulmonary vascular redistribution
 - c. Cerebral vascular disease
 - i. History of transient ischemic attack (TIA)
 - ii. History of stroke
 - d. Preoperative insulin treatment for diabetes mellitus
 - e. Preoperative creatinine higher than 2 mg/dL (renal insufficiency)
 3. Minor predictors of clinical risk
 - a. Recognized markers for cardiovascular disease that have not been proven to independently increase peri-operative risk. The presence of multiple minor predictors might lead to a higher suspicion of coronary artery disease but is not incorporated into the recommendations for treatment.
 - i. Age greater than 70
 - ii. Abnormal EKG
 - (1) Left ventricular hypertrophy
 - (2) Left bundle branch block
 - (3) ST-T abnormalities
 - iii. Abnormal rhythm (non-sinus rhythm)
 - iv. Uncontrolled systemic hypertension

Cardiac Risk Stratification for Non-cardiac Surgical Procedures

1. Vascular (high risk): reported cardiac risk is often more than 5%.
 - a. Aortic and other major vascular surgery
 - b. Peripheral vascular surgery
 - c. Emergency surgery
 - d. Anticipated long procedures with associated fluid shifts and large estimated blood loss
2. Intermediate-risk surgery: reported cardiac risk is generally 1–5%.
 - a. Intraperitoneal and intrathoracic surgery
 - b. Carotid endarterectomy
 - c. Head and neck surgery
 - d. Orthopedic surgery
 - e. Prostate surgery
3. Low-risk surgery: reported cardiac risk is generally less than 1%.
 - a. Endoscopic procedures
 - b. Superficial procedures
 - c. Cataract surgery
 - d. Breast surgery
 - e. Ambulatory surgery

Table 34.1. Flow Chart Derived from the ACC/AHA Guidelines for the Peri-operative Management of Patients

	Low Surgical Risk	Intermediate Surgical Risk	Vascular (High) Risk
Active Cardiac Condition	4	4	4
Stable Cardiac Condition	1	2	3
Minor Cardiac Risk	1	1	1

Key:

- (1) Go to the operating room (OR).
- (2) If the functional capacity is >4 METs without symptoms, proceed to the OR. If one or more clinical risk factors exist, then proceed to surgery with heart rate control or consider non-invasive testing if it will change management.
- (3) If the functional capacity is >4 METs without symptoms, proceed to the OR. If 1 or 2 clinical risk factors exist, proceed with the planned surgery with heart rate control or consider non-invasive testing if it will change management. If 3 or more clinical risk factors are present, consider cardiac testing if it will change your management.
- (4) Optimize risk factors; evaluate and treat per the ACC/AHA guidelines.
For emergency non-cardiac surgery, the patient should proceed to the operating room and have continued peri-operative surveillance, post-operative risk stratification, and risk-factor management.

Application of Recommendations

1. Class I: benefit >>> risk. Procedure/treatment should be performed/administered.
2. Class IIa: benefit >> risk. Additional studies with focused objectives are needed. It is reasonable to perform/administer treatment.
3. Class IIb: benefit ≥ risk. Additional studies with broad objectives are needed. The procedure/treatment may be considered.
4. Class III: benefit ≤ risk. No additional studies are needed. Procedure/treatment should not be performed/administered since it is not helpful and may be harmful.

RECOMMENDATIONS**Recommendations of Pre-operative Resting 12-Lead ECG**

1. Class I:
 - a. Patients with at least one clinical risk factor who are undergoing vascular surgical procedures.
 - b. Patients with known coronary artery disease, peripheral arterial disease, or cerebrovascular disease who are undergoing intermediate-risk surgical procedures.
2. Class IIa:
 - a. Persons with no clinical risk factors who are undergoing vascular surgery procedures.
3. Class IIb:
 - a. Patients with at least one clinical risk factor that are undergoing intermediate-risk operative procedures.
4. Class III:
 - a. Not indicated in asymptomatic persons undergoing low-risk surgical procedures.

Recommendation for Non-invasive Stress Testing Before Non-cardiac Surgery

1. Class I:
 - a. Patients with active cardiac conditions in whom non-cardiac surgery is planned.
2. Class IIa:
 - a. Patients with three or more clinical risk factors and poor functional capacity (less than 4 METs) who require vascular surgery, if it will change management.
3. Class IIb:
 - a. Consider for patients with at least one to two clinical risk factors and poor functional capacity (less than 4 METs) who require intermediate-risk non-cardiac surgery if it will change management.
 - b. Consider for patients with at least one to two clinical risk factors and good functional capacity (greater than or equal to 4 METs) who are undergoing vascular surgery.
4. Class III:
 - a. Not useful for patients with no clinical risk factors undergoing intermediate-risk non-cardiac surgery.
 - b. Not useful for patients undergoing low-risk non-cardiac surgery.

Recommendations for Pre-operative Coronary Revascularization with Coronary Artery Bypass Grafting or Percutaneous Coronary Intervention

1. Class I:
 - a. Useful in patients with stable angina who have significant left main coronary artery stenosis.
 - b. Useful in patients with stable angina who have three-vessel disease.
 - c. Useful in patients with stable angina who have two-vessel disease with significant proximal left anterior descending stenosis and either an ejection fraction less than 0.5 or demonstrable ischemia on non-invasive testing.

34. Cardiac Evaluation for a Non-Cardiac Surgery

- d.** Recommended for patients with high-risk unstable angina or non-ST-segment elevation myocardial infarct.
 - e.** Recommended in patients with acute ST-elevation myocardial infarction.
2. Class IIa:
 - a.** In patients who require coronary revascularization for mitigation of cardiac symptoms and will need an elective surgery within the next 12 months. A balloon angioplasty or a bare-metal stent followed by 4–6 weeks of dual-antiplatelet therapy is probably indicated.
 - b.** In patients who have recently received a drug-eluting stent and must undergo an urgent surgical procedure that mandates the discontinuation of thienopyridine (clopidogrel/Plavix) therapy. It is reasonable to continue aspirin if at all possible and then restart the thienopyridine therapy as soon as possible.
 3. Class IIb:
 - a.** Usefulness is not well established in high-risk ischemic patients.
 - b.** Usefulness is not well established in low-risk ischemic patients with an abnormal dobutamine stress echocardiogram.
 4. Class III:
 - a.** Not recommended for patients with stable coronary artery disease before a non-cardiac surgery.
 - b.** Elective non-cardiac surgery is not recommended within 4–6 weeks of bare-metal coronary stent implantation or within 12 months of drug-eluting coronary artery stent implantation. Thienopyridine therapy (with or without aspirin) will need to be discontinued peri-operatively.
 - c.** Elective non-cardiac surgery is not recommended within 4 weeks of coronary revascularization with balloon angioplasty.

Recommendations for Beta-Blocker Medical Therapy

1. Class I:
 - a.** Continued in patients undergoing surgery who are already receiving beta-blockers to treat angina, symptomatic arrhythmias, hypertension, or other ACC/AHA Class I guideline indications.
 - b.** Given to patients undergoing vascular surgery who are at high cardiac risk owing to the finding of ischemia on pre-operative testing.
2. Class IIa:
 - a.** Probably recommended for patients undergoing vascular surgery in which the pre-operative assessment identifies coronary heart disease.
 - b.** Probably recommended for patients in which the pre-operative assessment for vascular surgery identifies high cardiac risk, as defined by more than one clinical risk factor.
 - c.** Probably recommended for patients in whom the pre-operative assessment identifies coronary artery disease or high cardiac risk, as defined by the presence of more than one clinical risk factor.

3. Class IIb:
 - a.** Recommendation is uncertain for patients who are undergoing either intermediate-risk procedures or vascular surgery, in which the pre-operative assessment showed a single clinical risk factor.
 - b.** Recommendation is uncertain for patients undergoing vascular surgery with no clinical risk factors who are not currently taking beta-blockers.
4. Class III:
 - a.** Should not be given to patients undergoing surgery who have an absolute contraindication to beta blockade.

Recommendations for Statin Therapy

1. Class I:
 - a.** Continue if currently taking statins.
2. Class IIa:
 - a.** Statin use is reasonable for patients undergoing vascular surgery with or without clinical risk factors.

Recommendations for Use of Pulmonary Artery (PA) Catheters

1. Class IIb:
 - a.** Pre-operative admission to the intensive care unit (ICU) for PA catheter monitoring is rarely required and should be restricted to a very small number of highly selected patients whose presentation is unstable and who have multiple co-morbid conditions.
 - b.** Intra-operative and post-operative use may be reasonable in patients at risk for major hemodynamic disturbances. The decision must be based on patient disease, surgical procedure (i.e., intra-operative and post-operative fluid shifts), and practice setting (experience in pulmonary artery catheter use and interpretation of results). Incorrect interpretation of the data from a PAC may cause harm.
2. Class III:
 - a.** Routine use of a PA catheter in patients at low risk of developing hemodynamic disturbances is not recommended.

Recommendation for Use of Transesophageal Echocardiography (TEE)

1. Class IIa:
 - a.** Emergency use of TEE is reasonable to determine the cause of an acute, persistent, and life-threatening hemodynamic abnormality.

Recommendation for Maintenance of Body Temperature

1. Class I:
 - a.** Maintenance of normothermia is recommended for most procedures other than during periods in which mild hypothermia is intended to provide organ protection (e.g., during high aortic cross-clamping).

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Recommendations for Peri-operative Control of Blood Glucose Concentration

1. Class IIa:
 - a. Reasonable in diabetics or patients with acute hyperglycemia who are at high risk for myocardial ischemia or who are undergoing vascular and major non-cardiac surgical procedures with planned ICU admission.
2. Class IIb:
 - a. Strict control of glucose peri-operatively is uncertain in diabetics who are undergoing non-cardiac surgical procedures without planned ICU admission.

Recommendations for Intra-operative and Post-operative Use of ST-Segment Monitoring

1. Class IIa:
 - a. Useful in patients with known coronary artery disease (CAD) or those undergoing vascular surgery.
2. Class IIb:
 - a. May be useful in patients with single or multiple risk factors for CAD who are undergoing non-cardiac surgery.

Recommendations for Surveillance for Peri-operative MI

1. Class I
 - a. Post-operative serum troponin measurement is recommended in patients with EKG changes or chest pain typical of acute coronary syndrome.



KO TREATMENT PLAN

Pre-operative

1. Assuming the patient has no diabetes or diagnosed cardiovascular risk, he does not require any further cardiac workup.

2. The patient has no clinical predictors and is having an intermediate-risk surgery.

a. Minor clinical predictors:

i. Age greater than 70

ii. Abnormal EKG

(1) Left ventricular hypertrophy

(2) Left bundle branch block

(3) ST-T abnormalities

iii. Abnormal rhythm (non-sinus)

iv. Uncontrolled systemic hypertension

b. The patient does not meet any of these criteria.

i. Obesity, less than 4 METs functional capacity, and shortness of breath with activity are not clinical predictors.

3. According to the ACC/AHA guidelines, he can proceed to the operating room.

4. No further cardiac testing is required.

a. EKG: No clinical predictors and age less than 50 years.

b. Cardiac stress test: a stress test is not indicated in this patient. He falls in the class III recommendations.

i. Not useful for patients with no clinical risk factors undergoing intermediate-risk non-cardiac surgery.

TKO: Remember that you can always order any test that you want; you just have to be able to explain why you are ordering it and be prepared to respond to the results.

BIBLIOGRAPHY

1. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 Guidelines on the perioperative cardiovascular evaluation and care for noncardiac surgery: Executive summary; *Circulation* 2007;116:000-000
2. Houser CW. ACC/AHA 2007 Guidelines on perioperative cardiovascular evaluation, and care for noncardiac surgery: Executive summary. *Anesth Analg* 2008 Mar;106(3):685-712.

35. Pacemaker/Automatic Internal Cardiac Defibrillators (AICD): Considerations for Anesthesiologists^{1,2}

Donald M. Voltz, MD

SAMPLE CASE

A 71-year-old man presents for a right hemicolectomy after a suspicious polyp was found during a routine colonoscopy. He has a past medical history of hypertension and sick-sinus syndrome for which he had a pacemaker placed 8 years ago. He is currently active, has no known drug allergies, and has been NPO for 16 hours except for a bowel prep. He last saw his cardiologist 1 year ago and was without any limitations according to the patient. What are your concerns? Does this patient need to be seen by his cardiologist prior to his surgery? What do you need to know about his pacemaker? Will the pacemaker affect your anesthetic?

CLINICAL ISSUES

Incidence of Pacemakers and Automatic Internal Cardiac Defibrillators (AICD)

1. There are roughly 200,000 pacemakers and 60,000 AICDs implanted each year.
2. With recent recalls on some of the leads and/or devices, the number of devices needing to be replaced also adds to these figures.

Indications for Pacemaker/AICD Placement

1. Pacemaker
 - a. Sick-sinus syndrome
 - b. 3rd degree atrio-ventricular (AV) block
 - c. Symptomatic first- or second-degree heart block
 - d. Non-ischemic, exercise-induced heart block
 - e. Bradycardia associated with syncope or near syncope
 - f. Bradycardia associated with ventricular arrhythmias
 - g. Dilated cardiomyopathies
 - h. Hypertrophic cardiomyopathies
2. AICDs
 - a. Patients at high risk for fatal ventricular arrhythmias:
 - i. Ventricular tachycardia
 - ii. Ventricular fibrillation
 - b. Patients at high risk for sudden cardiac death (SCD)
 - c. Prior myocardial infarct with ventricular tachycardia or ventricular fibrillation
 - d. Patients with moderate to severe cardiomyopathies

Pacemaker Nomenclature

The North American Society for Pacing and Electrophysiology (NASPE) along with the British Pacing and Electrophysiology Group (BPEG) devised a standardized nomenclature for all implantable pacemakers and defibrillators. This nomenclature consists of a five letter code to describe the functionality of these devices. This standardized code was last updated in 2002.

Position 1 - Chamber being paced

- O - No chamber paced
- A - Atria
- V - Ventricle
- D - Dual - atria and ventricle

Position 2 - Chamber being sensed

- O - No chamber sensed
- A - Atria
- V - Ventricle
- D - Dual - atria and ventricle

Position 3 - Response of device to a sensed event

- O - No response to sensed event
- T - Trigger device when event sensed
- I - Inhibit device when event sensed
- D - Dual - triggered and inhibited

Position 4 - Programmability and rate modulation

- O - No programmability
- R - Rate modulation activated

Position 5 - Multi-site pacing

- O - No chamber paced
- A - Atria
- V - Ventricle
- D - Dual - atria and ventricle

How Magnets Affect Pacemakers and AICDs

1. Most of the pacemakers that have been developed and are clinically in use contain an electronic component (Reed switch) which is affected by the application of a magnetic field to the device.
2. When the Reed switch comes in contact with a magnetic field, it can either close a circuit or disrupt electrical flow.

3. In a wide number of pacemakers, placement of a magnet over the generator will result in the pacemaker being converted to a continuous asynchronous mode.

- a.** This is not the case for all pacemakers.
- b.** Those in anesthesiology sometimes get the sense that the addition of a Reed switch was placed for anesthesiologists to quickly set a pacemaker to an asynchronous mode and prevent the interference from electrocautery.
- c.** Magnets were not initially intended to change pacemaker modes in the operating room, but instead were incorporated to test certain functions of these devices such as battery life.
- d.** Not all pacemakers are switched to an asynchronous mode with the application of a magnet and some pacemakers may not revert back to their baseline settings once the magnet is removed.

4. Some pacemakers will be completely deactivated with the placement of a magnet.

5. In order to be clear about what will happen with the placement of a magnet, one needs to know the brand and model of the implanted device.

6. This can sometimes be uncovered with a chest X-ray; however, most need to be interrogated by specialized computer devices that are most commonly found in the cardiology department.

7. AICDs and pacemakers respond differently to the placement of a magnet.

- a.** The algorithms for detection of fatal ventricular arrhythmias lead these devices to respond to electromagnetic interference differently from standard pacemakers.
- b.** In light of this, AICDs need to be deactivated prior to surgical procedures where electrocautery or other electromagnetic interference may occur.
- c.** Deactivating the device renders these patients without protection from ventricular arrhythmias so one will need to be prepared to apply external cardiac defibrillation should a life-threatening arrhythmia develop when the patient is in the operating room.
- d.** Many AICDs also contain protection against bradycardia, thereby functioning as pacemakers in addition to their anti-arrhythmic functionality.
- e.** Inactivation or disabling an implantable device:
 - i.** Not straightforward
 - ii.** Demands vigilance and investigation prior to proceeding to the operating room.
- f.** Application of a magnet to an AICD will most commonly disable the defibrillation option. This is not an absolute, however, since different models exist with a wide variety of default configurations in the presence of a magnet. Another important point to realize is that combined pacemakers and AICDs do not uniformly respond to the application of a magnet. Many of the current pacemakers revert to an asynchronous state when a magnet is placed over the device. Combined AICD and pacemakers do not all revert to an asynchronous pacing mode in the presence of a magnetic field.

TKO: Due to inconsistencies between the many pacemakers and AICD devices on the market, it is advisable to interrogate each device to know what a patient's underlying rhythm is, what the device defaults to in the presence of a magnet, and the state of the device (battery charge, lead integrity, overall device health). Following a case, the device should be re-interrogated to determine whether any changes are required to restore it to its pre-operative status.

PRE-OPERATIVE EVALUATION OF PACEMAKERS AND AICDS

1. Retrospective investigation into the failure rates of implantable pacemakers and AICDs showed an outright failure in 1.4 pacemakers and 36.4 AICDs per 1,000 implants.²

2. In light of this failure rate, the large variation in how the devices respond to electromagnetic interference, and often unclear reason for device placement, it is important to investigate these devices prior to elective and semi-emergent surgical procedures.

3. Depending upon how long the device has been in place as well as the number and types of leads that have been placed, a pre-operative chest X-ray might help to determine which vessels contain the pacemaker/AICD leads.

4. If a central line is planned for a given procedure, understanding the path of the leads might influence which vessel is cannulated, since lead disruption or central line entanglement has been reported.

5. All implantable devices should be interrogated by someone who is competent to interrogate these devices and make peri-operative setting changes for the safety of the patient.

6. A copy of the interrogation and settings should be kept with the patient's chart so any changes can be undone after the procedure is completed.

7. The interrogation will provide valuable information about the device.

- a.** Remaining battery life
- b.** Integrity of the leads
- c.** Patient's underlying cardiac rhythm
- d.** Current settings and recent discharges of the device (AICDs)

8. Understanding the patient's underlying rhythm is important.

- a.** Determines the mode and rate at which to set the backup pacing.
- b.** Gives an understanding of what the device will default to when a magnet is placed over the generator.

9. Some pacemakers have modes that respond to minute ventilation in order to increase the pacing rate with exercise.

- a.** During a surgical procedure, this minute ventilation rate responsiveness and other programmed rate responsive functions should be disabled.

10. Any anti-tachycardic functionality should be disabled since electrocautery interference with the device can lead to inappropriate discharge.

PERI-OPERATIVE PACEMAKER SETTINGS

1. Intrinsic pacemaker functions are often disrupted by the electromagnetic interference that occurs during surgery.
 - a. Pacemakers need to be reprogrammed prior to the procedure
 - b. The patient's baseline settings should be restored following the conclusion of the procedure.
2. Reprogramming a pacemaker to an asynchronous mode at a rate greater than the patient's underlying basal rate ensures that over- and under- sensing by the device does not occur.
 - a. An asynchronous mode is not completely protective and can result in a malignant arrhythmia.
 - i. Most patients who undergo pacemaker placement have co-existing disease of the myocardium.
 - ii. This places them at a higher risk for arrhythmias.
3. In a device that contains anti-tachycardia protection (AICDs), this feature should be turned off.
 - a. One needs to be prepared for external cardiac defibrillation in the event of a malignant ventricular arrhythmia occurring while the device is inactive.
 - b. External defibrillation pads or external paddles should be available.

PERI-OPERATIVE PACEMAKER FAILURE

1. Pacemakers are mechanical devices with the potential to fail at any time. Pacemaker failure can result from
 - a. Inability to capture
 - b. Lead disruption
 - c. Generator failure
 - i. Peri-operative lead or generator failure occurs at a much lower level than failure to capture.
 - ii. Proper pre-operative interrogation should confirm the integrity of the device and leads.
2. Pacemaker wires are placed so that contact between the wire and the myocardium occurs.
 - a. Over time, the pacemaker lead often becomes densely adherent to the myocardium due to a fibrotic reaction between the leads and the myocardium.
3. Certain situations that occur in the operating room can inhibit the conduction of an electrical impulse from the pacemaker generator to the myocardium and therefore impede conduction and capture of the signal resulting in a failure to capture.
 - a. Ischemia
 - b. Acidosis
 - c. Anti-arrhythmic medications
 - d. Electrolyte disturbances
4. In the event of a non-capturing pacemaker in a patient who is dependent on the device for cardiac function, emergency external pacing might be required.

POST-OPERATIVE PACEMAKER MANAGEMENT

1. Any changes that were made to a pacemaker or AICD prior to surgery should be restored to the patient's baseline settings to ensure the best outcome for the patient.
2. Many of these devices are adjusted on an individual basis to ensure device longevity as well as to optimize a patient's cardiac function.
3. Leaving them in a peri-operative default setting can
 - a. Greatly impact the patient's performance.
 - b. Increase the drain on the battery leading to a more rapid deterioration of the device and necessitate a replacement.
4. If an AICD has been deactivated, this should be restored as quickly as possible following the procedure.
 - a. Until AICD function has been restored, an external pacer/defibrillator should be immediately available.
5. Most pacemaker manufacturers recommend a complete re-interrogation of the devices following surgery to ensure that
 - a. Electrocautery did not inflict any damage to the device
 - b. Electrocautery did not prematurely drain the battery.


KO TREATMENT PLAN
Pre-operative

1. This patient has had a pacemaker for a number of years and his underlying rate, rhythm, and dependence on the device are unclear.
2. The patient may or may not know if he also has AICD functionality of the device.
3. If there is no record of recent interrogation, the device should be studied prior to proceeding with surgery.
4. Assuming the device and leads are intact and adequate battery life remains, we can safely proceed with the procedure after the device is reprogrammed.
5. If on interrogation it was discovered that the pacemaker battery did not have sufficient charge, the leads had an unsafe level of resistance, or the device revealed some other problem, the case should be postponed until a new device and/or leads can be implanted.
6. Given that this patient has an underlying bradycardic rhythm of 60 beats per minute, the device should be reprogrammed to an asynchronous mode with a rate of 70 beats per minute.
7. In addition, if the device had anti-tachycardic features, these should be disabled.

Intra-operative

1. No special monitoring or techniques need to be employed in the operating room.
2. Be careful about the placement of the electrocautery grounding pad so that it does not cause damage to the pacemaker generator.

3. Be vigilant for any failure in pacemaker capture that could occur.
4. Correct any acid-base or electrolyte disturbances should they occur, as these can affect the pacemaker response.
5. If this case was a true emergency:
 - a. Place the electrocautery grounding pad away from the pacemaker.
 - b. Place external pacer pads on the patient and have a defibrillator/pacer connected in the monitoring mode.
 - c. Place a magnet on the pacemaker/AICD once external pads are in place and a continuous EKG tracing is present. Observe what happens to the pacemaker rhythm with the application of the magnet.
 - d. Obtain a stat chest X-ray intra-operatively; some devices have a series of codes that can be determined with a chest X-ray.
 - e. Consult cardiology to evaluate the patient intra-operatively. If this is not possible, the device needs to be interrogated immediately following the procedure.
 - i. Until the device is re-interrogated, you can not be certain that the pre-operative pacemaker settings have been restored or that the AICD is indeed functional.

- ii. Continuation of external pacing/defibrillation pads and monitoring should continue until the device is interrogated and reset by the cardiology team.

Post-operative

1. Following the conclusion of the procedure, re-interrogate the device to reset it to the pre-operative settings.
2. If an AICD was deactivated, it should be reactivated.
3. Finally, document the device integrity and battery life. Forward this information to the physician who is managing the device.

BIBLIOGRAPHY

1. Hillel, Z. and D. Thys. *Electrocardiography. Miller's Anesthesia*, R. Miller (ed). Philadelphia: Elsevier: 2005, pp 1416-8.
2. William H. Maisel, Megan Moynahan, Bram D. Zuckerman, Thomas P. Gross, Oscar H. Tovar, Donna-Bea Tillman, Daniel B. Schultz. Pacemaker and ICD generator malfunctions: Analysis of Food and Drug Administration annual reports. *JAMA* 2006; 295:1901-6. [link]

36. Cardiac Valvular Abnormalities^{1,2,3}

Donald M. Voltz, MD

SAMPLE CASE

A 71-year-old female was scheduled to undergo an elective total hip arthroplasty. Her activity level is minimal due to arthritic changes of her hips. She has a history of hypertension and hypothyroidism, both under good control with medication. Prior to her surgery, a systolic murmur was appreciated prompting a full cardiac workup. A transthoracic echocardiogram revealed aortic stenosis with a peak gradient across the aortic valve of 48 mm Hg and a left ventricular ejection fraction of 35% with concentric hypertrophy. No evidence of stress-induced ischemia was appreciated on a dobutamine stress test.

CLINICAL ISSUES

Importance of Understanding Cardiac Valvular Lesions

1. Myocardial optimization depends on a balance between oxygen utilization and its supply.

- a. Optimization of myocardial performance is greatly impacted both by imbalance in the oxygen balance as well as the loading conditions on the ventricles.
- b. Due to a high incidence and severity of complications that occur in patients with hemodynamically significant lesions it is important to understand how to optimize these patients.
2. Important hemodynamic variables in patients with valvular disease include
 - a. Rhythm
 - b. Heart rate (HR)
 - c. Preload (PL)
 - d. Contractility
 - e. Afterload (AL)
 - f. Systemic vascular resistance (SVR)
 - i. Control of these factors and understanding how they are altered in valvular pathologic situations is important to safely get these patients through surgery and optimize them both pre-operatively and in the peri-operative period.

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3. Important variables

- a. Systemic blood pressure is directly related to the product of cardiac output (CO) and SVR.
- b. CO is the product of stroke volume (SV) (ventricular end diastolic volume - ventricular end systolic volume) and HR.
- c. Ventricular end diastolic volume is impacted by preload conditions and the compliance of the ventricle during diastole.
- d. Ventricular end systolic volume is impacted by afterload conditions and the contractile state of the ventricle.
 - i. All of these variables are impacted by hemodynamically significant valvular disease and all play a role in the myocardial oxygen balance at both a baseline state and during surgical and anesthetic stresses.
 - ii. Understanding the impact of these factors on myocardial performance and oxygen balance is different for each of the major valvular disorders.

4. Principles of normal and pathologic flow states across a valve orifice

- a. Flow across any orifice depends on
 - i. Cross-sectional valve area
 - ii. The square root of the pressure gradient across the valve
 - iii. The amount of time a given volume of blood has to transit the valve orifice
 - (1) An increase in any of these conditions translates into greater flow across the valve orifice while a decrease in any of these leads to a decrease in flow.
 - (2) Since flow across any of the cardiac valves impacts overall cardiac output and the perfusion pressure of the body, optimizing flow across diseased valves while limiting oxygen debt situations is important for improving patient outcomes.
- b. Flow conditions are responsive to loading conditions in some pathologic states (i.e., regurgitant lesions) while in others they are fixed (i.e., stenotic lesions).
 - i. Transvalvular flow depends on the point of the cardiac cycle and the type of valvular pathology.
 - ii. Valvular disease presents two main physiologic mechanisms that result in the pathology and also allow for compensation by the cardiovascular system.
 - (1) In stenotic valvular lesions, there is a pressure overload state on the downstream cardiac chamber (i.e., the left ventricle in aortic stenosis).
 - (a) For stenotic lesions, the ratio of transvalvular flow/duration of the cardiac cycle where flow occurs (i.e., systole for aortic stenosis) is important for cardiac function.
 - (b) In the case of aortic stenosis, when the flow increases or the duration of the systole shortens, this ratio increases leading to more pressure strain on the left ventricle.
 - (c) Minimizing any increases in flow or increasing the systolic period will help to reduce the pressure stress on the heart.

(2) In regurgitant lesions, there is a volume overload state placed on the downstream chamber (i.e., the left atrium in mitral regurgitation).

(a) Management of all cardiac lesions depends on minimizing either the pressure or volume overload state while maintaining or enhancing systemic cardiac output.

5. Understanding how changes in heart rate can affect the duration of systole and diastole is also important when it comes to valvular lesions and their optimization.

- a. A rapid heart rate leads to a proportionately greater decrease in filling time during diastole than systole.
- b. Slowing the heart increases both the diastolic filling time as well as the duration of systole.
- c. This is important to understand for both stenotic and regurgitant lesions.

6. Systemic and pulmonic vascular resistance (afterload on the left and right ventricles, respectively) is also important to understand.

- a. Increasing the afterload can lead to a greater degree of regurgitation while a significant drop in afterload can be poorly tolerated in stenotic lesions.
- b. When afterload is reduced, the resulting blood pressure drop triggers the cardiac system to increase its output, either by an increase in heart rate, increase in stroke volume, or often an increase in both.
- c. For regurgitant lesions, a decreased afterload makes forward cardiac flow easier, leading to a physiologic improvement and less regurgitation.
- d. This is not the case for stenotic lesions, however.
 - i. During a reduction in afterload, the cardiac response is an elevation in heart rate leading to more blood that must traverse a stenotic valve.
 - ii. In a fixed stenosis, in order to increase blood flow you need to increase the velocity of the blood, which occurs by increasing the pressure behind the stenosis.
 - iii. As the stenosis becomes worse, a patient's ability to increase his or her cardiac output during a reduction in afterload becomes limited; patients often will not be able to maintain their blood pressure.
 - iv. With a decrease in blood pressure, there is a reduction in cardiac perfusion (that occurs during diastole when the heart is relaxing) leading to myocardial ischemia that further limits cardiac output.
 - v. In patients with significant aortic stenosis, a drop in afterload can result in cardiac arrest.
 - vi. Slow induction of anesthesia is important to limit the amount of afterload reduction and ensure that cardiac wall stress and perfusion are optimized.

Pathophysiology of Aortic Stenosis

1. The main etiology for aortic stenosis is senile fibrocalcific degeneration leading to aortic sclerosis and stenosis.
2. The main etiology used to be rheumatic heart disease, although this has been decreasing in frequency.

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3. In a subset of patients, a congenital bicuspid aortic valve leads to an acceleration of fibrocalcific deposits and these patients tend to present with significant aortic stenosis at an earlier age.
4. In about 50% of the patients who present with aortic stenosis, significant coronary artery disease is also present, leading to a combined aortic valve replacement as well as a bypass operation.

Symptoms of Aortic Stenosis

1. Patients who develop aortic stenosis are often asymptomatic for the majority of the disease.
 - a. As the degree of stenosis impacts the myocardial reserve, symptoms begin to develop.
 - b. The presence and severity of symptoms are related to the prognosis of this disease.
 - c. Ideally, intervention is best during the early stages to avoid irreversible myocardial damage and to allow the myocardium to remodel itself.
 - d. Patients with worsening aortic stenosis often present with SAD: syncope, angina, and dyspnea.
2. Syncope
 - a. In the presence of a normal cardiovascular system, as systemic oxygen requirements increase during exercise or other physiologic stress, both the heart rate and stroke volume increase to produce a higher cardiac output, thereby increasing the delivery of oxygen and removal of waste generated by the tissues.
 - b. In the presence of aortic stenosis, a fixed defect limiting the amount of blood that can be ejected from the heart, there is a limit on increasing cardiac output to meet the peripheral oxygen demands.
 - c. When tissues increase their metabolism, such as during times of exertion or other physiologic stresses, vasodilatation of the capillary beds occurs to increase tissue perfusion and prevent an oxygen debt.
 - i. In patients who have severe aortic stenosis, higher oxygen requirements by tissues during times of exertion lead to vasodilatation; however, a concomitant increase in cardiac output to overcome this drop in SVR cannot occur, resulting in hypotension and a decreased perfusion of the brain.
 - ii. If the drop in cerebral perfusion is severe enough, syncope can result.
 - iii. This problem occurs when the aortic valve becomes severely narrowed limiting an increase in cardiac output.
 - d. When patients with aortic stenosis present with syncope, their 3 year mortality without aortic valve replacement approaches 50%.²
3. Angina
 - a. Aortic stenosis leads to progressive myocardial hypertrophy in an effort to generate higher left ventricular pressures and maintain cardiac output.
 - b. Both myocardial oxygen demand and supply are altered in the presence of aortic stenosis.
 - c. Myocardial hypertrophy necessitates a higher oxygen requirement while aortic stenosis leads to a decrease in aortic root pressure, the driving force behind myocardial perfusion pressure during diastole.
 - d. When the oxygen demand is not met by the supply, an oxygen debt results in the development of angina.
 - e. Patients with critical aortic stenosis are not able to increase their cardiac output to any great extent without developing ischemia and angina.
 - f. This becomes a vicious spiral since ischemia leads to myocardial dysfunction which further impacts left ventricular function, a drop in cardiac output, and further drops in myocardial perfusion.
 - g. In patients who develop angina in the presence of aortic stenosis, their 5 year mortality approaches 50%.²
4. Dyspnea
 - a. Dyspnea in the presence of aortic stenosis arises from the increased left ventricular pressures required to pump blood across the stenotic aortic valve.
 - b. As the valve area decreases, the left ventricular pressure must increase if one is to maintain a normal cardiac output.
 - c. An increase in pressure develops when one attempts to pump the same volume of blood across a narrower orifice.
 - d. If the left ventricular pressure becomes too great, the pressure is transmitted across the mitral valve and into the pulmonary system.
 - e. In the initial phases of aortic stenosis, the myocardium hypertrophies in an attempt to generate these higher pressures; but with increasing degrees of stenosis, the myocardium is not able to keep up, leading to progressive systolic and diastolic dysfunction culminating in congestive heart failure.
 - f. When patients progress to congestive heart failure, their prognosis becomes poor without an aortic valve replacement.
 - i. Two year mortality as high as 50% has been reported in patients who develop congestive heart failure in the presence of aortic stenosis.²
5. Summary
 - a. Patients who present with syncope, angina, or dyspnea and have severe or critical aortic stenosis have a much increased risk of dying without surgical aortic valve replacement.
 - b. In order to preserve cardiac function, medical management attempts to delay the progression of this disease or intervene surgically before irreversible myocardial damage has resulted.
 - c. With replacement of the diseased aortic valve, a fair amount of myocardial remodeling can occur, allowing for an increased longevity in these patients.

Assessment of the Severity of Aortic Stenosis

1. The normal aortic valve area is 1.6–2.5 cm².
 - a. As stenosis progresses, this valve area decreases with a concomitant increase in left ventricular pressures to move the same amount of blood across the valve.

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- b.** In a normal situation, the pressure gradient between the left ventricle and the aorta is only a few mm Hg; but as the stenosis progresses, this pressure gradient can become quite elevated.
 - c.** The degree of aortic stenosis as measured by the pressure gradient across the valve correlates to the degree of aortic valve area.
- 2.** Mild aortic stenosis
 - a.** The valve area falls to 1.5–1.0 cm² resulting in a minimal elevation in peak gradient to less than 20 mm Hg.
- 3.** Moderate aortic stenosis
 - a.** Valve area between 1.0–0.8 cm² and a peak gradient of greater than 50 mm Hg.
- 4.** Severe aortic stenosis
 - a.** Valve areas below 0.8 cm² and peak gradients greater than 50 mm Hg and can be as high as 100 mmHg.³

Management of Cardiac Valvular Disease

- 1.** In order to safely manage patients who present with cardiac valvular disease, you must understand the following five factors and how changes in these physiologic parameters can improve or impede the clinical situation of patients with valve disease.
 - a.** Cardiac rhythm
 - b.** Heart rate
 - c.** Preload
 - d.** Contractility
 - e.** Afterload
- 2.** Optimal management of patients who present with any cardiac valvular lesion will depend on these parameters and are addressed below for each pathologic condition.

KNOCK OUT MANAGEMENT STRATEGIES

Aortic Stenosis (AS)

- 1.** Rhythm
 - a.** Patients with aortic stenosis often develop both systolic and diastolic dysfunction as a result of this disease.
 - b.** Myocardial hypertrophy leads to a decrease in the compliance of the left ventricle (LV) and thereby a decrease in the passive filling of the LV during diastole.
 - c.** In a normal situation, atrial systole provides 20–30% of the LV filling volume.
 - d.** Due to a decreased compliance, the importance of the left atrial systole is exaggerated in patients with aortic stenosis if they are to maintain their cardiac output.
 - e.** Maintenance of a normal sinus rhythm is important; atrial fibrillation is poorly tolerated in this patient population.
 - f.** If these patients develop an unstable or rapid arrhythmia, immediate cardioversion should be performed.
- 2.** Heart rate
 - a.** Patients with severe aortic stenosis have a narrow window in regard to heart rate.
- b.** In all situations, maintenance of their baseline, resting heart rate should be maintained.
- c.** As mentioned above, these patients have a poorly compliant ventricle and therefore require a longer time and higher left atrial pressures to adequately fill the LV.
- d.** This filling comes during diastole, the period of the cardiac cycle most affected by increases in heart rate.
- e.** A rapid heart rate increases oxygen requirements while at the same time limiting supply, placing these patients at an increased risk for ischemia leading to further myocardial dysfunction.
- f.** Based on this information, one would assume bradycardia would be best for patients with aortic stenosis; however, due to the fixed lesion at the aortic valve limiting the amount of blood ejected during each cardiac cycle (i.e., a fixed stroke volume), a drop in heart rate can significantly limit cardiac output leading to hypotension and peripheral perfusion problems.
- g.** Maintenance of heart rate at the patient's baseline level, usually between 70–90 beats/minute allows for an adequate cardiac output while at the same time maintaining a balanced oxygen delivery and consumption.

- 3.** Preload
 - a.** With a decrease in LV compliance and therefore a higher requirement on left-sided filling pressures to ensure an adequate cardiac output, adequate preload is important in these patients.
 - b.** A loss of intravascular volume resulting from dehydration or blood loss is poorly tolerated since this leads to under filling of the LV and a resulting decrease in cardiac output.
 - c.** Overzealous fluid administration can also lead to complications since the left atrial pressures and pulmonary capillary pressures are elevated to overcome the poorly compliant left ventricle and allow for adequate filling.
 - d.** Too much intravascular volume can tip the balance and place these patients in pulmonary edema.
 - e.** Careful monitoring and management of preload status is important in the optimization of patients with aortic stenosis.
- 4.** Contractility
 - a.** As with all valvular lesions, the ventricular and atrial chambers become modified to deal with the abnormal fluid and pressure dynamics.
 - b.** As a result of ventricular hypertrophy to overcome the narrowing of the aortic valve, ventricular performance decreases.
 - c.** Individual myocytes are forced to work harder and their contractile force for unit of energy burned is lessened resulting in an overall decrease in contractility.
 - d.** Further decreases in contractility that result from ischemia or other myocardial insults must be avoided to maintain adequate cardiac output and systemic pressures.
- 5.** Afterload
 - a.** Like many of the other cardiac parameters, maintenance of systemic vascular resistance and afterload is important so that coronary perfusion is not impacted.

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- b.** As mentioned in the case above, peripheral vasodilatation is normally offset by a compensatory increase in cardiac output.
- c.** With aortic stenosis and a relatively fixed stroke volume, a drop in SVR leads to a precipitous fall in blood pressure and a significant drop in coronary perfusion pressure.
- d.** It is important to maintain the afterload at baseline levels since hypotension leads to a decrease in coronary perfusion, further dropping cardiac output, perpetuating the problem.

Aortic Insufficiency (AI)

1. Rhythm

- a.** Patients with aortic insufficiency maintain the best forward cardiac output with a slightly tachycardic state.
- b.** Uncontrolled tachycardia leads to a decrease in performance and an increase in oxygen requirements.
- c.** Typically patients with aortic insufficiency are best optimized with heart rates between 90–110 beats/minute.

2. Heart rate

- a.** The hemodynamic goals in aortic insufficiency are to maintain an adequate forward cardiac output.
- b.** In any regurgitant valvular lesion, there is a balance between forward blood flow and the regurgitant flow that results through the incompetent valve.
- c.** It is only the forward flow that allows for end-organ perfusion; the regurgitant flow is lost at the expense of extra-myocardial work.
- d.** In AI, regurgitation occurs during diastole, so limiting this period of the cardiac cycle will limit the amount of backward flow.
- e.** A slightly tachycardic rate allows for both an overall increase in cardiac output as well as a decrease in the amount of regurgitation.
 - i.** This comes at the expense of an increased oxygen requirement that comes with an increasing heart rate.
- f.** Bradycardia results in a lower oxygen requirement for each beat of the heart but allows for a longer diastolic time and therefore a reduction in forward cardiac output.
 - i.** This inefficient cardiac output leads to more myocardial work without a resulting benefit in systemic perfusion.
- g.** Patients tolerate a mild increase in heart rate to promote forward flow and to compensate for the regurgitant flow.
- h.** Aortic insufficiency also leads to a precipitous drop in aortic root pressure during diastole.
 - i.** This drop in pressure results in a drop in coronary perfusion pressure and impacts coronary blood flow.
 - ii.** An increased heart rate and limitation of diastole corrects some of this problem.

3. Preload

- a.** The left ventricle in patients with aortic insufficiency tends to dilate to make room for the extra volumes that occur in diastole.
- b.** These patients have normal filling during diastole from the left atrium; however, they also have a fraction of the

forward cardiac output entering the LV through an incompetent aortic valve.

- c.** This results in a situation in which the LV has an exaggerated preload for the next cardiac cycle.

- d.** Remodeling of the ventricle to accommodate this extra volume is accomplished by dilation.

- e.** In the event of significant intra-vascular depletion, the LV can become under filled and have a detrimental effect on cardiac output.

- f.** Patients with AI must not be overzealously fluid resuscitated leading to acute decompensation of the LV and concomitant pulmonary edema.

4. Contractility

- a.** With dilatation of the left ventricle to accommodate the extra volume present as a result of aortic regurgitation, the myocytes become stretched.

- b.** With continual dilation, contractility decreases.

- c.** Uncontrolled AI can result in myocardial failure.

5. Afterload

- a.** With an increase in systemic pressure and elevations in afterload, the forward cardiac output decreases leading to a higher fraction of regurgitant blood.

- b.** A slight, controlled drop in systemic vascular resistance promotes forward cardiac flow and a decrease in regurgitation.

- c.** Uncontrolled drops in SVR or exaggerated increases in blood pressure/afterload can lead to cardiovascular collapse and therefore need to be eliminated.

Mitral Stenosis (MS)

1. Rhythm

- a.** Maintaining these patients in a sinus rhythm at their baseline heart rate is ideal; however, due to a chronic increase in left atrial pressures and a resulting left atrial dilation to accommodate these pressures, many patients present with atrial fibrillation.

- b.** Atrial fibrillation leads to a decrease in late diastolic filling and can reduce end diastolic volumes by up to 30%.

- i.** This is overcome by the increased left atrial pressures and higher than normal early and late diastolic fillings that result from these increased pressures.

- c.** Given that many of these patients present in atrial fibrillation and most are unlikely to be successfully converted to a normal sinus rhythm due to atrial dilation, control of their heart rate between 70–90 beats/minute is important.

- i.** Rapid ventricular response is not well tolerated in these patients since an increase in ventricular rate impacts diastolic filling times and a decrease in cardiac output.

2. Heart rate

- a.** As with aortic stenosis, ventricular filling occurs during diastole.

- b.** Given this situation, maintaining a baseline heart rate between 70–90 beats/minute provides adequate diastolic filling time while preserving a heart rate-dependent cardiac output.

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c. Both an uncontrolled increase and a precipitous decrease in heart rate lead to an uncompensated situation and an overall reduction in cardiac output.

i. If the heart rate falls too low, the fixed nature of aortic stenosis limits the amount of LV filling and therefore results in a fall in cardiac output.

ii. Increasing the heart rate normally would lead to an increase in cardiac output; however, in patients with flow limiting mitral stenosis, every elevation in heart rate decreases filling of the LV due to a shortening of the diastolic time as well as the flow limitation imposed by the stenosis.

3. Preload

a. A fixed, reduced mitral valve area in patients with flow limiting mitral stenosis necessitates maintenance of adequate preload.

b. Ventricular filling both during early and late diastole is limited by the reduction in cross-sectional area of the stenotic mitral valve.

c. A drop in preload results in a drop in left atrial pressure resulting in a decrease in LV filling.

d. With a fall in LV end diastolic volume, a concomitant fall in cardiac output occurs.

e. Patients with mitral stenosis compensate by maintaining a greatly elevated left atrial pressure that ultimately leads to atrial dilatation, and a vast majority of patients present with atrial fibrillation.

4. Contractility

a. In the early stages of mitral stenosis, the left ventricle contractility is not impacted.

b. With progression of the disease or the presence of multiple valvular lesions such as combined mitral and aortic stenosis that occurs in rheumatic heart disease, the left ventricular function may be impacted.

5. Afterload

a. Mitral stenosis results in a fixed lesion and therefore a fixed stroke volume due to limitations on getting blood into the left ventricle during diastole.

b. In a normal situation, a fall in afterload would be compensated by an increase in cardiac output.

c. In patients with mitral stenosis, they are not able to increase their cardiac output by a modification of stroke volume.

d. In addition, an increase in heart rate, in a normal situation, would lead to an increased cardiac output, but in these patients, it further limits diastolic filling and is detrimental to LV filling.

e. Given this, patients with mitral stenosis do not respond well to precipitous drops in afterload requiring an increase in their cardiac output.

Mitral Insufficiency (MI)

1. Rhythm

a. Maintenance of a mild degree of tachycardia is best to promote adequate LV filling while limiting the amount of time in diastole.

b. With significant regurgitation, the left atrium can become enlarged and dilated to accommodate the regurgitant volumes.

i. Patients are more likely to develop atrial fibrillation.

ii. Uncontrolled tachycardia that can result from a rapid ventricular response should be avoided.

2. Heart rate

a. In a manner similar to aortic insufficiency, patients with mitral insufficiency are attempting to optimize the amount of forward cardiac output.

b. A slight increase in heart rate leads to a decrease in the diastolic time and therefore limits the amount of regurgitation that can occur.

c. Excessive elevations in heart rate are not good since these ultimately impact coronary oxygen supply and demand.

d. A mild tachycardia between 90–110 beats/minute is usually optimal for these patients.

e. Profound bradycardia is not well tolerated since this can lead to acute left ventricular fluid overload when a significant amount of the cardiac output flows through the incompetent mitral valve.

3. Preload

a. Patients with mitral insufficiency often maintain adequate levels of preload and the left ventricle is presented with higher than normal volumes during diastole as a result of the mitral insufficiency.

4. Contractility

a. Many patients who present with mitral insufficiency also have ventricular dysfunction.

b. A poorly functioning ventricle leads to hypertrophy and dilation of the mitral annulus resulting in chronic mitral insufficiency.

c. In these patients, the ventricular function is depressed and further myocardial depressants can lead to decompensation.

d. It is important to understand the etiology of valvular dysfunction and how the inciting disease processes can be worsened leading to cardiac decompensation.

5. Afterload

a. The balance between afterload and left atrial pressures determines the fraction of regurgitant flow and forward cardiac output.

b. A slight reduction in systemic vascular resistance leads to an increase in forward cardiac output and is well tolerated by patients with mitral insufficiency.

c. Precipitous increases in afterload decrease forward cardiac output and cause an increase in left atrial and pulmonary capillary pressure.

d. If this increase is too great, pulmonary edema can result.

e. In patients with severely depressed left ventricular function leading to a significant amount of mitral regurgitation, surgical repair of the valve can lead to left ventricular decompensation.

f. This occurs because in the diseased state, any increase in afterload does not present as a further strain on the left ventricle.

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- g. The lower-pressured left atrium would serve as a “pop-off” mechanism thereby limiting the strain on the LV.
- h. Once the mitral valve is replaced or repaired, restoring competence, any increase in afterload strain is directly transferred to the left ventricle.
- i. If the increase in afterload is too great, left ventricular failure can develop in the peri-operative period.



KO TREATMENT PLAN

Pre-operative

1. The patient presented above is at an increased risk of developing complications in the peri-operative period.
2. Although her pre-operative stress echocardiogram did not reveal any areas of ischemia, she has moderate to severe aortic stenosis with a decrease in her left ventricular function (LVEF 35%).
3. Since this is an elective case, it is prudent to evaluate this patient for an aortic valve replacement prior to repairing her hip.
4. If this patient presented for an emergency operation, the luxury of repairing her valve would not be present.
 - a. In an emergent situation, pre-operative optimization would be required.
 - i. In the setting of an emergency case, a pre-induction arterial line is required to be able to closely monitor and respond to any fluctuations in blood pressure.
 - ii. Ideally, maintaining this patient at her baseline heart rate and blood pressure is the best.
 - iii. Avoidance of profound drops in blood pressure is important since this drop greatly effects myocardial perfusion pressure and can result in ischemia followed by cardiac arrest.
 - (1) Due to the limitation in aortic valve area, CPR is often not effective in patients with severe AS and therefore any drops in blood pressure should be managed aggressively.
 - iv. In choosing an anesthetic technique, medications that maintain blood pressure without significantly impacting heart rate should be chosen.
 - (1) Many people avoid the use of spinal anesthetics due to the potential for a profound and unpredictable drop in blood pressure with this technique.
 - (2) This being said, patients have successfully been managed with regional anesthesia provided they are dosed correctly and slowly.

- v. A gentle induction with etomidate and fentanyl will likely provide hemodynamic stability from a blood pressure and heart rate standpoint.

Intra-operative

1. During this case, the patient's heart rate and blood pressure need to be continually observed and addressed to reduce peri-operative complications.
2. Despite not having any areas of ischemia on her pre-operative stress echocardiogram, she is at an increased risk to develop ischemia as a result of higher oxygen requirements of her myocardium.
3. Her history of hypertension as well as aortic stenosis have likely led to hypertrophy of her myocardium, bringing about these increased oxygen requirements.
4. During the procedure, with swings in blood loss or fluid administration, we can see episodes of hyper- or hypotension occurring.
5. In addition, she may also develop tachycardia, further adding to her oxygen requirements.
6. Any deviation from her baseline hemodynamic status should be addressed, albeit slowly and carefully, to avoid large swings in blood pressure or profound decreases in heart rate.
7. Since these patients do not tolerate atrial fibrillation, you must be prepared to cardiovert them to restore them to sinus rhythm.

Post-operative

1. Following her procedure, close monitoring of her blood pressure, heart rate, and end-organ perfusion needs to continue.
2. The stress response to surgery and shifts in fluids can occur for many days following the operation.
3. Restoration of her baseline medications and maintenance of her blood pressure and heart rate to her pre-surgical baseline are the best for this patient.

BIBLIOGRAPHY

1. Kurup V., Haddadin A. S. Valvular heart diseases. *Anesthesiology Clinics* 2006; 24:487-508.
2. Rapaport E. Natural history of aortic and mitral valve disease. *Am J Cardiol* 1975; 35:221.
3. Otto C.M. Valvular aortic stenosis: Disease severity and timing of intervention. *J Am Coll Cardiol* 2006; 47:2141-51.

37. Cardiopulmonary Bypass (CPB) and Associated Anticoagulation¹⁻¹¹

Donald M. Voltz, MD

SAMPLE CASE

A 69-year-old female is scheduled to undergo a coronary revascularization as well as an aortic valve replacement for severe aortic stenosis. Her past medical history is significant for a non-ST segment MI, hypertension, progressive aortic stenosis with mildly depressed left ventricular function, and diabetes mellitus. Her past surgical history is significant for endoscopic sinus surgery 3 years prior and a left fem-distal bypass graft 2 years ago. She does not report any adverse anesthetic complications but does report a fall in her platelet count when she was hospitalized for lower extremity ischemia. What are your concerns? What preoperative workup would you like? How does her medical history affect your intraoperative plans?

CLINICAL ISSUES

Main Components of a Cardiopulmonary Bypass System

1. Diverting blood away from the heart and lungs is of paramount importance for cardiac surgery; however, the systemic functions must be maintained if one is to avoid serious physiologic problems.
2. The evolution of the cardiopulmonary bypass machine has resulted in sophisticated ways to maintain the normal functions of the heart and lungs during the period of bypass.
3. The main functions that are taken over by the CPB circuit are as follows:
 - a. **Oxygenation of venous blood:** one of the primary functions of the lungs is to saturate venous blood returning from the body, through the inferior and superior vena cava, with oxygen.
 - i. During cardiac surgery and CPB, the surgeon places one or two cannula into the vena cava in order to drain venous blood into the CPB circuit's venous reservoir.
 - ii. This blood is then passed through either a membrane or bubble oxygenator.
 - iii. Once the blood picks up oxygen, it passes through a filter to remove any cellular fragments or aggregated blood components and is also either cooled (during the early phase of surgery) or warmed (near to conclusion of CPB) before passing on to the next stage.

- iv. The blood is delivered back to the patient by a roller pump.
- v. Freshly oxygenated blood is returned to the patient through the aortic cannula just distal to the aortic cross clamp for systemic perfusion before the cycle is repeated.
- b. **Removal of carbon dioxide:** in addition to restoring oxygen balance to the blood, the lungs are equally important in removing waste from the system through the exhalation of carbon dioxide.
 - i. The CPB machine must take over since the lungs do not receive any blood flow and are not ventilated during CPB.
 - ii. Carbon dioxide is extracted from the blood during the course of adding oxygen back.
 - iii. The amount of carbon dioxide removed and therefore returned to the patient can be adjusted by the use of frequent blood gas samples.
- c. Maintenance of system perfusion pressure
 - i. During CPB, roller pumps maintain a continuous pressure.
 - (1) During normal cardiac function, peripheral flow occurs in a pulsatile manner.
 - (2) Tissue perfusion is better with pulsatile flow.
 - (3) The CPB roller pumps do not supply pulsatile pressure.
 - (a) Mean perfusion pressure must be maintained to avoid organ system dysfunction or irreversible damage.
 - (b) In adult CPB, a perfusion pressure between 50 and 80 mm Hg is recommended to maintain systemic organ function.

4. One of the main problems with CPB is that the entire circuit is foreign and highly thrombogenic necessitating the use of profound anticoagulation to prevent any disruption in patient blood flow and organ perfusion¹.

Evaluation of Adequacy of Anticoagulation for Cardiopulmonary Bypass²

1. In order to successfully transition a patient through cardiac surgery, we need to have profound anticoagulation so thromboses do not occur in the CPB circuit and then relatively

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quickly restore the coagulation system back to normal following separation from the bypass circuit so the patient does not exsanguinate from all of the arterial and venous injuries that occurred to repair the heart.

2. In order to achieve these extremes, heparin is often utilized for the following reasons.

- a. Consistent in its dose and the achievement of adequate anticoagulation.
- b. Rapid onset time
- c. Easy to monitor in the operating room setting
- d. Adequate therapeutic window
- e. Specific means of reversing its effects to restore coagulation back to baseline

3. The main modality to monitor anticoagulation with heparin in the operating room is the activated clotting time (ACT).

- a. A small aliquot of blood is added to a medium, which stimulates thrombosis to occur.
- b. Blood reacts with diatomaceous earth present in standard ACT vials.
 - i. The diatomaceous earth stimulates the coagulation system, leading to the deposition of fibrin strands and the eventual formation of a stable clot.
 - ii. The reaction is allowed to occur under a constant 37 °C temperature while the time required to form a clot is measured.

c. In a normal, pre-heparinized state, the ACT ranges from 90 to 150 seconds.

- i. Following the administration of heparin (300–400 U/kg is the standard initial dose used in cardiac surgery), an ACT is repeated after 2 minutes.
- ii. Most surgeons desire an ACT of greater than 400 seconds before initiating CPB; however, some advocate an ACT of greater than 480 seconds.

(I) A study looked for fibrin monomers in blood at different levels of anticoagulation and found that an ACT above 400 seconds did not contain any fibrin monomers, prompting this number to be used for CPB.³

Despite a Full Dose of Heparin, the Patient ACT Does Not Reach an Adequate Level to Safely Proceed to CPB. What is The Likely Mechanism for This Lack of Anticoagulation?⁴

1. Heparin is not a direct acting anticoagulant; instead, it works through the body's intrinsic anticoagulation system.

- a. Normally, the coagulation cascade is balanced by the anticoagulation system.
- b. Numerous factors such as protein C, protein S, and anti-thrombin III become activated when the other coagulation factors are activated.
- c. The purpose of these enzymes is to down-regulate the coagulation and restore the systemic system to its baseline state.
- d. Heparin enhances the activity of anti-thrombin III leading to an enhanced destruction of thrombin and thereby making it difficult to form clots.

2. When patients are on heparin infusions or have hepatic dysfunction so that they do not adequately synthesize factors of coagulation, they can become deficient in anti-thrombin III thereby limiting the effectiveness of heparin.

3. In this case discussion, if the patient was admitted to the hospital and placed on heparin for her non-ST segment MI, she might have decreased levels and/or a decreased activity of anti-thrombin III resulting in an inability to reach adequate therapeutic ACT values.

4. In patients for whom one can not reach an adequate ACT value to go on CPB even with an increased dose of heparin, replacement of anti-thrombin III will likely lead to adequate anticoagulation.

- a. Fresh frozen plasma (FFP) contains pro-coagulant factors as well as anti-thrombin III.
- b. For patients who are not adequately elevating their ACT level prior to CPB, the administration of 1–2 units of FFP will replace the level and activity of anti-thrombin III and restore heparin's ability to provide anticoagulation.

CPB Effects on the Coagulation System^{2,5}

1. During CPB, numerous mechanical and physiologic changes are placed upon the blood, leading to potential adverse effects on the natural coagulation system.

2. Ideally, heparin would lead to profound anticoagulation, which is reversed once the patient is separated from the CPB circuit.

3. Passage of blood through the CPB circuit results in

- a. Destruction of red blood cells
- b. Activation of platelets
- c. Destruction of platelets
- d. Stimulation of both the inflammatory and complement system leading to further stimulation of the coagulation system following the reversal of heparin.
- e. All of these effects are partially related to the total CPB time.

i. The longer a patient is on CPB, the more profound the destruction of red cells, depletion of platelets, and consumption/dilution of coagulation factors.

ii. In this patient, the addition of a valve replacement to a coronary artery bypass increases the time she will be on bypass, and therefore this patient has a higher likelihood of requiring blood or blood product administration.

4. In addition to all of the physiologic and mechanical effects of CPB, most patients undergoing cardiac surgery are cooled to varying degrees.

- a. Hypothermia decreases how well enzymes function.
 - i. Since the coagulation system consists of proenzymes, their activity is not as great at colder temperatures.
 - ii. One factor to consider in coagulopathies following cardiac surgery is the patient's core body temperature.
 - iii. Restoration of this to normal levels is an important component with regard to coagulation.

5. Platelet activation and destruction can lead to profound decreases in platelet levels and/or function requiring the

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administration of platelets once the patient is safely separated from bypass.

6. Numerous ions and other factors are involved with the initiation and propagation of clotting at sites of endothelial injury.

- a. Factors such as ADP and calcium are both involved with normal clotting.
- b. Medications that interfere with these cofactors or lack of adequate levels can potentiate coagulopathies in the cardiac surgery patient.

Mode for the Reversal of Heparin after Separation from CPB

1. Heparin is the anticoagulant of choice for cardiac surgery based on

- a. Quick onset
- b. Adequate therapeutic window
- c. Quick and effective reversal of its effect.

2. The *in vivo* half-life of heparin is 90 minutes; however, this is far too long to allow for its natural removal for restoration of the coagulation system.

- a. Intravenous protamine is used to reverse the effects of heparin.

3. Following the administration of protamine, ACT returns to pre-anticoagulation levels within a few minutes due to the strong electrostatic interaction between heparin and protamine.

- a. One must keep in mind that protamine is itself a weak anti-coagulant and if a large excess of protamine is administered to reverse the effect of heparin, anticoagulation can be the result instead of a restoration of normal clotting functions.
- b. Each milligram of protamine effectively neutralizes 100 U of heparin.
- c. In a standard cardiac case, the initial 300–400 U/kg administration of heparin is reversed, even though additional heparin may be used by the perfusionist during the course of CPB.
 - i. The initial heparin dose is used for the reversal calculation to account for metabolism and removal of heparin from the system.

Adverse Effects of Protamine¹

1. Protamine is a relatively safe polypeptide medication derived from salmon sperm used primarily for the reversal of heparin's activity and restoration of normal coagulation.

2. There are three potential reactions that can occur with the administration of protamine.

- a. Patients who have been exposed to protamine in the past or similar antigens have the potential, albeit a rare adverse reaction, to develop an anaphylactic reaction resulting in profound vasodilatation and cardiovascular collapse.
 - i. Previous exposure
 - ii. Fish allergies

iii. Diabetics using insulin (NPH): since protamine is used to prolong insulin effects

iv. Men who have undergone vasectomies or episodes of orchitis are also at risk for exposure to antigens leading to immunologic sensitization.

(1) Treatment of this reaction is the same as any other anaphylactic reaction

(a) Support of the cardiovascular system

(b) Administration of diphenhydramine and epinephrine to block histamines effects as well as halt the degranulation of leukocytes

b. A second severe, but also rare reaction is fulminate pulmonary vasoconstriction that can lead to right ventricular failure.

i. This reaction is not well understood; however, patients with pre-existing problems with elevated pulmonary pressures and/or right ventricular function seem to be more at risk for this complication.

ii. In the event that this occurs, stopping the administration of protamine and supporting the cardiovascular system are important.

iii. There has been some suggestion that the administration of intravenous methylene blue might help to counteract the precipitous increase in pulmonary pressures.

c. Finally, too rapid administration of protamine can lead to a decrease in intra-vascular calcium as well as stimulate the release of histamine leading to a drop in systemic vascular resistance and hypotension.

i. This reaction is directly related to the speed with which protamine is administered and it is recommended that no more than 50 mg of protamine be administered over a 10 minute period.

Contraindications to Heparin Use^{6,7}

1. The only contraindication to the use of heparin for cardiac surgery is a history of heparin-induced thrombocytopenia (HIT).

a. This is not entirely clear since some patients who have experienced HIT have been safely anti-coagulated with a heparin for cardiac surgery. (For more information on this, see next section.)

Potential Coagulation Problems that Occur with Cardiopulmonary Bypass

1. Heparin-induced thrombocytopenia (HIT)

a. Develops in 5–28% of patients who receive heparin.⁶

b. There are two commonly agreed upon subtypes of this syndrome.

i. HIT type I

(1) Results in a mild decrease in the number and/or function of platelets following the administration of heparin.

(2) This drop in platelet levels is thought to be due to an enhanced aggregation of platelets.

(3) Heparin binds to the surface of platelets as well as to platelet factor 4.

(4) This binding facilitates platelets clumping together and these aggregates are removed from circulation by the reticulo endothelial system.

ii. HIT type II

(1) More severe syndrome

(2) Shares some similarity with type I

(3) Patients who present with HIT type II tend to have received heparin for a more prolonged time period (5-10 days).

(4) There is a decrease in platelet levels due to aggregation and removal from circulation; however, there is an additional antibody-mediated effect.

(a) Antibodies are generated and bind to the heparin-platelet factor 4 complex.

(b) This leads to activation of the inflammatory system as well as activation of the complement cascade.

(c) These reactions and activations appear to lead to endothelial cell damage and additional activation and clumping of platelets at these areas of endothelial damage.

(5) Thrombotic complications in patients with HIT have been reported to be as high as 20% and if these clots occur in the heart, lungs, or brain, a mortality rate as high as 40% has been reported in patients.^{6,7}

c. A final understanding of HIT has not yet been completed.

i. Measurement of antibody levels in patients who survive an HIT type II reaction become undetectable several weeks after cessation of heparin.

ii. In addition, repeated exposure to heparin does not always reproduce the HIT type II reaction.

iii. It is unclear what to do with these patients who are in need of cardiac surgery and anticoagulation.

iv. There have been reports of patients who were removed from heparin for a number of weeks resulting in a disappearance of antibodies and then a safe, brief administration of heparin for CPB and surgery.⁸

Alternatives for Anticoagulation During CPB

There are a few experimental alternatives to heparin for anticoagulation during cardiac surgery. None of these compounds have been approved for clinical use at this point; however, they have interesting properties and may at some point be used for clinical anticoagulation. One example is

1. Bivalirudin

a. Small peptide with a short half-life: 24 minutes

b. It was synthesized from hirudin and acts by binding to the active site on thrombin, preventing its ability to cleave fibrinogen into fibrin.

c. It is cleaved by thrombin itself leading to elimination in the plasma and is not dependent on an organ for its removal.

i. There is no specific reversal agent for bivalirudin; one must wait for it to be cleaved by thrombin for restoration of normal clotting activity.

d. In order to be used for cardiac surgery, a bolus is delivered followed by an infusion to maintain anticoagulation for the duration of the CPB.

e. This peptide has been used for some interventional cardiology procedures; however, monitoring its activity is not as easy as for heparin.

f. The anticoagulation effects of bivalirudin are not accurately measured with ACT and it is recommended that an ecrin clotting time be utilized to guide therapeutic levels.

i. The ecrin clotting time is not a standard assay as of yet, making this difficult to use in surgery.

Role of Anti-fibrinolytic Medications in CPB

1. The coagulation system tends to favor anticoagulation until endothelial damage occurs.

2. In a normal setting, the endothelial cells play a role in the prevention of platelet activation, aggregation, and the generation of fibrin clotting.

a. Endothelial cell membranes contain heparin sulphate, which inhibits clotting factors.

b. Endothelial cells are also responsible for releasing prostacyclin, a molecule that leads to vasodilatation and stabilizes platelet cells, preventing their activation and the formation of a platelet plug.

c. Endothelial cells, except those present in the cerebral circulation, synthesize and display thrombomodulin on their cell surface.

i. Thrombomodulin greatly decreases activation of the clotting cascade.

d. When endothelial cells are damaged, these factors are no longer present and the balance is switched to a pro-thrombotic state in the local area where the endothelial cells are absent.

3. When endothelial cells become damaged, the sub-endothelial matrix becomes exposed and there is an activation of the clotting pathway.

4. Thrombosis occurs when both platelets and fibrin become intertwined, filling the area of endothelial damage.

a. The first phase of clotting occurs (after endothelial damage) with a decrease in prostacyclin and an increase in thromboxane.

i. This results in vasoconstriction.

b. A platelet plug forms at the area of endothelial damage and this coupled with the vasoconstriction limits bleeding and provides a scaffolding upon which a clot can form.

5. Tissue factor becomes exposed when the endothelial cells are damaged and is the primary initiator of the clotting cascade.

6. Once clotting is initiated, there is activation of not only pro-coagulant enzymes, but also activation of molecules that will help to down-regulate and stop the clotting process once the tissue damage has been occluded.

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- a. Thrombin appears to be the main initiator of clotting; however, it has a central role in turning off the clotting system.
- 7. The final phase of coagulation is a remodeling of the clot and a direct repair of the endothelial cell injury.
 - a. The clotting cascade activates the fibrinolytic system whose role is to remodel and degrade clot formation, allowing for endothelial cell repair.
- 8. CPB stimulates the coagulation system as well as the fibrinolytic system^{9, 10, 11}.
 - a. Specific drugs are available to decrease the activation of the fibrinolytic system, decrease the destruction of clots, and decrease the amount of bleeding.
 - b. During cardiac surgery, as well as other major surgeries where there is a supra-physiologic increase in the fibrinolytic system, the use of these agents can significantly decrease the amount of blood loss and thereby decrease the transfusion requirements.
 - c. One must be cautious with their use, however, because complete inhibition of the fibrinolytic system can lead to an increased risk of thrombosis.
 - d. In cardiac surgery, there are three main anti-fibrinolytic drugs that have been successfully used to decrease peri-operative blood loss and decrease the amount of blood transfusions.
 - i. These medications are epsilon-amino caproic acid (amicar), tranexamic acid, and bovine pancreatic trypsin inhibitor (aprotinin).
 - ii. Tranexamic acid and epsilon-amino caproic acid are lysine analogs that reversibly bind to plasminogen, preventing it from being activated.
 - iii. Aprotinin, a serine protease, decreases activation of fibrinolysis. This action is similar to that of epsilon-amino caproic acid and tranexamic acid; however, it also binds to kallikrein, an initiator of the clotting cascade, thereby limiting activation of the intrinsic clotting pathway.
 - (1) Recently, aprotinin has not been used in cardiac surgery due to increases in renal dysfunction and higher cardiac mortality; however, this issue has not been completely clarified. One of the confounding issues surrounding the use of aprotinin has been renal dysfunction. Prolonged CPB has been implicated in peri-operative renal dysfunction and some authors argue that complex surgeries requiring prolonged CPB are more likely to receive aprotinin over the other antifibrinolytic agents. Randomized controlled studies might better answer these questions.^{10,11}

Additional Medications This Patient Might Have Been Exposed to That Can Lead to Coagulopathies During CPB

- 1. Patients often present to the operating room following an unsuccessful intervention in the cardiac catheterization lab.

- a. Anti-platelet medications
 - i. A class of medications that are great for minimally invasive interventions such as balloon angioplasty or stenting.
 - ii. Peri-operative period risk: increased blood loss
 - iii. The most commonly used anti-platelet medication, especially in patients who are admitted to the hospital with an acute myocardial infarction, is aspirin.
 - (1) Aspirin irreversibly inhibits the enzyme cyclo-oxygenase which is in the prostaglandin synthesis pathway and leads to the production of thromboxane, a potent platelet activating molecule.
 - (2) Aspirin, due to its irreversible inhibition of cyclo-oxygenase, causes platelet dysfunction for the life of the platelet: 10 days.
 - iv. In addition to aspirin, potent anti-platelet medications are used by cardiologists to decrease the risk of thrombosis in coronary arteries following interventions to restore their patency.
 - (1) Glycoprotein IIb/IIIa receptor
 - (2) This receptor is inhibited by a number of agents used in the cardiology suite.
 - (a) Abciximab (Reopro)
 - (b) Eptifibatid (Integrelin)
 - (c) Tirofiban (Aggrastat)
 - (i) All work by binding to this receptor and greatly decreasing the activation and aggregation potential of platelets.
 - (ii) This is a great benefit when thrombotic stents or the exposure of atherosclerotic plaques occur in angioplasty; however, when the patient proceeds to surgery, the lack of platelet function greatly limits the initial phase of clot formation.
 - v. A third class of anti-platelet medications acts by binding to and inhibiting platelet ADP receptors.
 - (1) ADP is a potent molecular stimulant of platelet aggregation.
 - (2) Ticlopidine (Ticlid) and clopidogrel (Plavix) are two medications in this class.
 - (3) All of these medications have been shown to cause an increase in operative blood loss leading to a higher incidence of peri-operative blood transfusions and/or a higher requirement for re-exploration due to bleeding or a bleeding complication such as cardiac tamponade.
- 2. Since none of these medications have specific reversal agents, the only treatment is to administer platelets to patients who have received these medications prior to cardiac surgery.
 - a. One consideration we must be aware of with these medications is that they can have an effect on the platelets that are transfused as well as the patient's native platelets.
 - b. Transfusion of platelets will interact with these medications until the free serum level of the anti-platelet medications is zero.

37. Cardiopulmonary Bypass

- c. To reduce the indiscriminate transfusion of platelets in these patients, specific platelet function testing may be useful although it can take time to perform in the operating room.
- d. The use of a thromboelastograph has shown some utility in the operating room to diagnose and monitor the treatment of platelet dysfunctions as well as other coagulopathies.
 - i. Routine use of these devices has not yet occurred and some skill is required to interpret the results.



KO TREATMENT PLAN

Pre-operative

1. The anti-coagulation in this patient can present some challenges.
 - a. The history of a fall in platelet count following her revascularization surgery is of note.
 - b. In patients who have had a drop in platelet count following the administration of heparin, one must be concerned about heparin-induced thrombocytopenia.
 - c. An evaluation of her current platelet count should be done as well as a determination of the presence of anti-heparin antibodies.
 - i. This is a specialized test necessitating the involvement of a hematologist for administration and interpretation of the result.
 - d. Currently, there is no good clinical substitute in the United States for heparin.
 - e. Heparin has been used safely in CPB in patients who have a history of HIT; however, consultation with a hematologist would be warranted.

Intra-operative

1. The presence of aortic stenosis along with coronary artery disease warrants a combined aortic valve replacement as well as coronary revascularization.
 - a. This will prolong CPB and therefore place this patient at a higher risk for coagulopathies.
 - i. Knowing this, as well as her history of thrombocytopenia, she will likely require the administration of platelets and possibly coagulation factors once she is weaned from CPB.
 - b. Her prior history of vascular surgery suggests that she has been exposed not only to heparin, but also likely was reversed with protamine.

- i. Keeping this in mind will be important during reversal of her heparin.
- ii. Prior exposure to protamine places her at a small increased risk of undergoing an anaphylactic reaction to repeat exposure.
- iii. Test-dosing the protamine and closely monitoring for any adverse reaction will be important.

Post-operative

1. Following the separation from CPB and the reversal of her heparinization with protamine, close monitoring of her coagulation system and blood count will determine whether additional platelets or clotting factors will be required.
2. With her prior history of thrombocytopenia, daily monitoring of her platelet level and potentially the measurement of anti-heparin antibodies will be important to ensure she does not become profoundly coagulopathic.

BIBLIOGRAPHY

1. Miller, RD. *Miller's Anesthesia*, 6th ed. Elsevier, Churchill Livingstone, 2005. Philadelphia, PA.
2. Despotis GJ, Joist JH. Anticoagulation and anticoagulation reversal with cardiac surgery involving cardiopulmonary bypass: an update. *J Cardiothorac Vasc Anesth* 13(4 Suppl 1):18-29, 1999.
3. Young, JA, et al. Adequate anticoagulation during cardiopulmonary bypass determined by activated clotting time and the appearance of fibrin monomers. *Ann Thorac Surg* 26:231, 1978.
4. Despotis GJ, et al. Antithrombin III during cardiac surgery: effect on response of activated clotting time to heparin and relationship to markers of hemostatic activation. *Anesth Analg* 85(3):498-506, 1997.
5. Hartmann M, et al. Effects of cardiac surgery on hemostasis. *Transfus Med Rev* 20(3):230-41, 2006.
6. Castelli R, et al. Heparin induced thrombocytopenia: pathogenetic, clinical, diagnostic and therapeutic aspects. *Cardiovasc Hematol Disord Drug Targets* 7(3):153-62, 2007.
7. Kam PC. Anaesthetic management of a patient with thrombocytopenia. *Curr Opin Anesthesia* 21(3):369-74, 2008.
8. Warkentin, TES. Heparin-induced thrombocytopenia and the anesthesiologists. *Can J Anaesth* 49:S36-49, 2002.
9. Taneja R, et al. Perioperative coagulation management and blood conservation in cardiac surgery: a Canadian survey. *J Cardiothorac Vasc Anesth* 22(5):662-9, 2008.
10. Ngaage DL, et al. Aprotinin in primary cardiac surgery: operative outcome of propensity score-matched study. *Ann Thorac Surg* 86(4):1195-202, 2008.
11. Maslow AD, et al. Perioperative renal outcome in cardiac surgical patients with preoperative renal dysfunction: aprotinin versus epsilon aminocaproic acid. *J Cardiothorac Vasc Anesth* 22(1):6-15, 2008.

38. Carotid Surgery^{1–20}

Matthew A. Joy, MD

SAMPLE CASES

An 80-year-old male presents for a left carotid endarterectomy (CEA). How would you monitor the patient's central nervous system (CNS) during the procedure? Would you use a regional or general anesthetic? Why?

CLINICAL ISSUES

Introduction

1. One of the more challenging cases facing anesthesiologists is the management of the patient presenting for a carotid endarterectomy (CEA).

a. The patient population presenting with carotid atherosclerotic stenosis (CAS) typically possesses multiple medical problems.

i. Significant coronary artery disease (CAD): may predispose the patient to significant hemodynamic changes in the operating room.

(1) Accordingly, cardiac complications arising perioperatively during CEA still remain a major source of morbidity and mortality.

ii. Hypertension

iii. Diabetes

iv. Tobacco abuse and its associated pulmonary consequences

v. Angina and previous myocardial infarction

2. The current ACC/AHA guidelines for cardiac risk stratification for non-cardiac surgical procedures list carotid surgery as an intermediate risk procedure.

a. Reported risk: 1–5%

b. The anesthesiologist managing these patients with cardiovascular disease is encouraged to read these guidelines in depth.¹

Definition of the CEA

1. The CEA is a surgical procedure that attempts to remove an atherosclerotic carotid artery plaque.

2. It is one of the more commonly performed vascular procedures in the United States.

3. CEA is done to reduce the potential risk of stroke in selected patients.

4. Locations

a. Carotid division, into the external and internal carotid arteries

b. Proximal internal carotid artery²

5. Plaques tend to form at arterial branches due to turbulent blood flow.

Pre-operative Assessment

1. The patient presenting for CEA surgery poses numerous issues for the surgeon and anesthesiologist.

2. Patient selection is critical for successful outcomes, and although operative candidates are primarily determined by the vascular or neurosurgeon performing the procedure, the anesthesiologist should be familiar with the published guidelines for appropriate patient choice and identification of risk factors and predictors of morbidity and mortality, which may have an impact on outcome.^{3,4}

a. The general indications are that symptomatic patients with angiographic evidence of 70–99% carotid stenosis are candidates for a CEA.

b. Individuals should not be considered for surgery if the stenosis is less than 50%.³

c. For asymptomatic disease with stenosis between 60–99% CEA can be considered.

d. Stratification of symptomatic versus asymptomatic disease is only one part of the criterion for selection of patients for surgery.

e. Other clinical data should be ascertained prior to a CEA. (See following)^{3,5}

3. Critical information obtained should include

a. Overall pre-operative health status:

i. Presence of clinical markers

(1) Major, intermediate, and minor clinical predictors

(2) Functional capacity

(3) Cardiac risk of the noncardiac procedure: as mentioned, CEA is typically regarded as an intermediate-risk procedure.

b. Comprehensive cardiac evaluation is indicated in selected patients

i. Typically those patients with major clinical predictors of increased cardiac risk have^{1,6,7}

(1) Unstable coronary syndromes

(2) Decompensated heart failure

(3) Significant arrhythmias

- (a) High grade atrioventricular block
- (b) Symptomatic ventricular arrhythmias in the presence of underlying heart disease
- (c) Supraventricular arrhythmias with an uncontrolled ventricular rate
- (4) Severe valvular disease

4. The ultimate goal is to identify patients who are at significant risk for adverse myocardial outcomes and reduce the risk by either peri-operative management (e.g., aggressive β -blockade, blood pressure control, smoking cessation) or pre-operative cardiac intervention, as might be the case in the very small subset of patients with highly symptomatic coronary disease and carotid stenosis.^{2,6}

Surgical Considerations

1. The degree of carotid stenosis of the ipsilateral artery as well as the degree of contralateral carotid disease is critical for collateral perfusion during cross-clamping.

2. Clearly define whether the patient is presenting for asymptomatic versus symptomatic revascularization.

a. This has direct ramifications on surgical indication, risk stratification, and post-operative neurologic outcome.

i. Categorization of patients into symptomatic versus asymptomatic is derived from the combined results of multiple prospective trials, from which these evidence-based recommendations are derived.^{3,8,9,10,11}

ii. The type of symptoms, if present, should also be clearly delineated as part of the patient's risk profile, as these variables are also factored in patient selection, prognostication, and timing of surgery.

- (1) Transient ischemic attacks (TIA)
- (2) Reversible ischemic neurologic deficit (RIND)
- (3) Cerebral vascular accident (CVA)
- (4) Neurologically symptomatic patients
 - (a) Active pre-operative TIAs are typically in need of more urgent carotid revascularization than the patient with a recent CVA.^{7,11}

3. As previously mentioned, the angiographic anatomy has direct bearing on patient selection and outcome predictions.

a. Additionally, the native carotid anatomy influences the selection of

- i. Neuromonitoring: EEG, BIS, carotid stump pressure, cerebral oximetry
- ii. Surgical approach: shunting, patch angioplasty
- iii. Anesthetic choice: regional, local, and general

Anesthetic Approaches

1. Regional technique (awake)

a. Several different approaches have been described for performing regional anesthesia for a CEA.

i. Cervical plexus block (CPB) either deep and/or superficial block involves blockade of the cervical plexus (cervical nerves 2-4).

(Although both blocks have advantages and disadvantages, both are considered to be equally efficacious.⁵)

(1) Deep CPB

- (a) Frequently utilized because of its reliability
- (b) Most commonly employed regional technique for CEA²
- (c) Disadvantages
 - (i) Diaphragmatic dysfunction: due to inadvertent phrenic nerve block
 - (ii) Greater risk of epidural, subarachnoid, and vertebral artery injection^{2,5}

(2) Superficial CPB

- (a) Lower reported complication rate and easier to perform than the technically challenging deep CPB, the superficial CPB may be the preferred technique for regional CEA anesthesia in select patients.^{5, 11, 12,13}

ii. Cervical epidural block (CEB)

(1) Utilized by a few experienced regional anesthesiologists via a catheter placed in the epidural space.

(2) This technique has not gained widespread use due to the potential of associated blockade of the thoracic nerve roots which could result in hypotension, bradycardia, and possible respiratory failure due to changes in pulmonary function.^{2,10,14}

(3) This block has not been reported to be more efficacious than both deep and superficial CPB, and carries more risk potential; therefore it is typically not recommended.¹⁵

iii. Local anesthetic infiltration block

(1) Straight local anesthetic infiltration performed by either the surgeon or anesthesiologist.

(2) This technique is rarely performed due to inefficacy, resulting commonly in significant patient discomfort.

b. Despite regional modalities being utilized much less frequently than general anesthesia for CEA, the cervical plexus block (CPB) remains a useful technique in selected patients undergoing this procedure, especially when there is concern of severe carotid disease and possible plaque embolization during the surgery, which might not be discernible in an unconscious patient under general anesthesia regardless of the neuromonitoring techniques employed.

c. Unlike cerebral monitors, which lack sensitivity and specificity (see following), an awake patient provides a very specific and sensitive indicator of cerebral function.

d. Before one embarks upon a regional technique, it is imperative that this approach be acceptable to both the surgeon and patient; hence a lengthy discussion regarding the anesthetic plan and expectations should be held with both the patient and surgeon pre-operatively.

TKO: It is beyond the scope of this chapter to review the detailed techniques involved in performing the previously mentioned blocks. However, anesthesiologists should be

38. Carotid Surgery

familiar with both deep and superficial CPB, and an ABA candidate should be able to accurately describe the technique employed, regional anatomy, landmarks, distribution of anesthesia, block dynamics, choice of local anesthetic, and possible complications involved. Additional reading can be obtained from any standard regional anesthesia textbook^{16,17,18} and the informative website: www.nysora.com.

2. General anesthesia

- a. The overall majority of CEA cases in North America are still performed with a general anesthetic technique.⁸
- b. Although an awake patient is considered the gold standard for cerebral monitoring for a CEA, there is no conclusive evidence that this procedure is best performed under a regional technique.^{19,20}
- c. In addition to maintaining hemodynamic stability, a general anesthetic poses additional challenges compared to regional techniques.
 - i. Unlike regional anesthesia, additional neuromonitoring techniques may need to be employed for assessment of cerebral perfusion, since an awake responsive patient is no longer an option (see below for neuromonitoring techniques).
- d. Several different techniques for general anesthesia have been proposed, each with purportedly superior advantages.
 - i. Total intravenous anesthesia techniques (TIVA)
 - (1) Induction and maintenance with propofol and remifentanyl
 - (2) Advocates cite superior hemodynamic stability and rapid emergence as justification for this particular technique.^{2,14}
 - ii. Inhalational technique
 - (1) Takes advantage of the theoretical improved cerebral protection with agents such as isoflurane as the basis of their selection.
 - (2) It is recommended that if used as part of a general anesthetic technique, nitrous oxide should be discontinued prior to carotid artery cross-clamping in order to minimize the risk of air embolism that may occur during this phase of the procedure.¹⁴
- e. Regardless of the general anesthetic technique employed the overall goal should remain the same.
 - (1) Blunting of noxious stimuli: intubation, incision
 - (2) Reliable hemodynamic control
 - (3) Maintaining cardiovascular stability (especially in this patient population)
 - (4) Expeditious emergence from the anesthetic, allowing for rapid neurologic assessment
 - (5) An anesthetic that does not negatively impact the neuromonitoring technique employed (see below) for the surgery

Monitoring Techniques

1. Standard ASA monitors
2. Continuous ST monitoring of leads II and V5

3. Direct invasive intra-arterial blood pressure monitoring
4. Rarely is pulmonary artery catheter (PAC) monitoring indicated for this surgery, and clearly the routine use of this catheter for CEA procedures is unsupported.

5. Cerebral monitors

- a. A regional anesthetic technique with an awake and responsive patient likely provides the gold standard neurologic monitor for CEA procedures.^{2,7,11,14,15}
- b. General anesthetics require other modalities of monitoring cerebral ischemia, and anesthesiologists should be aware of the advantages and disadvantages of the various techniques employed.
 - i. A 16 channel electroencephalogram (EEG)
 - (1) Considered by some sources as the gold standard of neuromonitoring under general anesthesia.¹⁵
 - (2) Advantages
 - (a) Ready availability
 - (b) Reliability
 - (c) Correlation with cerebral ischemia
 - (3) Disadvantages
 - (a) Possible need of a trained technician for interpretation
 - (b) High false positive rate
 - (c) Possible anesthetic agent influences
 - (d) Inability to detect subcortical ischemia^{2,11,14,15}
 - ii. Processed EEG
 - (1) BIS monitoring techniques
 - (2) Advantages
 - (a) Availability
 - (b) Ability to identify severe cerebral ischemia
 - (c) Ease of use
 - (3) Limitations
 - (a) Reliability
 - (b) Inability to detect focal ischemia¹⁵
 - iii. Somatosensory evoked potentials (SSEP)
 - (1) Advantages
 - (a) Equivalent efficacy to EEG
 - (b) Detects deep brain structure injury
 - (2) Disadvantages
 - (a) Complexity
 - (b) Need for a trained technician
 - (c) Limitations of inhalational anesthetic use due to effects on interpretation^{2,11,14,15}
 - iv. Transcranial Doppler (TCD)
 - (1) Advantages
 - (a) Ability to monitor cross-clamp hypoperfusion and shunt malfunction
 - (b) Ability to assess cerebral blood flow and embolic phenomena
 - (2) Limitations
 - (a) Technical complexity involving monitor placement and interpretation of TCD data
 - (b) May require additional trained personnel^{2,11,14,15}
 - v. Cerebral oximetry devices (i.e., near infrared spectrophotometry [NIRS])

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- (1) Advantages
 - (a) Simplicity of use
- (2) Disadvantages
 - (a) Low sensitivity and specificity⁵

vi. Carotid stump pressure (CSP)

- (1) This is a simple technique in which the surgeon measures the mean arterial carotid pressure distal to the cross-clamp.
- (2) Presently, clinicians utilize 50 mm Hg as a cutoff for shunting.^{5,15}
- (3) Advantages
 - (a) Simplicity
 - (b) Lack of expense
- (4) Disadvantages
 - (a) Lack of validation
 - (b) Lack of a critical CSP value in assessing collateral flow, below which a shunt should be placed^{2,5,11,15}

6. The importance of such monitoring

- a. There is a subsequent clinical response if signs of altered cerebral perfusion are noted during carotid cross-clamping, which in most centers is the placement of a carotid shunt (selective shunting).
- b. The anesthesiologist managing these patients along with the candidate preparing for a clinical scenario of CEA should be aware of the signs and indexes of altered perfusion of these devices, especially the CSP, its clinical significance, and implication on intra-operative management.



KO TREATMENT PLAN

Pre-operative

1. Complete history and physical
 - a. Patients often have multiple associated medical problems.
 - i. Coronary artery disease: angina and myocardial ischemia
 - ii. Hypertension
 - iii. Diabetes
 - iv. History of CVA
 - (1) Ask about residual symptoms.
 - (2) This is especially relevant if a regional technique is planned (i.e., the patient must be able to follow commands).
 - v. Smoking history and related lung problems
2. Labs
 - a. This patient population is frequently on anticoagulant therapy.
 - i. Check with the prescribing physician before discontinuation, bridging therapy if needed, and resumption of therapy.
 - (1) Especially in the case of clopidogrel and warfarin
 - ii. These patients and others at risk for coagulation defects should have at minimum the following labs.

- (1) Coagulation panel (PT, INR, PTT)
- (2) Check a complete blood count (CBC) and electrolyte panel for baseline values for all patients.
- (3) For anemic patients, a blood type and screen is probably not unreasonable.

3. Cardiac workup

- a. EKG
- b. A comprehensive cardiac evaluation should be done in any patient who is considered to have major clinical predictors of increased cardiac risk.

4. Pulmonary workup

- a. Chest X-ray in select patients
 - i. Chronic obstructive pulmonary disease (COPD)
 - ii. Cardiopulmonary pathology

Intra-operative

1. The anesthetic technique chosen should be the best suited for the anesthesiologist, the patient, and the surgeon.
 - a. Regardless if regional or general, the goals are the same.
 - i. Avoid ischemic injury to the brain.
 - ii. Maintain adequate cerebral perfusion.
 - iii. Maintain cardiovascular stability.
 - iv. Provide a rapid anesthetic recovery allowing for a neurologic exam.
 - b. Pick an anesthetic plan and defend it.
 - c. For this particular case, a general anesthetic was chosen. After consultation with the surgeon, it was deemed that a general anesthetic would allow for better surgical exposure in this patient with complex carotid anatomy. The patient as well preferred a general anesthetic due to a previous unpleasant experience with regional anesthesia.
 - d. For other cases of CEA a regional technique might be a preferred method, and the ABA candidate should be prepared to discuss it.
2. Pre-induction arterial catheter for continuous blood pressure monitoring
3. Pre-medication with judicious amounts of intravenous fentanyl and lidocaine to blunt the sympathetic response to direct laryngoscopy
4. Application of the standard ASA monitors
5. Intravenous induction with etomidate for hemodynamic stability
6. Placement of the endotracheal tube
 - a. Monitor the patient closely during laryngoscopy.
 - b. Treat elevations of heart rate or blood pressure with short-acting medications such as esmolol and judicious amounts of opioid.
 - c. Also, be prepared to treat sudden hypotension on induction with bolus dosing of phenylephrine.
7. Maintenance of anesthesia would include isoflurane, based upon its purported cerebral protective effects, and a remifentanyl infusion, based on its short half-life which allows for a rapid anesthetic emergence.

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a. Phenylephrine and nitroglycerin infusions should be prepared and available in advance for administration in the event of significant hemodynamic changes as needed.

8. Cerebral monitoring

a. Carotid stump pressure (CSP)

- i. Simple technique
- ii. Inexpensive
- iii. Commonly used

b. Processed EEG (BIS): especially if an EEG is not available or practical

- i. Ease of use
- ii. Readily available
- iii. Interpret cautiously, as it is not validated for this particular indication and may not reliably detect cerebral ischemia.

TKO: Be prepared to discuss all of the techniques of cerebral monitoring for CEA.

Post-operative

1. The patient should be monitored closely in either the intensive care unit or on a telemetry floor.

- a. CEA surgery is associated with significant hemodynamic lability for the first 24 hours.

BIBLIOGRAPHY

1. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleishmann KE, Freeman WK, Froehlich JB, Kasper E, Kersten JR, Riegel B, Robb JF. ACC/AHA 2007 Guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *Circulation* 2007;117:1717-1718.
2. Huncke T., Bekker A. Carotid endarterectomy: anesthetic choices and cerebral monitoring. *Current Reviews in Clinical Anesthesia*. Lesson 20, Volume 28, 5/1/2008, 239.
3. Chaturvedi S, Bruno A, Feasby T, et al. Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Carotid endarterectomy - an evidence-based review: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2005;65(6):794-801.
4. Erickson KM, Cole DJ. Review of developments in anesthesia for carotid endarterectomy. *Current Opinions in Anesthesiology* 2005;18:466-67.
5. Maharaj R. A review of recent developments in the management of carotid artery stenosis. *Journal of Cardiothoracic and Vascular Anesthesia* 2008;22(2):277.
6. Fleischer L. Preoperative cardiac evaluation before major vascular surgery. In Kaplan JA, Lake CL, Murray MJ, eds., *Vascular Anesthesia*, 2nd ed. Philadelphia: Elsevier, Churchill Livingstone, 2004, pp 93-105.
7. O'Connor CJ, Tuman KJ. Anesthetic considerations for carotid artery surgery. In Kaplan JA, Lake CL, Murray MJ, eds., *Vascular Anesthesia*, 2nd ed. Philadelphia: Elsevier, Churchill Livingstone, 2004, pp 187-98.
8. Ferguson GG, Eliasziw M, Barr HW, et al. The North American Symptomatic Carotid Endarterectomy Trial: surgical results in 1415 patients. *Stroke* 1999; 30: 1751- 8.
9. Hobson RW II, Weiss DG, Fields WS, Goldstone J, Moore WS, Towne JB, Wright CB. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. The Veterans Affairs Cooperative Study Group. *N Engl J Med* 1993;328(4):221-7.
10. The European Carotid Surgery Trialists Collaborative Group. Endarterectomy for moderate symptomatic carotid stenosis: interim results from the MRC European Carotid Surgery Trial. *Lancet* 1996;347:1591-3.
11. Allain R, Marone LK, Meltzer J, Jeyabalan G. Carotid endarterectomy. 1: *Int Anesthesiol Clin* 2005;43(1):15-38.
12. Pandit JJ, Satya-Krishna R, Gratton P. Superficial or deep cervical plexus block for carotid endarterectomy: a systematic review of complications. *Br J Anaesth* 2007;99(2): 159-69.
13. Ivanec Z, Mazul-Sunkol B, Lovricevi I, Sonicki Z, Gvozdenovi A, Klican K, Krolo H, Halapir T, Novotny Z. Superficial versus combined (deep and superficial) cervical plexus block for carotid endarterectomy. *Acta Clin Croat* 2008;47(2):81-6.
14. Kim MS, Cole, DJ. Anesthesia for carotid surgery In Schwartz AJ, Matjasko MJ, Otto CW, American Society of Anesthesiologists Inc., *Refresher Courses in Anesthesiology*, vol. 31, chap.21. Philadelphia. Lippincott Williams & Wilkins, 2003, pp 91-103.
15. Yastrebov K. Intraoperative management: carotid endarterectomies. *Anesthesiol Clin North America* 2004;22(2):265-87.
16. Hadzic A, ed. The New York School of Regional Anesthesia. *Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill Medical, 2007, pp 387-95.
17. Chelly JE, ed. *Peripheral Nerve Blocks: A Color Atlas*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2009, pp 162-8.
18. Barrett J, Harmon D, Loughnane F, Finucane B, Shorten G. *Peripheral Nerve Blocks and Peri-operative Pain Relief*. London. Saunders Elsevier, 2004, pp 51-4.
19. Rerkasem K, Rothwell PM. Local versus general anaesthesia for carotid endarterectomy. *Cochrane Database of Systematic Reviews* 2008, Issue 4. Art.No. CD000126. DOI: 10.1002/14651858.CD000126.pub3. Accessed online 11.29.08.
20. Farhoomand L, Berger J, Lehfeldt S. Controversies in anesthesia for carotid endarterectomy: general versus regional anesthesia. *Seminars in Anesthesia, Perioperative Medicine and Pain* 2004;23(30):244-7.

39. Mediastinal Mass^{1,2,3,4,5}

Maureen S. Harders, MD

SAMPLE CASE

A 28-year-old male presents to his primary care doctor with weight loss and a nonproductive cough. He is diagnosed with a mediastinal mass. He presents to the operating room (OR) for a diagnostic mediastinoscopy. What are your concerns? How will you induce anesthesia?

CLINICAL ISSUES

Definition

- The mediastinum contains all of the organs in the chest except for the lungs.
 - Heart
 - Aorta
 - Thymus
 - Trachea
 - Esophagus
 - Nerves
 - Lymph nodes
 - Superior and inferior vena cava
- The mediastinum is divided into three parts.
 - The anterior portion is bordered by the sternum anteriorly and the pericardium posteriorly.
 - The middle mediastinum is defined as the pericardium and all of its contents.
 - The posterior mediastinum is defined as the area posterior to the pericardial wall and anterior to the thoracic vertebral bodies.
- Masses in the anterior mediastinum are more likely to be malignant.
- The four most common masses are the thymoma, (terrible) lymphoma, teratoma, and the ectopic thyroid (the 4 Ts).
- Myasthenia gravis is present in 25 % of the patients diagnosed with a mediastinal mass. Consider this in your anesthetic plan.

Mediastinoscopy

- Indications
 - Visualization of the contents of the mediastinum
 - To assess the spread of bronchial carcinoma
 - Staging of lymph nodes
 - Diagnosis of sarcoidosis
 - Diagnosis of lymphoma

- Contraindications (relative)
 - Previous mediastinoscopy (because of scarring)
 - Superior vena cava (SVC) syndrome
 - Thoracic aortic aneurysm
 - Cerebrovascular disease: occlusion of the innominate artery can cause cerebral ischemia in patients with already compromised cerebral blood flow.
 - Tracheal deviation
- Complications
 - Hemorrhage
 - Pneumothorax
 - Infection
 - Recurrent laryngeal nerve injury
 - Pressure on the right innominate (brachiocephalic) artery can cause decreased cerebral perfusion as well as decreased blood flow to the right upper extremity.
 - Chylothorax
 - Hemothorax

Differential Diagnosis of Increased Airway Resistance

- Bronchospasm
- Foreign body
- Tension pneumothorax
- Endobronchial intubation
- Poor compliance secondary to lung disease
- Mediastinal mass



KO TREATMENT PLAN

Pre-operative

- Review the patient's symptoms.
 - Dyspnea with positional changes
 - Shortness of breath in the supine position
 - Hoarse voice or noisy breathing
 - Dysphagia
 - Signs of superior vena cava (SVC) obstruction
 - Cerebral edema
 - Headache
 - Mental status change
 - Confusion
 - Coma
 - Facial cyanosis and flushing

39. Mediastinal Mass

- iii. Venous distension in the neck
- iv. Venous distension of the upper arms and chest
- v. Upper limb edema
- vi. Lightheadedness
- vii. Upper airway edema
 - (1) Cough
 - (2) Dyspnea
 - (3) Dysphagia
 - (4) Stridor

2. Review the pertinent studies.
 - a. CT scan: look for tracheal or main stem bronchial compression.
 - b. Flow volume loops: collapse during expiration is a sign of intrathoracic compression.
 - c. Echocardiogram: look for right ventricular (RV) outflow obstruction and/or right heart dysfunction.
3. If there is severe obstruction of the airway or right ventricular outflow tract (RVOT), discuss with the surgeon the possibility of performing the mediastinoscopy under local anesthesia. This will help avoid the possibility of further compression on the RVOT which could lead to cardiovascular collapse.
4. Remember that respiratory complications in the peri-operative period may occur even though pre-operative studies would not have predicted difficulties.

Intra-operative

1. Monitors and intravenous (IV) access
 - a. A large-bore IV is needed. Consider a lower extremity IV if there is significant compression on the SVC.
 - b. A right radial arterial line is very sensitive and will dampen with pressure of the mediastinoscope on the innominate artery.
 - c. The pulse oximeter waveform can also be used to help alert the anesthesiologist to pressure being placed on the innominate artery but is not quite as sensitive or as reliable as the arterial line.
 - d. Consider bilateral femoral lines if there is a possibility of complete airway obstruction and the need to start cardiopulmonary bypass.
 - e. Have two units of cross-matched blood available. Remember that the mediastinum is full of large blood vessels.
2. Airway management
 - a. The goal is to not give up the airway.
 - b. Keep the patient breathing spontaneously. Spontaneous respirations help maintain a normal transpulmonary pressure gradient and help distend the airway.

- c. Avoid muscle relaxation since it can diminish airway tone. This may be critical to the patient for maintaining his airway.
 - d. Awake fiber optic intubation with a reinforced endotracheal tube should be the first option for a patient who has an obstruction.
 - e. An inhalational induction with sevoflurane in the sitting position is also a consideration. Remember that you may lose the airway as the induction proceeds, and you must be prepared to deal with that complication.
3. Lost airway maneuvers
 - a. Change the patient's position to lateral or prone.
 - b. Reverse the muscle relaxant.
 - c. Try to get the patient breathing spontaneously.
 - d. Use a rigid bronchoscope to push past the obstruction.
 4. Maintaining spontaneous respirations will not only aid in maintaining the airway but it is also important in patients with RVOT obstruction, since compression of the RVOT can lead to cardiovascular collapse.
 - a. Intermittent positive pressure ventilation (IPPV) may decrease the cardiac output.
 - b. The supine position also may aggravate the RV outflow obstruction.

Post-operative

1. Extubation
 - a. The patient needs to be watched closely. He may develop a complication that requires reintubation due to increased airway obstruction from swelling or impaired respiratory muscle function.
 - b. Respiratory complications including airway obstruction occur with a higher incidence in patients with greater than 50% compression of the trachea in pre-operative exams.
2. Patients who remain intubated may need pressure control ventilation to maintain an open airway.

BIBLIOGRAPHY

1. Brodsky JB. Thoracic anesthesia. *Problems in Anesthesia*, vol. 4, no. 2. Philadelphia: Lippincott, 1990, pp 334-8.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1920-3.
3. Bechard P, Letourneau L, Laacasse Y, et al. Perioperative cardiorespiratory complications in adults with mediastinal mass: incidence and risk factors. *Anesthesiology* 2004;100(4): 826-34.
4. Pullerits J, Holzman R. Anaesthesia for patients with mediastinal masses. *Canadian J Anesthesia* 1989;36:681-8.
5. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 840-2.

40. Treatment of Elevated Intracranial Pressure (ICP)^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 19-year-old male presents to the emergency room after falling two stories off of a balcony. He is confused and agitated. His blood pressure is 198/99 mm Hg and his heart rate is 110. The emergency medical services (EMS) reports that the patient was vomiting during his transport to the hospital. His pupils are dilated. How do you determine whether the patient's ICP is increased? Why is it important to know? If elevated, what steps would you take to reduce it?

The neurosurgeon determines that the patient requires an emergent surgery. Immediately after opening the skull, the surgeon says that the dura is taut. How will you treat this? The surgeon says the brain is bulging. Now what would you do?

CLINICAL ISSUES

ICP

1. The normal ICP wave is pulsatile and varies with spontaneous respiration.
2. The mean ICP should remain below 15 mm Hg.
3. Treatment of elevated ICP should begin once it reaches values of greater than 20–25 mm Hg. Higher values may be tolerated if the cerebral perfusion pressure (CPP) is adequate.
4. $CPP = \text{mean arterial pressure (MAP)} - \text{ICP}$ or central venous pressure (CVP) (whichever is greater).

Signs and Symptoms of Elevated ICP

1. Altered and/or loss of consciousness
2. Dilated or non-reactive pupils
3. Flexor or extensor posturing
4. Nausea and vomiting
5. Headache

Causes of Elevated ICP

1. Anxiety
2. Painful stimulation
3. Induction of anesthesia
4. Increase in cerebral spinal fluid (CSF) volume because of a blockage of the circulation or absorption.
5. Increase in blood volume from vasodilation or a hematoma.

6. Increased brain tissue volume secondary to a tumor or edema.

Complications Associated with an Elevated ICP

1. Reduced blood flow to the brain.
2. Brain herniation across the meninges, down the spinal canal, or through an opening in the skull. This herniation can lead to neurologic deterioration and death.

Anesthetic Effects on Brain Physiology

1. Volatile anesthetics
 - a. Direct vasodilatory effects that increase cerebral blood flow (CBF) and can lead to an increased ICP under conditions of abnormal intracranial elastance.
 - b. Decreased cerebral metabolic rate (CMR)
2. Intravenous anesthetics
 - a. Propofol
 - i. Preferred over volatile anesthetics because it does not have a direct vasodilatory effect; therefore, it does not increase the ICP.
 - ii. Decreases CMR.
 - iii. Decreases CBF.
 - iv. Decreases ICP.
 - v. Can decrease the MAP, leading to a decrease in CPP. Doses must be titrated carefully.
 - b. Etomidate
 - i. Decreases CMR.
 - ii. Decreases CBF.
 - iii. Direct vasoconstrictor
 - iv. It does not produce clinically significant cardiovascular depression.
 - v. Likely decreases ICP.
 - vi. Considered the induction agent of choice
 - c. Benzodiazepines (midazolam)
 - i. Decrease CMR.
 - ii. Decrease CBF.
 - iii. May decrease the ICP.
 - d. Opioids (fentanyl and remifentanyl)
 - i. Minor reduction or no effect on CBF and CMR
 - e. Barbiturates (thiopental)
 - i. Decrease CMR.
 - ii. Decrease CBF.
 - iii. Decrease ICP.

40. Treatment of Elevated Intracranial Pressure

- iv. Can decrease the MAP leading to a decrease in CPP. Doses must be titrated carefully.
- v. At high doses (10–55 mg/kg), thiopental can produce an isoelectric EEG and decrease CMR by 50%.⁴
- vi. Decrease seizure activity.

Drugs to Avoid

1. Etomidate
 - a. The standard propylene glycol formulation may induce cerebral tissue hypoxia, tissue acidosis, and neurological deficits. Therefore, it has some theoretical concerns. Currently, still considered the induction agent of choice.
2. Nitrous oxide given alone
 - a. NMDA receptor antagonist
 - b. Possible direct neurotoxic effects
 - c. Increases CMR.
 - d. Increases CBF.
 - e. Increases ICP.
 - f. Disturbs CBF/CMR coupling in humans receiving sevoflurane.⁴
 - g. The increased CBF and ICP can be blunted by administering barbiturates, benzodiazepines, and morphine.
3. Flumazenil
 - a. Benzodiazepine antagonist
 - b. Give with caution, if at all, in patients with an elevated ICP secondary to the reversal of the CMR, CBF, and ICP lowering effects of benzodiazepines.
4. Ketamine
 - a. Increases CBF and CMR.



KO TREATMENT PLAN

Pre-operative

Indications for ICP monitoring

1. Patients with a severe head injury defined by the Glasgow coma scale (GCS) as less than 8.
2. Abnormal head CT findings including hematoma, contusions, edema, or compressed basal cisterns should be managed with the aid of an ICP monitor.
3. Severe traumatic brain injury, even with a normal head CT, if the patient has any of the following conditions:
 - a. Age greater than 40 years
 - b. Motor posturing
 - c. Systolic blood pressure less than 90 mm Hg

Ventriculostomy

1. Compared to intraparenchymal monitoring, a ventriculostomy is considered the most safe, accurate, and cost-effective method to monitor ICP.
2. A ventriculostomy also has diagnostic and therapeutic capabilities.
3. Complications such as infection and hemorrhage are relatively infrequent.

Plan for Treatment of Elevated ICP

Step one

1. Positional therapy
 - a. Allows for venous drainage and the movement of CSF from the cranium to the spinal canal.
 - i. Place the patient in a slight head up or neutral position.
 - ii. The patient's head should be about 30 degrees above heart level.
 - iii. Check that the cervical collar or tracheotomy tie is not too tight.
 - b. This sitting position also allows for improved ventilation-perfusion matching and therefore improved cerebral oxygen delivery.
2. Support hemodynamics
 - a. Systolic blood pressure should be higher than 110 mm Hg with a goal MAP greater than 90 mm Hg.
 - b. The CPP should be at a minimum of 70 mm Hg.
 - c. The blood pressure should be maintained until direct ICP monitoring can be instituted and the CPP directly targeted.
3. Analgesia and sedation
 - a. Adequate sedation and pain control are important secondary to the resulting increase in ICP from agitation.
 - b. Intravenous esmolol, narcotics, and lidocaine can be used to blunt the sympathetic response to direct laryngoscopy and other painful stimuli.
 - c. Be aware that oversedation in the post-operative period may impede sequential neurological assessments.
 - d. A short-acting agent such as propofol may be preferable.
 - i. The use of propofol usually requires the subsequent use of a vasoactive drug to maintain the mean arterial pressure (MAP) and, in turn, the cerebral perfusion pressure (CPP).
 - e. Intravenous barbiturates and benzodiazepines can also be used.
4. Avoid hypoxemia ($\text{PaO}_2 < 60$ mm Hg).
 - a. Maintain adequate oxygenation and ventilation.
 - b. Positive end-expiratory pressure (PEEP) was once believed to increase the ICP, but it may actually decrease the ICP by improving cerebral oxygenation.
5. Hyperventilation
 - a. Appropriate treatment only if the patient has imminent signs of cerebral herniation.
 - b. The current guidelines recommend maintenance of PaCO_2 at about 30–35 mm Hg, with a hyperventilation to less than 30 mm Hg only for episodes of elevated ICP that cannot be controlled by other means.
 - c. Recent studies have shown that the hyperventilation-induced vasoconstriction, which leads to the initial decrease in ICP, will after long-term use result in hypoxic injury and possible stroke.
6. The goal hematocrit should be greater than 30%.
7. Patients should be normothermic.

40. Treatment of Elevated Intracranial Pressure

- a. Hypothermia: if the patient presents to the hospital in a hypothermic state, active and aggressive rewarming should be avoided secondary to an increase in observed mortality. Special care should be used to avoid shivering.
- b. Hyperthermia: hyperthermia should be treated aggressively, since the increase in CMR has been shown to worsen the neurologic exam.

Step Two

1. Drainage of cerebrospinal fluid (CSF) per the surgeon.
 - a. This decreases the intracranial volume and the ICP.
 - b. Drainage of CSF is obtained from the lateral cerebral ventricles or the lumbar subarachnoid space.
 - c. Lumbar drainage of CSF is usually not recommended secondary to the risk of cerebellar herniation.

Step Three

1. Osmotic therapy
 - a. Mannitol
 - i. The dose is 0.25–1.0 g/kg IV over 15–30 minutes. This dose usually results in a 100 ml removal of water from the patient's brain and a decrease in the ICP within 30 minutes. The maximum effect is seen within 1–2 hours.
 - ii. Urine output can exceed 1–2 liters within an hour. Infusion of a crystalloid solution is necessary to replace the intravascular fluid loss.
 - iii. Reduces ICP by its actions as an osmotic diuretic and cerebral arteriolar vasoconstrictor.
 - iv. Helps to pull the excess interstitial fluid into the vascular space and therefore lower the ICP.
 - v. Mannitol is associated with rebound intracranial hypertension.
 - vi. It is contraindicated in patients who are not adequately volume resuscitated.
 - b. Furosemide
 - i. Useful in a patient with increased vascular fluid and pulmonary edema
 - ii. Dose: 1mg/kg IV
 - c. Hypertonic saline (NaCl 3% to 5%)
2. Decrease intravenous fluids.

Step Four

1. Barbiturate coma
 - a. This is used in patients with intractable elevations in ICP.
 - b. The barbiturate coma lowers the CMR and results in a decrease in excitatory neurotransmitter release.
 - c. Considered only when CPP can not be maintained with previously described therapies. It usually requires pulmonary artery catheterization and vasoactive agents.
 - d. High dose barbiturates are especially effective for treating elevated ICP after an acute head injury.
2. Neuromuscular blockade: these drugs may be necessary for a temporary treatment of an elevated ICP, but they are not recommended for long-term use secondary to their interference with the neurologic exam.

3. Corticosteroids

- a. Effective for patients with elevated ICP secondary to localized cerebral edema around a brain tumor, particularly metastatic tumors and glioblastomas.

Step Five

1. Decompressive craniectomy: a surgical procedure that is used to control severely elevated ICP and prevent herniation and stroke.
2. Decompressive laparotomy: indicated in patients with coexisting injuries or a vigorous volume infusion in which the patient develops increased intra-abdominal pressure to greater than 20 mm Hg. This increase in abdominal pressure worsens the pulmonary mechanics and therefore requires a higher mean arterial pressure (MAP) to maintain arterial oxygen saturation. The increase in ventilating pressure leads to an increase in intra-thoracic pressure and impaired venous drainage from the head, therefore leading to an increased ICP if not treated.

Step Six

1. Avoid ketamine, etomidate, nitrous oxide, hypotonic crystalloid solutions, and glucose-containing solutions.

Intra-operative

1. Transport of the patient should be kept to a minimum.
2. Surgical interventions should be for life- or limb-threatening procedures only.
3. Urgent surgeries – for example, the fixation of a long bone – should only be performed once the CPP is being successfully managed.
4. Continue all of the above therapies during the surgery, including positional therapy when possible, aggressive hemodynamic monitoring and resuscitation, administration of osmotic agents, and deep levels of anesthesia and sedation.
5. Induction of anesthesia is usually performed with a rapid sequence intubation (RSI).
 - a. Blunt the sympathetic response to laryngoscopy with intravenous fentanyl (3 mcg/kg) and/or intravenous lidocaine (1.5 mg/kg).
 - b. Pretreatment with 0.01 mg/kg of vecuronium 3 minutes prior to giving succinylcholine helps to prevent the associated increase in ICP.
 - c. Etomidate is the induction agent of choice secondary to its hemodynamic stability and decreasing effect on ICP.
 - d. Thiopental can be used. It has cerebroprotective effects but can cause hypotension, so it should be titrated carefully.
6. Maintenance anesthesia should include narcotics and low concentrations of volatile anesthetics. Avoid nitrous oxide.
7. Treat a taut dura or a bulging brain with IV boluses of propofol, barbiturates, benzodiazepines, and/or mannitol/furosemide. Also consider positional therapy, hyperventilation, increased neuromuscular blockade, and drainage of the CSF. Verify normothermia and proper oxygenation.

Continued communication with the surgeon is extremely important in this situation.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2469–74.
2. Rakel RE, Bope ET. *Conn's Current Therapy*. Philadelphia: Saunders, 2008, chapter 240.
3. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 236–8.
4. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 750–8.

41. Somatosensory-Evoked Potentials (SSEP) and Motor-Evoked Potentials (MEP)^{1–15}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 47-year-old male is scheduled for a surgical repair of his cervical spine. He was in a high-speed motor vehicle accident 1 week ago and has been in a cervical collar since. Does this patient require SSEP and/or MEP monitoring? Why? If SSEP/MEP monitoring is used, how will this affect your anesthesia? Would a wake up test be better than SSEP/MEP monitoring?

CLINICAL ISSUES

Evoked Potentials

1. Used intra-operatively to monitor the integrity of a specific sensory or motor pathway.

General Treatment Issues

1. Communication between the anesthesia team, neuromonitoring clinician, and surgeon is critical. The neuromonitoring clinician should be informed prior to any significant changes in the administration of anesthesia during the case (i.e., any bolus of medication or nitrous oxide [N₂O]/volatile anesthetic change). The goal should be to make no major anesthetic changes after the induction of anesthesia and the baseline neuromonitoring measurements.
2. Changes in levels of inhaled anesthetics, temperature, CO₂, and blood pressure affect interpretations of SSEP/MEP monitoring. Therefore, these levels should be kept as constant as possible during the case. In the management of patient hemodynamics, it is preferred that boluses of medications are avoided. Adjusting the infusion rates of the intravenous medications is preferred. Boluses of β-blockers, hydralazine, phenylephrine, and ephedrine are preferred if a bolus is required.

3. Compatibility of a given anesthetic protocol with SSEP or MEP monitoring is not all-or-nothing; it is a continuum. It depends on multiple factors, and the same anesthetic protocol will not work with all patients.
4. Each patient should have baseline SSEP/MEP measurements once anesthesia is induced and the patient is positioned.
5. The anesthesia and surgical teams must provide full disclosure of the risks and benefits of the neuromonitoring and anesthesia, including but not limited to the possibility of awareness and intra-operative movement.

SSEP

CLINICAL ISSUES

Definition

1. SSEPs evaluate the functional integrity of the ascending sensory pathways.
2. SSEPs are recorded after electrical stimulation of a peripheral mixed nerve.
3. The stimulation is usually with a surface electrode or a fine-needle electrode.
4. Nerves commonly stimulated include the median nerve at the wrist, the common peroneal nerve at the knee, and the posterior tibial nerve at the ankle.
5. Compromise or injury to the pathway manifests as an increase in the latency and/or a decrease in the amplitude of the evoked potential waveform.
6. A 50% reduction in amplitude is considered to be significant and warrants a change in action, either by anesthesia or the surgeon.

41. Evoked Potential Monitoring

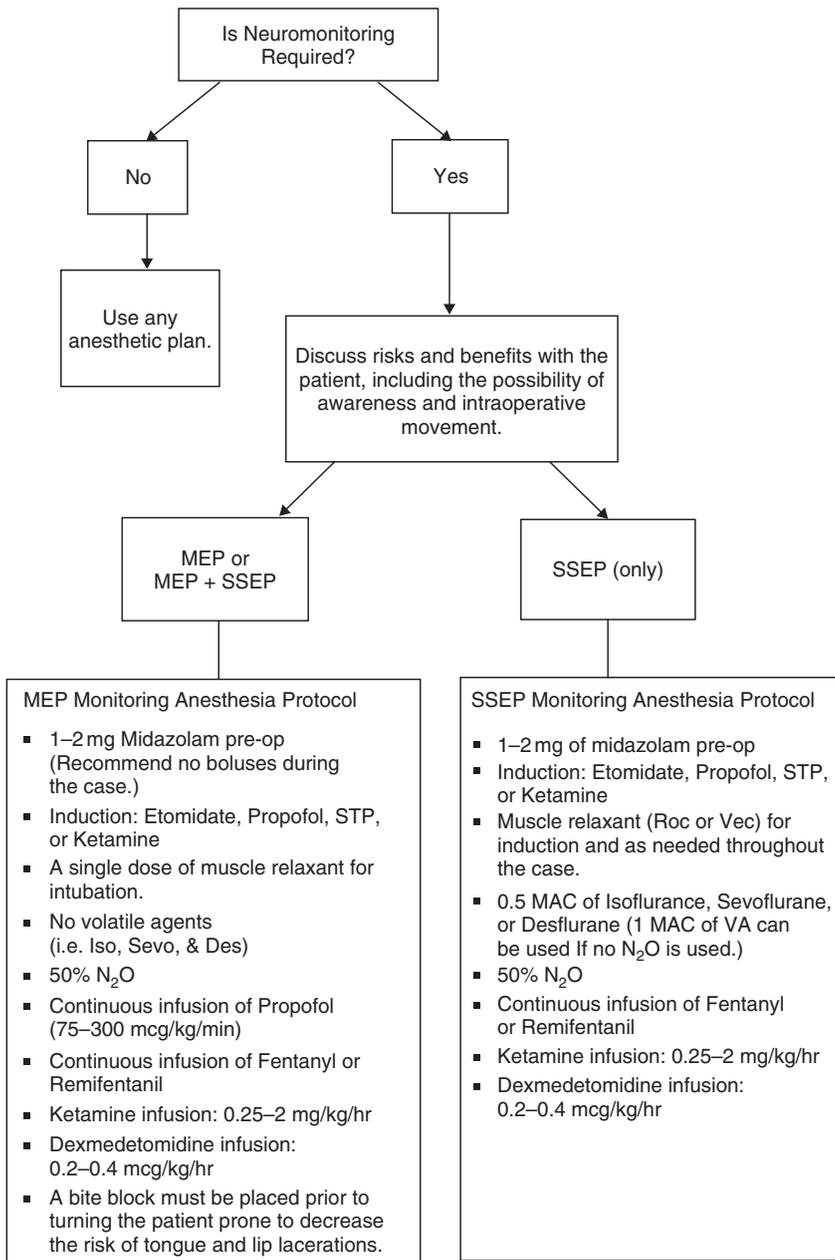


Figure 41.1. SSEP and MEP monitoring. Source: Lovich-Sapola JA, Steele E. *SSEP and MEP Monitoring Anesthesia Protocol*. Metro Health Medical Center, Cleveland, OH, 2005 and 2009.

7. It is important to limit outside factors that can also affect the waveform.

- Maintain a constant level of the anesthetic drug.
- Avoid boluses of anesthetics and significant changes in the inhaled anesthetic.

Indications

- Intramedullary/extramedullary spinal cord tumors or cysts
- Vascular lesions in the spine
 - Arterial-venous malformations
 - Arterial-venous fistulas
- Cervical or thoracic spine herniated discs that cause spinal cord compression

4. Spinal cord decompression and stabilization after an acute spinal cord injury

5. Spinal fusion

6. Cervical spondylosis

7. Ossification of the posterior longitudinal ligament

8. Scoliosis correction

9. Brachial plexus exploration after injury

10. Resection of a fourth ventricular cyst

11. Release of a tethered spinal cord

12. Clipping of an intracranial aneurysm

13. Carotid endarterectomy

14. Resection of a thalamic tumor

15. Abdominal and thoracic aneurysm repair

16. Repair of coarctation of the aorta



KO TREATMENT PLAN SSEP

Intra-operative

1. Benzodiazepines produce minimal changes on the SSEP waveform when given in low doses.
 - a. 1–2 mg of midazolam IV can safely be given in the pre-operative period.
 - b. At high doses, diazepam can cause an increase in latency and a decrease in amplitude.
 - c. Midazolam causes a decrease in amplitude, with no change in latency, at escalating doses.
2. IV induction doses of thiopental, propofol, ketamine, or etomidate do not tend to affect the SSEP waveform recordings.
 - a. Propofol and thiopental can have an effect when given at high doses.
 - b. An increasing dose of thiopental results in a progressive dose-dependent increase in latency and decrease in amplitude.
 - c. Etomidate actually causes an increase in the latency and amplitude.
3. High concentrations of volatile agents essentially eliminate the evoked potential. They cause a dose-dependent increase in latency and a decrease in amplitude.
 - a. Up to 0.5 MAC of isoflurane, sevoflurane, or desflurane is acceptable.
 - b. One MAC of isoflurane, sevoflurane, or desflurane can be used if no N₂O is used.
4. N₂O produces a profound depressant effect on the SSEPs, especially when used in combination with volatile anesthetics. It causes a decrease in amplitude with minimal effects on the latency.
 - a. Up to 50% N₂O is usually acceptable.
5. Opioids produce minimal changes in the SSEP waveform even in high doses.
 - a. Continuous infusion of fentanyl or remifentanyl is recommended.
 - b. Boluses of opioids should still be avoided during critical times when neurologic injury may occur.
6. Muscle relaxant (i.e., rocuronium or vecuronium) can be used for induction and as needed throughout the case.
7. Clonidine and dexmedetomidine are α₂-receptor agonists that can be used to decrease anesthetic requirements. Both agents can be used without compromising SSEP monitoring.
 - a. Recommended dexmedetomidine infusion rate: 0.2–0.4 mcg/kg/hr
8. Ketamine increases the cortical SSEP amplitude.
 - a. Recommended infusion rate of 0.25–2 mg/kg/hr.
9. Physiologic factors such as temperature, blood pressure, PaO₂, and PaCO₂ can alter the SSEPs. The goal is to maintain a fairly constant value without any major fluctuations.
 - a. Maintain normothermia. Hypo- and hyperthermia both can affect the SSEP monitoring.

- b. Hypotension less than the cerebral autoregulation set point produces a progressive decrease in amplitude on the SSEPs.
- c. Hypoxia produces SSEP changes including a decrease in amplitude.
- d. Anemia results in increased latency of the SSEP waveform.

MEP

CLINICAL ISSUES

Definition

1. MEPs test the integrity of the descending motor pathway.
2. Eliminate the need for a “wake up test.”
 - a. A “wake up test” is when you lighten the anesthesia of a patient during the surgery and ask him or her to move the extremities to evaluate for any spinal cord damage. This can be dangerous, especially if the patient is in the prone position. It also carries a higher risk of intra-operative awareness, which can be very disturbing for many patients.
3. MEPs should universally be combined with SSEP monitoring.

Indications

1. Intramedullary spinal cord tumors
2. Vascular lesions in the spine
 - a. Arterial-venous malformations
 - b. Arterial-venous fistulas
3. Cervical or thoracic spine herniated discs that cause spinal cord compression
4. Scoliosis surgery
5. Removal of cerebral tumors involving the motor cortex or subcortical motor pathways
6. Aortic reconstruction

Contraindications to Transcranial MEP

1. Patients with a history of seizure
2. Skull fracture
3. Implanted metal devices
 - a. Cardiac pacemakers
 - b. Central venous or pulmonary artery catheters



KO TREATMENT PLAN MEP

Intra-operative

1. Small 1–2 mg IV boluses of midazolam can be safely given in the pre-operative period.

41. Evoked Potential Monitoring

2. Induction with IV propofol, thiopental, etomidate, or ketamine can be done safely.
3. Volatile anesthetics have significant depressant effects on the MEP waveform. No volatile anesthetic agents should be used. (i.e., isoflurane, sevoflurane, or desflurane)
4. Nitrous oxide causes less suppression of the MEP waveforms than volatile anesthetics.
 - a. 50% N₂O
5. Continuous infusion of propofol (75–300 mcg/kg/min)
6. Continuous infusion of fentanyl or remifentanyl
7. A single dose of muscle relaxant can be used for the intubation but should not be repeated.
 - a. Some sources say that a low-dose infusion of IV muscle relaxants, maintaining one or two twitches in a train of four, can produce a reliable MEP waveform. This is very department specific since there is no set protocol in the literature.
8. A bite block must be in place prior to turning the patient prone to decrease the risk of tongue and lip laceration.
9. Ketamine infusion can be used at a recommended rate of 0.25–2 mg/kg/hr.
10. Dexmedetomidine infusion can be used at a recommended rate of 0.2–0.4 mcg/kg/hr.
11. Avoid hypothermia, hypoxia, and hypotension.

BIBLIOGRAPHY

1. Lovich-Sapola JA, Steele E. *SSEP and MEP Monitoring Anesthesia Protocol*. MetroHealth Medical Center, Cleveland, OH, 2005 and 2009.
2. The Los Angeles Anesthesia Friendliness Scale. <http://www.neuromon.com/neuro/lafs.html> 24 Oct 2005.
3. Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials. *Anesthesiology*. 99:3 Sep 2003. pp 716–37.
4. Calancie B, Harris W, Broton JG et al. “Threshold-level” multipulse transcranial electrical stimulation of motor cortex for intraoperative monitoring of spinal motor tracts: description of method and comparison to SSEP monitoring. *J Neurosurg*. 90:2 Feb 1999. p 376.
5. Clapcich AJ, Emmerson, RG, Roye DP. Propofol/remifentanyl is superior to nitrous oxide/isoflurane/remifentanyl for cortical somatosensory evoked potentials assessment in children undergoing spinal surgery. *Anesthesiology*. 96:2002. p A306.
6. Clapcich AJ, Emmerson, RG, Roye DP et al. The effects of propofol, small-dose isoflurane, and nitrous oxide on cortical somatosensory evoked potential and bispectral index monitoring in adolescents undergoing spinal fusion. *Anesthesia & Analgesia*. 99:5 1 Nov 2004. pp 1334–40.
7. van Dongen EP, ter Beek HT, Schepens MA et al. Within-patient variability of myogenic MEP’s to multipulse transcranial electrical stimulation during two levels of partial neuromuscular blockade in aortic surgery. *Anesthesia & Analgesia*. 88:1 Jan 1999. pp 22–7.
8. Fletcher JE, Hinn AR, Heard CM et al. The effects of isoflurane and desflurane titrated to a bispectral index of 60 on the cortical somatosensory evoked potential during pediatric scoliosis surgery. *Anesthesia & Analgesia*. 100:6 1 Jun 2005. pp 1797–803.
9. Haghghi SS. Influence of isoflurane anesthesia on motor evoked potentials elicited by transcortical, brainstem, and spinal root stimulation. *Neurol Res*. 20:6 Sep 1998. pp 555–8.
10. Lyon R, Feiner J, Lieberman J. Progressive suppression of motor evoked potentials during general anesthesia: the phenomenon of “anesthetic fade.” *J Neurosurg Anesthesiol*. 17:1 Jan 2005. pp 13–9.
11. Miller RD, Cucchiara RF, Millere Ed, et al. *Anesthesia*, 5th ed. Philadelphia: Churchill Livingstone, 2000, pp 1335–45.
12. Strahm C, Min K, Boos N et al. Reliability of perioperative SSEP recordings in spine surgery. *Spinal Cord*. 41:9 Sep 2003. pp 483–9.
13. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1525–30, 1532–7, 1539–40.
14. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 760–3.
15. Bal E, Sessler DI, Nair DR, et al. Motor and sensory evoked potentials are well maintained in patients given dexmedetomidine during spine surgery. *Anesthesiology*. 2008; 109:417–25.

42. Posterior Fossa Craniotomy^{1,2,3,4}

Joni Maga, MD

SAMPLE CASE

A 5-year-old boy presents with a 1 week history of headaches, visual changes, and uncoordination. A large infratentorial mass is discovered on MRI. He is scheduled for craniotomy and resection of the tumor. What are your specific concerns: pre-operative, intra-operative, and post-operative? How will you induce anesthesia? What are your concerns about the patient's intra-operative positioning? Will you extubate the patient at the end of the case?

CLINICAL ISSUES

Non-expandible Space

1. The posterior fossa contains the medulla, pons, cerebellum, motor and sensory pathways, respiratory and cardiovascular centers, and cranial nerve nuclei.
2. Mass effect from tumors, bleeding, and edema can cause profound neurological damage leading to obstructive hydrocephalus and brainstem compression.
 - a. Obstructive hydrocephalus leads to increased ICP, which results in mental status changes, visual changes, headaches, nausea, and vomiting.
 - b. Brainstem compression leads to changes in the level of consciousness, depressed respirations, cardiac dysrhythmias, and cranial nerve palsies.
3. It is not uncommon that a patient will require intubation pre-operatively for mental status changes and airway protection.

Patient Positioning

1. Sitting
2. Horizontal
 - a. Lateral
 - b. Park bench
 - c. $\frac{3}{4}$ lateral
 - d. Prone
3. There are risks and benefits to each.

Sitting Position

1. Risks
 - a. Venous air embolism (VAE): one of the most concerning risks of the sitting position is the increased incidence (45%)

of VAE and the associated paradoxical air embolism (PAE). Both VAE and PAE will be addressed in a separate chapter (Chapter 43).

b. Hypotension

- i. Decreased venous return: there is decreased venous return resulting in a decrease in cardiac output and cerebral perfusion pressure (CPP).

- ii. Impaired regulatory mechanisms: general anesthesia can impair baroreceptor (carotid sinus and aortic arch) and renin-angiotensin-aldosterone compensatory mechanisms. This lack of compensation is exaggerated in the elderly.

- iii. Decreased systemic vascular resistance (SVR): anesthetic agents and positive pressure ventilation can directly decrease SVR, which is more pronounced in the sitting position.

- iv. Arterial blood pressure monitoring is necessary not only to monitor cardiovascular changes but also to gauge CPP.

- (1) Keep in mind that accurate CPP monitoring can be obtained with the transducer at the level of the head

- (a) Obtain the mean arterial pressure (MAP) reading with the arterial line transducer at head level, and then subtract the ICP to get the CPP.

- v. The severity of hypotension can be blunted with adequate hydration and gradual positioning.

- (1) Placing the patient in a semi-recumbent (modified sitting) position may also diminish the hypotension.

c. Hyperflexion of the neck

- i. Jugular venous obstruction can result in brain and facial swelling.

- ii. Resulting quadriplegia or paraplegia from compromised blood flow to the cervical cord or direct compression of the cord

- (1) This is more likely in the elderly and those with pre-existing cervical spine disease and is exacerbated by hypotension.

- (2) Head and neck range of motion should be assessed pre-operatively.

- (3) Consider radiographic studies (lateral X-ray, CT scan) to assess the width of the cervical canal.

- iii. Flexion of the head can also cause the endotracheal tube to migrate deeper.

42. Posterior Fossa Craniotomy

(1) Equal breath sounds should be verified after positioning to ensure that the tube is not in the right mainstem.

iv. There should be at least a 2 fingerbreadth distance between the mandible and the sternum to ensure the neck is not over-flexed.

v. Somatosensory-evoked potentials should also be considered if there is concern.

d. **Peripheral nerve injuries: especially for the ulnar, peroneal, and sciatic nerves**

e. **Pneumocephalus:** air enters the cranium through the craniotomy and can get trapped in the supratentorial space. Imagine an inverted soda pop bottle.²

i. It has been advised to discontinue nitrous oxide (which will expand the air volume) before dural closure and to continue to avoid it for two weeks if more surgery is necessary.

ii. Can develop into tension pneumocephalus postoperatively.

(1) When the patient is placed supine post-operatively, the beneficial effects of the sitting position are lost. There is no longer superior venous and cerebral spinal fluid (CSF) drainage. In addition, the effects

of hypocapnea and osmotic diuresis may be lost post-operatively. The brain, blood, and CSF volume increase which, combined with trapped air, can result in tension pneumocephalus.

(2) This should be considered with the patient who has delayed awakening or mental status changes.

2. Benefits

a. **Fewer cranial nerve deficits:** the sitting position provides the best surgical access to the infratentorial fossa requiring less aggressive surgical retraction, which has been reported to result in fewer cranial nerve deficits.³

b. **Edema and hemorrhage:** there is less potential for swelling of the brain and hemorrhage secondary to the superior venous and cerebrospinal fluid drainage of the head aided by gravity.

i. Blood drains away easily from the operative site, which gives surgeons a superior surgical field.

ii. There is less facial and conjunctiva swelling than is seen in patients who have been in the prone position.

c. **Ventilation:** improved in the sitting position due to

i. Increased functional residual capacity (FRC)

ii. Better ventilation perfusion matching

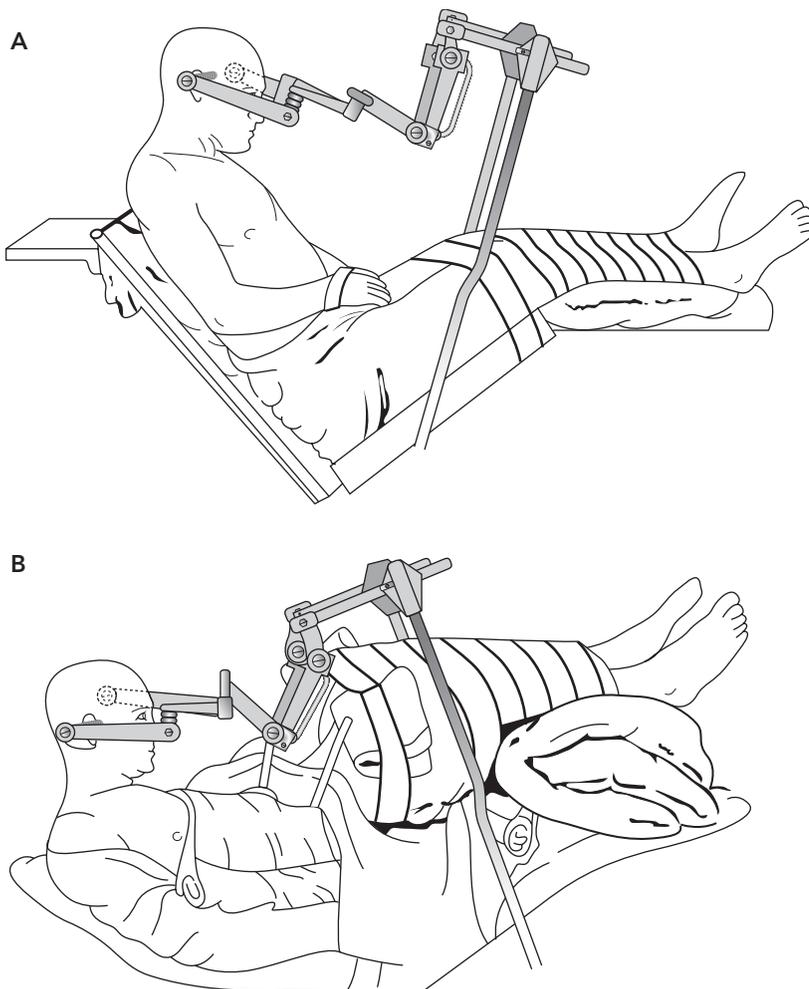


Figure 42.1. Sitting positioning: (A) classical sitting position (B) modified sitting (semi-recumbent) position.

Source: This figure as been reprinted with permission from *Youman's Neurological surgery, 5th Edition*, Richard H. Winn and Julian R. Youmans, General Principles of Operative Positioning, Page 2134, Copyright Elsevier (2004).

Table 42.1. Pros and Cons of Sitting Position

Sitting Position	Pros	Cons
	Superior surgical exposure	VAE, PAE
	Improved venous and CSF drainage	Possible CV instability
	Reduced facial and conjunctival edema	Jugular venous obstruction
	Improved ventilation	Quadriplegia, paraplegia
	Better access to airway, chest, extremities	Peripheral nerve injuries
		Pneumocephalus

Adapted from Barash, M.D., Paul G., Bruce F., Cullen, M.D., and Robert K. Stoelting, M.D., eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 772–5; Miller, M.D., Ronald D., ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier, Churchill Livingstone, 2005, pp 2127–43; and Schubert, Armin. *Clinical Neuroanesthesia*. Newton, MA: Butterworth-Heinemann, 1997, pp 31–42.

Table 42.2. Pros and Cons of Lateral Positioning

Horizontal: Lateral, Prone Position	Pros	Cons
	Less VAE	Challenging surgical exposure
	Improved CV stability	Cerebellar edema
		Hemorrhage
		Ischemic optic neuropathy

Adapted from Barash, M.D., Paul G., Bruce F., Cullen, M.D., and Robert K. Stoelting, M.D., eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 772–5; Miller, M.D., Ronald D., ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier, Churchill Livingstone, 2005, pp 2127–43; and Schubert, Armin. *Clinical Neuroanesthesia*. Newton, MA: Butterworth-Heinemann, 1997, pp 31–42.

Horizontal Positions

1. Risks

- Poor access to the airway, chest, and extremities
- Cerebellar edema and hemorrhage
- Surgical access is less ideal and may require more aggressive surgical retraction, which can result in more post-operative neurological dysfunction.³
- The prone position especially has been associated with facial and tongue swelling and ischemic optic neuropathy.
- Peripheral nerve injuries are not uncommon.

2. Benefits

- Lowered risk of VAE
- Improved cardiovascular stability
- Decreased chance of cervical cord injury

Summary of Patient Positioning

Both positions have advantages and disadvantages and both can be used safely when proper precautions are taken. The decision of position should be made between the anesthesiologist and surgeon after careful evaluation of the patient's

medical history, tumor size and location, and the risks and benefits associated with the position. The ideal choice will balance surgical comfort against the risks related to the patient.⁴ There is no clear best position and the decision should be individualized for each patient.

Monitoring

- EKG and arterial blood pressure monitoring is essential.
 - Surgical manipulation can result in arrhythmias, compromising blood pressure.
 - In addition, an arterial line can aid in the measurement of CPP and is necessary to monitor changes associated with VAE.
- If the sitting position is chosen, a plan to monitor for VAE needs to be established pre-operatively and may require a transesophageal echocardiogram (TEE), precordial Doppler, and/or central line placement. (See Chapter 43 for more on VAE.)
- Somatosensory evoked potentials (SSEP) and brainstem auditory-evoked potentials (BAEP) may be considered to monitor brainstem- or position-related ischemia.

Post-operative Management

1. The decision for extubation must be made carefully since there may have been damage to respiratory centers causing temporary or permanent central apnea.
2. In addition, due to possible manipulation of cranial nerves IX, X, and XII, the patient may be at risk for aspiration and have difficulty swallowing.
3. Frequent neurological assessments, arterial blood pressure monitoring, and EKG should be continued for at least 24 hours post-operatively due to the significant risk of neurologic, cardiac, and respiratory deterioration.
4. Any bleeding or edema that occurs in the post-operative period can result in hypertension, bradycardia, and loss of consciousness due to increased intracranial pressure, bleeding, and brainstem compression or infarction.



KO TREATMENT PLAN

Pre-operative

1. Perform a complete history and physical on the child with careful attention to neurologic function including any cranial nerve deficits, mental status, volume status, or signs of increased ICP (headache, vomiting, blurry vision, mental status change, hypertension).
2. The size of the tumor, vascularity, and surgical access are issues that need to be addressed with the surgeon.
3. The decision of position needs to be made based on weighing surgical access and safety of the patient.
4. Typed and crossed blood, baseline hematocrit, and electrolytes to assess volume status are necessary.
5. An arterial line is essential and possibly a central line: especially if the sitting position is required, as it may be necessary to aspirate air from the right atrium.
6. Consider precordial Doppler when the risk of VAE is increased.

Intra-operative

1. Induction agent choices should be chosen based on cardiovascular history, ICP status, and volume status, especially if the sitting position is chosen.
2. A rapid sequence induction may be appropriate if there is a recent history of vomiting. The risk of transiently increased ICP with succinylcholine must be weighed against the risk of aspiration.

3. The endotracheal tube should be secured well and any gastric tubes should be placed before positioning.
4. Arterial line, central line, precordial Doppler, and pulmonary artery catheter (PAC) should all be considered.
5. Patient positioning should be done gradually and after adequate hydration, especially when changing to the sitting position.
6. Extremities and pressure points need to be adequately padded.
7. The eyes must be free and clear.
8. When in the sitting position, special attention should be given to the flexion of the neck and possible endotracheal tube migration.
9. Be prepared for hemodynamic instability as well as arrhythmias during dissection. These may be signs of VAE or brainstem compression.

Post-operative

1. When considering extubation, remember the risk of aspiration from cranial nerve injury and central apnea from respiratory center damage.
2. The candidate for extubation should have had an uncomplicated surgery, be hemodynamically stable, awake, spontaneously breathing adequate tidal volumes, and displaying adequate strength as well as have intact airway reflexes.
3. The post-operative management should focus on frequent neurological exams and monitoring for cardiovascular and respiratory instability.
 - a. Any alterations in these systems especially mental status changes, hypertension, or bradycardia could be an indication of bleeding, edema, or brainstem compression. Immediate re-exploration should be considered.
 - b. For the non-awakening patient, consider bleeding, edema, and tension pneumocephalus especially if the operation was completed in the sitting position.

BIBLIOGRAPHY

1. Barash, M.D., Paul G., Bruce F., Cullen, M.D., and Robert K. Stoelting, M.D., eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 772-5.
2. Miller, M.D., Ronald D., ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier, Churchill Livingstone, 2005, pp 2127-43.
3. Schubert, Armin. *Clinical Neuroanesthesia*. Newton, MA: Butterworth-Heinemann, 1997, pp 31-42.
4. Rozet, Irene, and Monica S. Vavilala. Risks and benefits of patient positioning during neurosurgical care. *Anesthesiology Clinics* 2007;25(3):631-53.

43. Venous Air Embolism (VAE)^{1,2,3,4}

Joni Maga, MD

SAMPLE CASE

A 77-year-old, 120 kg female presents for a craniotomy and resection of her posterior fossa tumor. The surgeon prefers her to be placed in the sitting position for the operation. She has a medical history significant for diabetes (DM), hypertension (HTN), congestive heart failure (CHF), and gastroesophageal reflux disease (GERD). She is a non-smoker with unknown exercise tolerance due to her decreased mobility secondary to osteoarthritis (OA) of her knees. What pre-operative labs and studies would you like? What monitors will you use? Should the procedure be done in the sitting position? Is VAE a concern? Will you use N₂O? How would you identify a VAE? Can you prevent a VAE? How would you treat a VAE?

CLINICAL ISSUES

Incidence and Risk of VAE

- VAE can occur when the operative field is greater than or equal to 5 cm above the level of the right atrium or more specifically when there is more than a 5 cm H₂O gradient between non-collapsible venous openings (e.g., diploic veins and dural sinuses) and the right atrium.
 - With a VAE, the surgical site is elevated such that the pressure at that height is greater than the central venous back pressure, therefore setting the environment to entrain air.
- Risk of occurrence is 25% and 45% during sitting cervical laminectomies and sitting posterior fossa procedures, respectively, using Doppler detection, and up to 76% during sitting posterior fossa procedures using transesophageal echocardiography [TEE] detection.²
 - Can also occur in the horizontal, lateral, supine, and prone positions.
 - Incidence of 12%⁴
- Hemodynamic consequences of air is approximated to occur at a cumulative volume of 1 mL/kg in animal studies.³
 - The lethal dose increases with slower entry of the air and decreases with a faster accumulation of air.

Pathophysiology of a VAE

- Air bubbles mechanically obstruct the pulmonary vasculature. This leads to hypoxemia and resultant vasoconstriction,

V/Q mismatch, increased pulmonary artery pressure (PAP), and reduced cardiac output (CO).

- The increase in pulmonary dead space will lower the end-tidal carbon dioxide (ETCO₂) and increase the arterial carbon dioxide (PaCO₂).
- The air bubbles cause a release of vasoactive mediators, which lead to increased vascular permeability and interstitial pulmonary edema further contributing to the increased pulmonary vascular resistance (PVR) and decrease in CO.
 - Small volumes of air can be cleared by the pulmonary vasculature without hemodynamic signs.
 - Increased filling pressures, decreased cardiac output, hypotension, and the classic “mill-wheel” murmur are late findings with a VAE.
 - Large volumes of trapped air in the right heart can cause an airlock.
 - Increasing right ventricular (RV) afterload, decreasing left ventricular (LV) filling resulting in cardiovascular (CV) collapse

Paradoxical Air Embolism (PAE)

- Risk is 5–10%.¹
- Air from a VAE can cross into the arterial system via a probe patent foramen ovale (PPFO), right to left intracardiac shunt, or transpulmonary passage.
 - The incidence of PPFO is 20–30% in adults and 50% in children less than 5 years old.
 - Air on the left side of the heart can pass into the cerebral, coronary, or renal arterial systems causing stroke, myocardial infarction (MI), or renal injury.
- PAE is more likely when the right heart pressures exceed the left.
 - When a VAE occurs, PVR increases due to increased right heart pressures.
 - This can result in air passage to the left heart via a PPFO or an intracardiac shunt.
 - In the sitting position, it has been estimated that 50% of patients have an increased right heart pressure over the left.¹
 - The normally decreased right atrial pressure (RAP) to pulmonary capillary wedge pressure (PCWP) gradient is said to reverse within 1 hour in the sitting patient but can be partially attenuated by volume loading.³

43. Venous Air Embolism

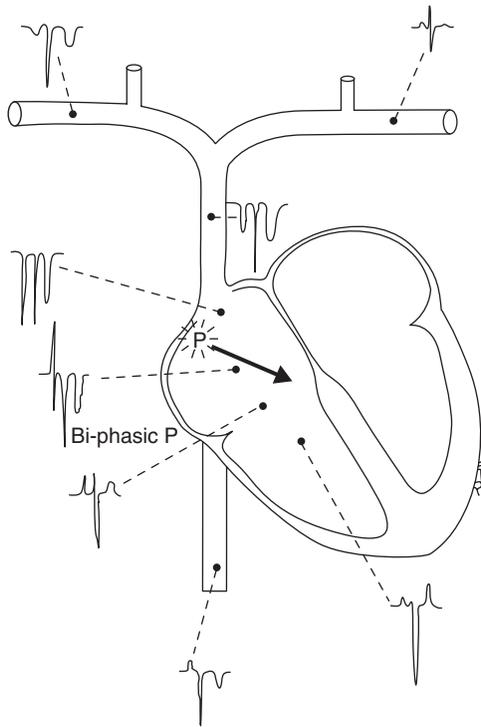


Figure 43.1. CVC line placement: The P indicates the sino-atrial node. The heavy black arrow indicates the P-wave vector. Source: This figure was published in *Anesthesia, 6th Edition*, RD Miller, LA Fleisher, RA Johns, et al., Page 2141, Copyright Elsevier (2005). Reprinted with permission.

- (d) The multi-orifice catheter is in the correct position when the P wave produces the most negative deflection, which indicates the SVC-RA junction. If using a single-orifice catheter, pull the catheter back from this position 2–3 cm.
- (e) Avoid microshock: disconnect any unnecessary electrical equipment from the patient.

- The decision of using a CVC should be based on risks of VAE, procedure (likelihood of open venous channels), position (essentially all procedures in the sitting position should be accompanied by a CVC), and the patient's medical history and physiologic reserve.
- If the concern for VAE is large enough to use a precordial Doppler or TEE, consider a CVC to have a way to remove the air that may collect.

Nitrous Oxide

- Can diffuse into trapped air and expand it, possibly making the effects of a VAE worse.
- Can be used in procedures where VAE is a risk as long as it is discontinued if a VAE occurs.
- Some would argue based on the above to avoid it all together and to eliminate N_2O as part of the equation if a VAE should occur.

Prevention of a VAE

- Early detection
- Minimize elevation of head.
- Use of bone wax: minimize open venous channels.
- Maintain euvolemia.
- Avoid PEEP/valsalva.



KO TREATMENT PLAN

Pre-operative

- Complete history and physical
 - Assess for any history of intra-cardiac shunts or PFO.
- Discuss with the surgeon concerns of VAE and together weigh the risks and benefits of the sitting position.
- Consider pre-operative TEE if the sitting position is absolutely necessary.
 - TEE is used to rule out a patent foramen ovale (PFO) or intracardiac shunt.

Intra-operative

- Choose monitors.
 - Arterial line
 - CVC
 - TEE vs. precordial doppler + PAC or $ETCO_2$
 - The addition of PAC or $ETCO_2$ to precordial Doppler will help differentiate clinically significant air before a significant hemodynamic response occurs.
- Prevention is key.
 - Bone wax
 - Avoid head-up positioning.
 - Proper hydration
- If a VAE is suspected
 - Notify surgeon and call for help.
 - Flood the surgical field with saline, use bone wax, and pack the surgical field.
 - Discontinue N_2O .
 - Compress the neck veins.
 - Increases venous pressure.
 - May prevent further air entry.
 - Place the patient in a Trendelenburg and left lateral decubitus position (LLD) to help release air from the RV outflow tract if an airlock is suspected.
 - RV is the most anterior part of heart.
 - Aspirate air from a properly positioned CVC.
 - Chest compression/percussion to release air lock or trapped air
 - Avoid PEEP and valsalva.
 - Supportive therapy
 - Maintain euvolemia.
 - May need pressors for blood pressure support.

CASE DISCUSSION

Pre-operative

1. Order a complete blood count (CBC) and basic metabolic panel to investigate any end-organ damage from the patient's HTN and DM.
2. Consider how well controlled the patient's HTN and DM are when formulating an anesthetic plan.
3. Assess the patient's current volume status and rule out any active CHF.
4. Order a chest X-ray for current evaluation and for a baseline since the patient is at risk for intra-operative and post-operative CHF exacerbation.
5. Obtain a baseline EKG in this patient who likely has some element of coronary artery disease based on her age and co-morbidities.
6. If the patient has poor exercise tolerance, order a stress echocardiogram prior to surgery.
 - a. The patient may benefit from revascularization; however, it may not be safe to delay the tumor resection for coronary artery stenting or cardiac bypass surgery.
 - b. Consider using the stress test results to guide your anesthetic plan.
 - i. Ejection fraction (EF): history of CHF
 - ii. Presence of PFO
 - iii. Patient's physiologic reserve
 - iv. Remember that hydration is one way to keep the RA pressure greater than the PCWP.
 - v. In this patient, hydration may not be a tool that can be utilized secondary to the history of CHF. Consider a PAC if the patient presents with low EF and chronic CHF.

Intra-operative

1. I would induce with etomidate because of its stable hemodynamic profile.

2. A balanced maintenance technique using a volatile anesthetic, opioid, and a muscle relaxant can be used to maintain hemodynamic stability.

- a. Consider short-acting opioids and desflurane secondary to their rapid onset and offset and therefore less long-term effect on hemodynamic stability.
3. Arterial blood pressure monitoring is indicated secondary to the patient's history of HTN, CHF, and DM.
 - a. The patient will require tight blood pressure control to avoid a CHF exacerbation.
 - b. This will be difficult with a blood pressure cuff alone, especially with her history of HTN.
 - c. In addition, the patient may need hourly glucose monitoring via an arterial blood draw and an insulin infusion.
4. If VAE is a concern, choose a way to detect it.
 - a. The combination of precordial Doppler and ETCO₂ is appropriate.

TKO: Make it easy for yourself, say, "assuming this patient has well-controlled DM and HTN and has a normal EF ..." and "assuming this patient had a recent echocardiogram with no signs of PFO, I would consider the sitting position only after the surgeon and I weighed the risk of VAE versus the need for sitting position."

BIBLIOGRAPHY

1. Barash, M.D., Paul G., Bruce F. Cullen, M.D., and Robert K. Stoelting, M.D., eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 773-5.
2. Miller, M.D., Ronald D., ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier, Churchill Livingstone, 2005, pp 2134-42.
3. Schubert, Armin. *Clinical Neuroanesthesia*. Newton, MA: Butterworth-Heinemann, 1997, pp 363-72.
4. Black, M.D., Susan, Doris B. Ockert, M.D., William C. Oliver, Jr., M.D., and Roy F. Cucchiara, M.D. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. *Anesthesiology* 69 (1988): 49-56.

44. Cerebral Aneurysm Surgery^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 60-year-old woman presents to the emergency department with a history of "the worst headache of her life." Paramedics report that although she is presently drowsy, she did briefly lose consciousness in the ambulance. Her blood pressure is 175/80 mm Hg with a heart rate of 60. Her other vital signs

are all stable. A CT scan reveals a Grade 3 subarachnoid hemorrhage (SAH). The neurosurgical team posts the case as an aneurysm clipping scheduled for the following morning. What are your concerns? How will you evaluate the patient? How will you induce and maintain general anesthesia in this patient?

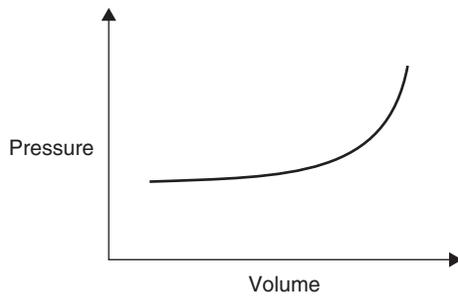


Figure 44.1. Pressure vs. volume in ICP.

CLINICAL ISSUES

Incidence of a Subarachnoid Hemorrhage

1. About 8–10 per 100,000
2. Predominantly occurring in the 55–60-year-old age group

Pathophysiology

1. With aneurysm rupture, the expanding mass effect of the hemorrhage and edema development leads to an increase in the intracranial pressure (ICP).
2. This increase in ICP leads to
 - a. Headache
 - b. Loss of consciousness
 - c. Possible motor deficits
3. Rising ICP leads to a reactive increase in systemic blood pressure to maintain the cerebral perfusion: classically referred to as the Cushing reflex.
 - a. $CPP = MAP - ICP$.
 - b. Cerebral perfusion pressure = CPP
 - c. Mean arterial pressure = MAP
4. Eventually the increase in ICP leads to a decrease in cerebral blood flow, limiting further leakage of blood from the aneurysm.

Rebleeding

1. 4% incidence within the first day.
2. After 48 hours, the incidence is 1.5% per day, with a cumulative rebleeding rate of 19% by the end of two weeks.

Transcranial Doppler Monitoring (TCD)

1. Direct and non-invasive monitor of cerebral blood flow (CBF)
2. Indications
 - a. Determine the severity of cerebral vasospasm after a subarachnoid hemorrhage.
 - b. Measure the CBF during carotid artery clamping in a carotid endarterectomy.
 - c. Detection of emboli after an arterial repair or during cardiopulmonary bypass
 - d. Brain death diagnosis



KO TREATMENT PLAN

Pre-operative

1. Hunt and Hess classification of patients with SAH¹
 - a. Grade 0: unruptured aneurysm
 - b. Grade I: asymptomatic or minimal headache and slight nuchal rigidity
 - c. Grade II: moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
 - d. Grade III: drowsiness, confusion, or mild focal deficit
 - e. Grade IV: stupor, moderate to severe hemiparesis, early decerebration, vegetative disturbance
 - f. Grade V: deep coma, decerebrate rigidity, moribund
 - i. Higher grades are associated with the presence of cerebral vasospasm, intracranial hypertension, and increased surgical mortality.
2. Neurogenic pulmonary edema (NPE)
 - a. Occurs in 8 to 29% of patients with SAH
 - b. NPE occurs with sympathetic activation secondary to the rise in ICP, leading to pulmonary and systemic vasoconstriction. This leads to an increase in pulmonary blood volume and a decrease in left ventricular compliance, with a concomitant rise in pulmonary capillary pressure and pulmonary capillary permeability.
 - c. Treat with immediate surgical or pharmacologic relief of intracranial hypertension, supportive respiratory care, and careful fluid management.
3. Cardiac dysrhythmias and EKG abnormalities
 - a. Due to excessive catecholamine release triggered by the SAH.
 - b. Correlates with degree of neurologic injury rather than cardiac dysfunction.
 - c. Commonly presents with
 - i. Very deep, widely splayed T wave inversions
 - ii. Long QT
 - iii. Prominent U wave
 - d. May also cause
 - i. Systemic and pulmonary hypertension
 - ii. Myocardial dysfunction and injury
 - iii. Neurogenic stunned myocardium
4. Electrolyte abnormalities
 - a. Hyponatremia
 - i. Cerebral salt-wasting syndrome or syndrome of inappropriate anti-diuretic hormone (SIADH)
 - b. Other electrolyte abnormalities, such as hypokalemia, hypocalcemia, and hypomagnesemia may occur secondary to diuretic therapy in an attempt to lower the ICP with loop or osmotic diuretics.

Intra-operative

1. The goals of intra-operative management of these patients are to avoid aneurysm rupture, maintain cerebral perfusion pressure, maintain transmural aneurysm pressure, and

44. Cerebral Aneurysm Surgery

provide a “slack brain.” The anesthetic management provided below attempts to maintain these goals.

- a. The induction agents can vary with or without pre-medication depending on the patient’s clinical status.
 - b. The patient should be considered a “full stomach” and requires a rapid-sequence intubation.
 - c. Blunt the sympathetic response to laryngoscopy with intravenous fentanyl (3 mcg/kg) and/or intravenous lidocaine (1.5 mg/kg).
 - d. Pre-treatment with 0.01 mg/kg of vecuronium three minutes prior to giving succinylcholine helps to prevent the associated increase in ICP.
 - e. Etomidate is the induction agent of choice secondary to its hemodynamic stability and decreasing effect on ICP.
 - f. Thiopental can be used. It has cerebroprotective effects but can cause hypotension, so it should be titrated carefully.
 - g. Maintenance of anesthesia is achieved with total intravenous anesthesia or inhalational agents, plus appropriate doses of narcotic to prevent pain sensation.
2. Intravenous access
 - a. At least two large-bore intravenous catheters
 - b. The large IVs are important if the aneurysm ruptures and large volumes of blood replacement are required.
 - c. Be careful to avoid giving the patient large amounts of crystalloid during a controlled case with minimal blood loss.
 3. Standard monitoring should be geared to the patient’s needs.
 - a. A central venous pressure (CVP) catheter may be inserted for guidance of intravascular volumes, especially in the face of severe cardiac instability.
 - b. Pulmonary artery catheter (PAC) monitoring is justified in the setting of myocardial dysfunction and post-operative Triple H therapy (see following).
 4. Avoid hypertension and resultant aneurysmal rupture.
 - a. May be accomplished with intravenous lidocaine, esmolol, labetalol, or the establishment of a deep plane of anesthesia.
 5. Intracranial hypertension treatment
 - a. Techniques to decrease intracranial pressure include
 - i. Hyperventilation
 - ii. Osmotic diuresis
 - iii. Administration of barbiturates
 - iv. Cerebral spinal fluid (CSF) drainage
 6. Neurophysiologic monitoring may be beneficial in areas potentially affected by the aneurysm clipping.
 7. Induced hypotension
 - a. Maintain cerebral perfusion pressure by avoiding sudden or profound decreases in systemic blood pressure.
 - b. Clips are often applied to the feeder vessels of the aneurysm in order to decrease bleeding and aid visualization rather than using deliberate hypotension.
 - c. Thiopental may be given to provide neuro-protection prior to the placement of a temporary clip.

Post-operative

1. The goals at emergence are to avoid coughing, straining, hypercarbia, and hypertension, as all these will lead to an increase in ICP.
 - a. Patients who were Hunt-Hess grades I and II pre-operatively, with no intra-operative complications, may have the endotracheal tube removed upon conclusion of surgery.
 - b. Patients who arrived in the operating room with decreased mentation should remain intubated until their neurologic status improves.
2. Post-operative monitoring for severe hypertension, hypotension, and vasospasm must be initiated.
 - a. **Severe hypertension occurs from pre-existing hypertensive disorders, pain, CO₂ retention, or “Triple-H” therapy (see following).**
 - i. It may lead to cerebral edema or hematoma formation, leading to increased ICP and vasospasm.
 - b. Hypotension must be avoided as the decreased blood pressure leads to decreased cerebral perfusion pressure, leading to neurologic compromise.
 - c. Cerebral vasospasm
 - i. Develops 3–12 days after SAH, with a peak on days six to seven.
 - ii. Presents with neurologic deterioration and drowsiness.
 - iii. Results in cerebral ischemia and is the leading cause of morbidity and mortality if the patient survives the initial bleed.
 - iv. Diagnosis is made via angiography, TCD, or clinical progression of neurologic deficits.
 - v. A combination of increased ICP and hypovolemia increases the likelihood for cerebral vasospasm.
 - vi. Prophylaxis and treatment of vasospasm
 - (1) Nimodipine: a calcium channel blocker documented to improve outcome via an unknown mechanism. The goal is to maintain a stable blood pressure and avoid hypotension.
 - (2) “Triple-H” therapy: hypertension, hypervolemia, and hemodilution
 - (a) The goal is to increase cerebral blood flow (CBF), increase CPP, and improve cerebral blood flow with decreased blood viscosity.
 - (b) Systolic blood pressure (SBP) is raised to 160–200 mm Hg in clipped aneurysms.
 - (c) Pulmonary artery wedge pressure is maintained at 12–18 mm Hg or central venous pressure at 10–12 mm Hg.
 - (d) Hematocrit is decreased to 33% to maximize oxygen delivery to tissues.
 - (e) It is contraindicated in patients with unclipped aneurysms.
 - (f) Monitor patients with concomitant cardiac dysfunction, myocardial injury, or respiratory impairment carefully.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 777–81.
2. Priebe HJ. Aneurysmal subarachnoid haemorrhage and the anaesthetist. *British J Anaesthesia* 2007;99(1):102–18.
3. Muroi C, Keller M, Pangalu A, Fortunati M, Yonekawa Y, Keller E. Neurogenic pulmonary edema in patients with subarachnoid hemorrhage. *J Neurosurg Anesth* 2008;20(3):188–92.
4. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1540–2, 2128.

45. Head Trauma^{1,2,3,4,5}*Kasia Petelenz Rubin, MD***SAMPLE CASE**

A 40-year-old man presents to the trauma bay after a prolonged extrication, following an ATV accident. He is intoxicated, incoherent, and combative. The trauma team would like to sedate the patient for a CT scan to evaluate his head, abdomen, and pelvis. The patient's blood pressure is 170/90 mm Hg, heart rate is 48, and temperature is 35.5°C. What are your concerns in a traumatic brain injury (TBI) patient? How will you evaluate the patient? How will you induce and maintain general anesthesia in this patient?

CLINICAL ISSUES**Trauma Assessment and Emergency Therapy: ABCD**

1. Airway and breathing: secure the airway in any patient with a Glasgow Coma Scale <8.
 - a. The most expeditious approach to secure the airway, assuming that the patient does not have a difficult airway, is pre-oxygenation of the patient followed by a rapid-sequence induction (RSI) and intubation with cricoid pressure and maintenance of in-line cervical stabilization.
 - b. Assume “full-stomach” in all head injury patients.
 - i. In the setting of urgent airway securement, the benefits of the rapid onset and elimination of succinylcholine outweigh the risk of a transient increase in intracranial pressure (ICP).
 - c. There is a 1–3% incidence of cervical spine injury in the setting of known head injury.
 - i. In-line cervical stabilization requires an assistant to hold the occiput down on a backboard, with fingers on the mastoid process.
 - ii. The goal of in-line cervical stabilization is to prevent any further cervical injury during the intubation of the patient.

- d. Avoid nasal intubations in the face of basal skull fracture, severe facial fractures, or suspected bleeding diathesis.
2. Circulation
 - a. Hypotonic solutions (LR or 0.45% NaCl) are more likely to increase brain water content with a large-volume fluid resuscitation secondary to a profound reduction in colloid oncotic pressure. This oncotic gradient leads to cellular swelling and brain edema.
 - b. Isotonic solutions (0.9% NaCl) prevent immediate increases in ICP and maintain plasma volume. Normal saline is typically the resuscitation fluid of choice in head trauma patients.
 - c. Hypertonic saline increases blood pressure and decreases overall fluid requirements. The use of hypertonic saline may become more prevalent but as yet is not common practice.
 3. Disability
 - a. Evaluate for other injuries including pneumothorax, intra-abdominal injury, and so on.

Optimize Cerebral Perfusion

1. CPP = MAP - ICP (CPP = cerebral perfusion pressure and MAP = mean arterial pressure)
2. Maintain the CPP above 60 mm Hg to maintain cerebral blood flow and avoid cerebral desaturation.
3. A low CPP correlates with poor neurologic outcomes, as cerebral ischemia may occur at levels less than 50 mm Hg.
4. Routine use of pressors or volume expanders to maintain CPP above 70 mm Hg leads to systemic complications and does not improve general outcomes.
5. An ICP monitor may be needed.

Avoid Secondary Damage

1. Avoid hypoxia
 - a. Hypoxia is significantly associated with increased morbidity and mortality.

45. Head Trauma

2. Avoid hypercarbia

- a. Increased CO₂ leads to increased cerebral blood volume and flow via cerebral vasodilation.
- b. In the setting of decreased intracranial compliance, ICP rises and cerebral perfusion pressure decreases.

3. Avoid hypotension

- a. A systolic blood pressure of 90 mm Hg is generally accepted as the lowest threshold.
- b. A single episode of pre-hospital or in-hospital hypotension significantly increases mortality and morbidity.
- c. Do not actively try to decrease the patient's elevated blood pressure.
- d. Hypertension may be precipitated by the increase in ICP: Cushing's reflex.
- e. A reduction in systolic blood pressure (SBP) can aggravate cerebral ischemia by reducing CPP.
 - i. Again: CPP = MAP - ICP

4. Avoid anemia.

- a. Minimum hematocrit between 30 and 33% is recommended to maximize oxygen transport.

5. Avoid hyperglycemia.

- a. Hyperglycemia (blood glucose >200 mg/dL) is correlated with the severity of injury and is associated with a poor outcome in regard to early mortality and functional recovery.

Non-operative Treatment of Diffuse Cerebral Swelling

1. Hyperventilation

- a. Reduces ICP by causing vasoconstriction with a subsequent decrease in cerebral blood flow.
- b. Hyperventilation is no longer recommended to less than 35 mm Hg in the first 24 hours, as the vasoconstriction that is induced may lead to increased ischemia.

2. Mannitol

- a. Osmotic diuretic
- b. Onset is delayed 15–30 minutes after administration, with effects persisting for 90 minutes to 6 hours or more.
- c. The immediate increase in plasma volume with administration leads to a decrease in hematocrit, reducing blood viscosity, increasing cerebral blood flow (CBF), and increasing oxygen delivery.
- d. There is insufficient evidence to conclude whether continuous infusion or intermittent boluses are optimal in reducing ICP.

3. Hypertonic saline: there is little evidence to determine the efficacy and relative benefit over mannitol for decreasing ICP.

- a. Effects of decreased ICP are due to osmotic movement of water across an intact blood-brain barrier.
- b. Leads to plasma volume expansion with improved blood flow.

4. Barbiturate administration

- a. Because of the coupling of cerebral blood flow to cerebral metabolic rate of oxygen consumption (CMRO₂), decreasing CMRO₂ leads to a decrease in ICP and decreased global cerebral perfusion.

Table 45.1. Glasgow Coma Scale

Glasgow Coma Scale		
Eyes open	Spontaneous	4
	To speech	3
	To pain	2
Best verbal response	None	1
	Oriented	5
	Confused	4
	Inappropriate words	3
Best motor response	Incomprehensible sounds	2
	None	1
	Follows commands	6
	Localizes pain	5
	Withdrawal to pain	4
Maximum Score/ Minimum Score	Flexion to pain	3
	Extension to pain	2
	None	1
	15/3	

Adapted from Miller RD, ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Churchill Livingstone, 2005.

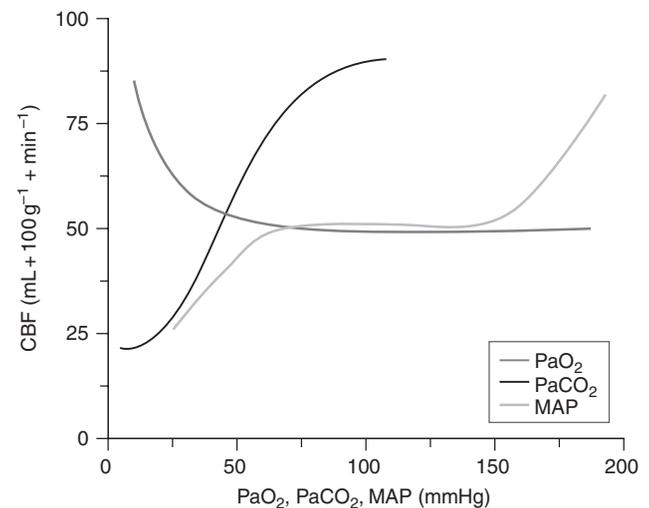


Figure 45.1. Cerebral perfusion pressure.

Source: This figure was published in *Anesthesia*, 6th Edition, RD Miller, LA Fleisher, RA Johns, et al., Copyright Elsevier (2005). Reprinted with permission.

- b. However, barbiturate therapy often results in a fall in blood pressure, offsetting any ICP-lowering effect on cerebral perfusion pressure. There is no evidence that this therapy improves outcomes.

5. Steroids do not improve outcome or lower ICP in severe TBI.

45. Head Trauma

6. Hypothermia

- a. Hypothermia decreases $CMRO_2$. Theoretically, decreasing the temperature will decrease cerebral blood flow, thus decreasing ICP. However, hypothermia is associated with significant other problems.
 - i. Coagulopathy
 - ii. Increased infection rate
 - iii. Delayed emergence from anesthesia
 - iv. Cardiac dysrhythmias
- b. Routine use of active cooling to less than $35^{\circ}C$ is therefore NOT advocated.



KO TREATMENT PLAN

Pre-operative

1. Primary and secondary surveys must be completed in the trauma bay. The patient must be fully evaluated prior to proceeding to another setting.
2. Assuming that the patient has a normal airway, due to his combative nature, intoxication, and probable head injury, proceed with a RSI with manual in-line cervical stabilization to secure the airway with an endotracheal tube, prior to proceeding to the CT scanner.
 - a. Obtaining lateral X-rays will only delay definitive airway control, and they are unreliable to rule out ligamentous injury.
 - b. Again, succinylcholine leads to a transient increase in ICP, but the benefits of rapid airway securement outweigh this transient change.
3. Do not treat the hypertension. It is likely elevated in response to the increase in ICP.
4. Initiate non-operative treatments to decrease the ICP. This decrease in ICP may result in a decrease in the patient's blood pressure, while maintaining the cerebral perfusion pressure.

Intra-operative

TKO: Sometimes more script is revealed during the exam. Roll with the following punches ... After the CT scan, it is discovered that the patient has a subdural hemorrhage and requires emergency surgery to decompress his brain.

1. In addition to standard ASA monitors, other monitors include
 - a. Foley catheter: if diuretics are to be given for ICP monitoring, a Foley is mandatory.
 - b. Arterial line: close blood pressure monitoring is critical for reasons described above.

Post-operative

1. Extubation should be carried out only when the patient is appropriately responsive. If the patient exhibited depressed mentation prior to the surgery, endotracheal intubation

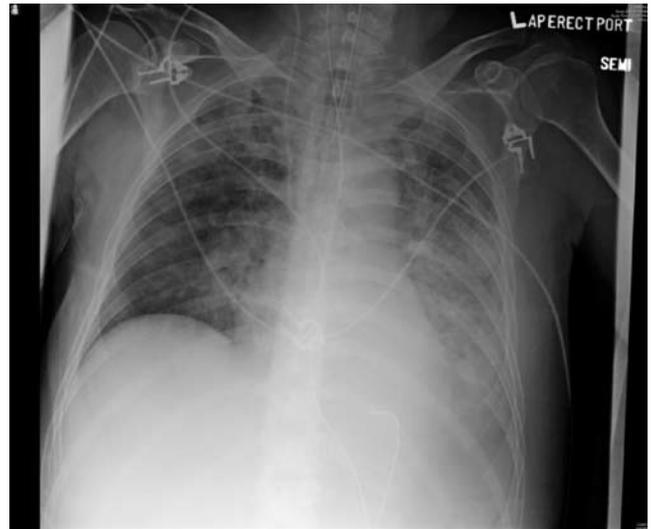


Figure 45.2. Trauma-associated acute lung injury. Photo credit: MetroHealth Medical Center, Cleveland, OH.

should be maintained through the initial post-operative time frame, until the patient's neurologic status improves.

2. Monitor the patient in an appropriate setting for possible systemic sequelae, including
 - a. Cardiopulmonary issues
 - i. Sympathetic hyperactivity is typical in the post-TBI setting, causing
 - (1) Left ventricular dysfunction which can lead to hypotension
 - (2) Hypertension with resultant
 - (a) Trauma-associated acute lung injury
 - (b) Neurogenic pulmonary edema
 - (c) Subarachnoid hemorrhage
 - b. Immune system depression leads to infectious complications.
 - i. Pneumonia
 - c. Pulmonary
 - i. Trauma-associated acute lung injury
 - d. Seizures
 - i. Relatively common after TBI
 - ii. Seizures increase the metabolic rate and increase ICP.
 - iii. Anti-epileptics are typically started in the immediate post-operative period.
 - e. Hematologic complications
 - i. Severe TBI patients are likely to develop a coagulopathy.
 - (1) Disseminated intravascular coagulation (DIC) can occur secondary to brain thromboplastin that is released into systemic circulation after brain injury.
 - (2) Therapy is supportive, with treatment and resolution of the underlying problem, in order to control the consumptive coagulopathy that may develop.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 782–6.
2. Miller RD, ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Churchill Livingstone, 2005, pp 815–45, 2144–64.

3. Brain Trauma Foundation. Guidelines for the Management of Severe Traumatic Brain Injury, 3rd ed. *J Neurotrauma* 2007;24S:1–95.
4. Moppett IK. Traumatic brain injury: assessment, resuscitation and early management. *British J Anaesth* 2007;99(1):18–31.
5. Lim HB, Smith M. Systemic complications after head injury: a clinical review. *Anaesthesia* 2007;62:474–82.

46. Syndrome of Inappropriate Anti-Diuretic Hormone Secretion (SIADH), Cerebral Salt-Wasting Syndrome (CSWS), and Diabetes Insipidus (DI)^{1,2,3}

Donn Marciniak, MD

SAMPLE CASE

A 50-year-old male is 24 hours status post transsphenoidal resection of a pituitary tumor. He is producing less than 50 mL/hr of urine for several hours and is complaining of thirst. What is the likely etiology of this process and what laboratory values are expected? How should this patient be managed if they return to the operating room emergently?

SYNDROME OF INAPROPRIATE ANTIDIURETIC HORMONE (SIADH)

CLINICAL ISSUES**Definition**

1. The syndrome of inappropriate antidiuretic hormone (SIADH)
 - a. Syndrome of excess release of ADH from the posterior pituitary gland or another source.
 - b. The release of ADH is then not inhibited by the resulting reduction in plasma osmolality.
 - c. State of hyponatremia in combination with elevated urine osmolality, excessive sodium secretion, and decreased serum osmolality
 - i. Condition of excess water and not a sodium deficiency
 - ii. Patients are usually hypervolemic.
 - d. Cells secreting antidiuretic hormone (ADH) are dysregulated and water absorption from the distal collecting tubules is enhanced.

Incidence

1. Hyponatremia
 - a. Defined as a plasma sodium concentration less than 135 mEq/L.
 - i. The most common electrolyte derangement in hospitalized patients
 - ii. Incidence may be as high as 30%.
 - b. A serum sodium concentration below 130 mEq/L is found in less than 3% of all hospitalized patients.
 - c. ADH dysregulation is the most common etiology of hyponatremia.

Etiology

1. Idiopathic: most common
2. Post-operative
3. Central nervous system disease
 - a. Head trauma or intracranial bleeding
 - b. Tumors and cerebrovascular accidents
 - c. Delirium tremens
4. Neoplastic
 - a. Lung: small cell
 - b. Pancreas
 - c. Ovary
 - d. Lymphoma
 - e. Thymoma
5. Endocrine
 - a. Glucocorticoid insufficiency
 - b. Hypothyroidism
6. Pulmonary disease
 - a. Pneumonia
 - b. Positive pressure ventilation
 - c. Chronic obstructive pulmonary disease (COPD)

46. SIADH, Salt-Wasting, and Diabetes Insipidus

7. Medications
 - a. Chlorpropramide
 - b. Cyclophosphamide
 - c. Tricyclic antidepressants (TCAs)
 - d. Selective serotonin reuptake inhibitors (SSRIs)
 - e. Nicotine
 - f. MDMA drugs
 - i. Ecstasy
 - g. Phenothiazine
8. Infectious
 - a. Cytomegalovirus
 - b. Mycobacteria associated with AIDS
 - c. Brain or lung abscess

Signs and Symptoms

1. Anorexia
2. Nausea and vomiting
3. Malaise
4. Headache
5. Confusion, stupor, and coma
6. Seizures

Differential Diagnosis

1. Adrenal insufficiency
2. CSWS (cerebral salt-wasting syndrome)
3. Congestive heart failure
4. Diabetes mellitus
5. Hypopituitarism
6. Hypothyroidism
7. Nephrotic syndrome
8. Polydipsia
9. Simple hyponatremia

Testing and Diagnosis of SIADH

1. Hyponatremia: serum sodium <130 mEq/L
 - a. Often a diagnosis of exclusion for euvolemic hyponatremia, defined as serum sodium <135 mEq/L.
2. Plasma osmolality <270 mOsm/kg.
3. Urine sodium concentration >20 mEq/L
 - a. Concentrated urine with a low flow rate and associated hypervolemia
 - b. Urine osmolality is usually >100 mOsm/kg and sodium osmolality is >40 mEq/L.
 - c. Urine sodium excretion is generally equal to intake.
4. Low
 - a. Blood urea nitrogen
 - b. Creatinine
 - c. Uric acid
 - i. Hypouricemia and increased fractional excretion of uric acid may normalize after the correction of hyponatremia.
 - d. Albumin

5. Exclude pseudohyponatremia by evaluating serum proteins, lipids, and glucose.
6. Normal serum bicarbonate and potassium levels

Treatment

1. Treat the underlying cause if possible.
2. Primarily fluid (water) restriction
 - a. 800–1,000 ml per day
3. Intravenous (IV) saline
 - a. Used for very symptomatic patients
 - b. Hypertonic saline (5%) 200–300 ml IV over 3–4 hours
4. Medications
 - a. Diuretics
 - b. Demeclocycline
 - i. Used for chronic cases when fluid restriction is difficult to maintain.
5. The water imbalance should not be corrected rapidly.

CEREBRAL SALT WASTING

CLINICAL ISSUES

Definition

1. Cerebral salt-wasting syndrome (CSWS)
 - a. State of hyponatremia with increased natriuresis
 - b. Hyponatremic dehydration owing to intracranial pathology
 - i. Excessive renal sodium excretion secondary to a centrally mediated process
 - ii. The patients are usually dehydrated and hypovolemic (unlike SIADH where the patient is usually hypervolemic).
 - c. There is some debate if this is a form of SIADH or a unique disease.

Incidence

1. The incidence of CSWS is unknown.
 - a. Affects patients of all ages.
 - b. Up to 60% of patients with intracranial tumors or brain injuries develop hyponatremia and CSWS may be responsible for an imbalance in sodium as frequently as SIADH.
 - c. Some experts question the existence of this syndrome and consider it an extension of SIADH.

Etiology

1. Head injury
2. Intracranial tumor or surgery
3. Intracerebral bleed or stroke
4. Meningitis

46. SIADH, Salt-Wasting, and Diabetes Insipidus

Signs and Symptoms

1. Similar to SIADH

Differential Diagnosis

1. Idiopathic hyponatremia
2. SIADH

Testing

1. Serum chemistry shows hyponatremia.
2. Dilute urine with a high flow rate
3. Random urine sodium greater than 40 mEq/L
4. Urine sodium excretion greater than intake: negative sodium balance
5. Hypouricemia and increased fractional excretion of uric acid persist after correction of hyponatremia.

Treatment

1. Fluids
2. Correction of the low sodium
3. Medications
 - a. Fludrocortisone: mineralocorticoid

DIABETES INSIPIDUS (DI)

CLINICAL ISSUES

Definition

1. Diabetes insipidus (DI)
 - a. State of hypernatremia with a normal total body concentration of sodium
 - b. An inability to concentrate urine due to either renal resistance to antidiuretic hormone (ADH), known as nephrogenic DI or a decrease in ADH secretion known as central DI¹
 - c. Excretion of large amounts of extremely dilute urine, which does not decrease when the fluid intake is decreased

Incidence

1. DI has an estimated prevalence of 1:25000 in hospitalized patients.

Etiology

1. Central diabetes insipidus
 - a. Traumatic
 - i. Surgical
 - ii. Accidental

- b. Neoplasm
 - i. Lymphoma
 - ii. Craniopharyngioma
 - iii. Metastasis
- c. Granulomatous disease
 - i. Histocytosis X
 - ii. Sarcoidosis
- d. Idiopathic
- e. Infectious
 - i. Meningitis
 - ii. Encephalitis
- f. Vascular
 - i. Cerebral aneurysms
 - ii. Sheehan's syndrome
2. Nephrogenic diabetes insipidus
 - a. Metabolic
 - i. Hypokalemia
 - ii. Hypercalcemia
 - b. Infectious
 - i. Pyelonephritis
 - c. Post-renal obstruction release
 - d. Vascular
 - i. Sickle cell anemia
 - e. Granulomatous disease
 - i. Sarcoidosis
 - f. Drug effects
 - i. Lithium
 - ii. Amphotericin B
 - iii. Demeclocycline
 - iv. Methoxyflurane
 - g. Genetic
 - i. X-linked
 - ii. Polycystic kidney disease

Signs and Symptoms

1. Excessive urination: day and night
2. Extreme thirst
3. Some patients will develop signs of dehydration.
4. May present with fever, vomiting, and diarrhea.

Differential Diagnosis

1. Untreated diabetes mellitus type 1
2. Psychogenic polydipsia
3. Osmotic diuresis

Testing

1. Diagnosis is often made clinically.
2. Check the patient's serum electrolytes.
 - a. Sodium
 - b. Glucose
 - c. Bicarbonate
 - d. Calcium

46. SIADH, Salt-Wasting, and Diabetes Insipidus

3. Urinalysis
 - a. Dilute urine
 - b. Low specific gravity: less than 1.005 and urine osmolality less than 200 mOsm/kg
4. Random plasma osmolality is often >287 mOsm/kg.
5. Water deprivation test can be used to determine if the DI is caused by
 - a. Excessive intake of fluid
 - b. A defect in ADH production
 - c. A defect in the kidney's response to ADH
 - i. The test measures the patient's weight, urine output, and urine composition when fluids are withheld.
6. Desmopressin stimulation test
 - a. Used to distinguish between nephrogenic and central DI
 - b. Central DI: the patient has a reduction in urine output and increased urine osmolality.
 - c. Nephrogenic DI: no change in the urine output or urine osmolality

Treatment

1. Central DI
 - a. Desmopressin: IV, nasal spray, or tablet
 - b. Adequate hydration
2. Nephrogenic
 - a. Diuretic: hydrochlorothiazide
 - b. Indomethacin
 - c. Adequate hydration



KO TREATMENT PLAN

The patient in the case stem question is likely experiencing SIADH associated with the transsphenoidal resection of his pituitary tumor. SIADH after resection is not uncommon and typically presents with hyponatremia, decreased serum osmolality, and increased urine sodium along with complaints of thirst.

Pre-operative

1. Patients with sodium level greater than 125 mEq/L are generally asymptomatic.
2. A serum sodium less than 105 mEq/kg is life threatening.
3. Electrolytes should be checked pre-operatively and optimized if possible.
4. Pre-operative sedation should be administered judiciously in patients with associated mental status changes.

5. Fluid balance should also be optimized pre-operatively if possible.

Intra-operative

1. Induction agents should be chosen with consideration to volume status.
2. A urinary bladder catheter should be placed to help monitor the patient's volume status.
3. Consider an arterial catheter if
 - a. Frequent laboratory values will be checked.
 - b. There is a concern of hemodynamic lability.
 - c. They aid in fluid resuscitation via pulse pressure variation.
4. Fluid replacement should commence judiciously due the concern of central pontine myelinolysis.
 - a. Central pontine myelinolysis is a non-inflammatory demyelination of the pons, associated with a rapid correction of hyponatremia.
 - b. Presents with sudden quadraparesis, dysphagia, dysarthria, diplopia, and loss of consciousness.
 - c. The patient may experience a "locked-in syndrome", where cognitive function is intact, but all muscles are paralyzed except for eye blinking.

Post-operative

1. Patient should be discharged to an appropriate level of care setting where resuscitation needs can be adequately met.
2. Hyponatremia associated with SIADH should be corrected with hypertonic saline in the symptomatic patient or a patient with serum sodium less than 110 mEq/L.
 - a. The rate of correction should be less than 0.5 mEq/L/hr.
 - i. More severe circumstances allow for a more rapid correction.
 - ii. Should not exceed 15 mEq in 24 hours.
 - b. Serum electrolytes must be closely monitored.
 - c. Furosimide may be considered since free water loss is relatively greater than sodium loss.

BIBLIOGRAPHY

1. Fleisher, LA. *Anesthesia and Uncommon Diseases*, 5th ed. Philadelphia: Saunders Elsevier, 2006, pp 430-2.
2. Yao, FSF. *Yao and Artusio's Anesthesiology*, 6th ed. Philadelphia: Lippincott, Williams, & Wilkins, 2008, pp 601, 826-7.
3. Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006, pp 669-74.

47. Spinal Cord Injury (SCI)^{1,2,3,4}

Melvyn J. Y. Chin, MD, Kamaljit K. Sidhu, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 32-year-old male with a spinal cord injury (SCI) at the level of T4 from a motor vehicle accident nine months ago presents for a pre-surgical anesthesia evaluation. He will be undergoing a lithotripsy for bladder stones. He has no sensation below the level of T4. Apart from the SCI, he has no other medical problems. What are your considerations for this patient? If this patient has autonomic dysreflexia, how would this change your anesthetic plan?

CLINICAL ISSUES

Acute SCI

1. Airway management/respiratory
 - a. Cervical stabilization: your goal is to prevent further spinal cord injury.
 - i. Manual in-line stabilization
 - ii. Glidescope
 - iii. Awake fiber optic intubation
 - b. Lesions above C5 usually require intubation and mechanical ventilation.
 - i. The phrenic nerve arises from C3 to C5.
 - c. Injuries below the cervical region may still require mechanical ventilation due to respiratory difficulties: weak accessory respiratory muscles.
 - d. Supine positioning improves respiration compared to an upright position in SCI patients.
 - i. Paradoxical respiration (chest wall collapse with inspiration) may occur from the loss of chest wall and abdominal muscle tone.
 - ii. In the upright position, the abdominal contents are not being pushed up against the diaphragm because of the loss of abdominal wall muscle tone. Since the diaphragm sits lower, the diaphragmatic excursion and vital capacity are reduced.
 - e. SCI patients are more prone to hypoxemia and pneumonia.
 - i. Atelectasis
 - ii. Unopposed vagal tone
 - (I) Increased airway secretions
 - iii. Weak accessory respiratory muscles
 - iv. Ineffective cough
 - v. Pulmonary edema: catecholamine surge that occurs after an acute trauma to the spinal cord.

- f. Post-operative respiratory care
 - i. Aggressive bronchial hygiene: suctioning, positive pressure, and assisted cough mechanisms
 - ii. Incentive spirometry
 - iii. Percussion and vibration
 - iv. Frequent position changes
 - v. Intermittent positive pressure breathing
 - vi. Beta agonist therapy (i.e., albuterol)
2. Spinal shock
 - a. Definition:
 - i. Loss or depression of all or most of the spinal reflex activity below the level of spinal injury
 - ii. The loss of sympathetic tone results in unopposed parasympathetic stimulation.
 - b. Timeline: duration can be 24 hours to 3 months after the initial injury, but on average, it only lasts 3 weeks post-injury.
 - c. Clinical manifestations: injuries above T6 are more likely to result in autonomic changes.
 - i. Hypotension
 - ii. Bradycardia: loss of activity in the T1-T4 cardioacceleratory fibers
 - iii. Decreased preload
 - iv. Decreased peripheral vascular resistance
 - d. Management
 - i. Fluids
 - ii. Catecholamine infusion
 - iii. May require temporary transcutaneous cardiac pacing.
 - iv. The ideal mean arterial pressure (MAP) that allows for maximal cord perfusion is uncertain; therefore, it is recommended that the patient be kept normotensive (MAP >80–90 mm Hg).

Chronic SCI

1. Autonomic dysreflexia
 - a. Definition
 - i. Noxious stimulus below the level of injury causes a massive reflex sympathetic discharge.
 - ii. Occurs in patients with a SCI lesion at T6 or above.
 - iii. Sympathetic discharge is unopposed because of the interruption of descending sympathetic control, and the receptors below the level of injury are hypersensitive.

47. Spinal Cord Injury

- b. Timeline**
 - i.** Develops after the resolution of spinal shock.
 - ii.** It usually develops within the first 6 months to 1 year after the initial SCI.
- c. Clinical manifestations**
 - i.** Noxious stimulus below the level of the spinal cord lesion results in an uninhibited sympathetic response.
 - ii.** The autonomic dysreflexia will occur independently of the patient's sensation or lack of sensation.
 - iii. Symptoms**
 - (1) Hypertension: secondary to sympathetically mediated vasoconstriction of the splanchnic, muscle, and skin vasculature
 - (2) Reflex bradycardia: carotid response to the hypertension
 - (3) Headache
 - (4) Malaise
 - (5) Piloerection
 - (6) Sweating and flushing above the level of the SCI
- d. Common noxious stimuli**
 - i.** Bladder distension
 - ii.** Bowel distension
 - iii.** Infection
 - iv.** Labor
 - v.** Any abdominal emergency
 - vi.** Fracture: occult or obvious
 - vii.** Surgery at a site below the level of the lesion
- e. Management: if untreated, it may lead to a cerebral vascular accident (CVA), subarachnoid hemorrhage (SAH), seizure, retinal hemorrhage, or death.**
 - i.** Sit the patient up.
 - ii.** Identify and remove the noxious stimulus (see above).
 - iii.** Treat the hypertension:
 - (1) Nitroglycerine (paste, SL, IV)
 - (2) Hydralazine
 - (3) Nifedipine
 - (4) Clonidine
 - (5) Nitroprusside
 - (6) Spinal anesthesia
- f. Prevention**
 - i.** Preventing the development of autonomic dysreflexia is better than having to treat it.
 - ii.** Examples: general anesthesia/epidural/spinal for laboring patients or surgical procedures, even if the patient has no sensation of pain.
 - iii.** A SCI patient with a lesion above T6 having surgical procedures below the level of the injury will require adequate anesthesia (neuraxial or general) to prevent autonomic dysreflexia.
- g. Anesthetic considerations**

h. Succinylcholine should be avoided in any SCI patient.

(1) Risk of sudden, severe hyperkalemia leading to cardiac arrest.

(2) This has been reported in patients with thoracolumbar SCI and muscle denervation.

(3) The massive efflux of potassium into the serum is presumed to be due to the proliferation of the extra-junctional acetylcholine receptors and its subsequent depolarization.

(4) The use of succinylcholine is probably safe within the first week after injury.



KO TREATMENT PLAN

Pre-operative

- 1. Standard history and physical**
 - a.** Level of injury
 - b.** Length of time of injury
 - c.** History of prior autonomic dysreflexia
 - d.** Baseline blood pressures
 - e.** Respiratory status
 - f.** Recent infections/pneumonias
 - g.** Prior anesthetic complications

Intra-operative

- 1. Despite the fact that the patient lacks sensation below T4, anesthesia is still required to prevent the onset of autonomic dysreflexia.**
 - a.** This can be accomplished by either neuraxial or general anesthesia.
- 2. One should have vasodilators readily available (ie, nitroglycerin, nitroprusside).**

Post-operative

- 1. Routine PACU monitoring with special attention to respiratory status and signs and symptoms of autonomic dysreflexia.**

BIBLIOGRAPHY

- 1. Lin, V., et al. *Spinal Cord Medicine: Principles and Practice*. New York: Demos Medical Publishing, 2003, pp 113–21, 189.
- 2. Stoelting, R. et al. *Anesthesia and Co-Existing Disease, 4th ed.* Philadelphia: Churchill Livingstone, 2002, pp 278–82.
- 3. Barash, P., Cullen, B.F., and Stoelting, R.K. *Clinical Anesthesia, 5th ed.* Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1272–4.
- 4. Cuccurullo, S. ed. *Physical Medicine and Rehabilitation Board Review*. New York: Demos Medical Publishing, 2004, pp 504, 512–15, 593.

48. Acute Renal Failure (ARF)^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

An 83-year-old male is post-operative day one after a coronary artery bypass graft (CABG). His urine has decreased to 15 ml/hour for the last 2 hours. Is this oliguria? What are the possible causes? How would you diagnose and treat this finding? How do you determine between prerenal, renal, and postrenal causes of ARF?

CLINICAL ISSUES

Definition of ARF

1. "Deterioration of renal function over a period of hours to days, resulting in failure of the kidneys to excrete nitrogenous waste products and to maintain fluid and electrolyte homeostasis"¹
2. Increase in serum creatinine concentrations of more than 0.5mg/dL compared with the baseline value
3. Often synonymous with acute tubular necrosis
4. A 50% decrease in the calculated creatinine clearance
5. Decreased renal function that results in the need for dialysis
6. Oliguric (urine output <400 mL/day or <0.5 mL/kg/hr for 6 hours) or nonoliguric (urine output >400 mL/day)

Mortality

1. Isolated ARF has a <10% mortality.
2. The mortality can exceed 50% and be as high as 90% when associated with multiple organ failure, severe hypotension, or respiratory failure.

Risk Factors

1. Co-existing renal disease
2. Advanced age
3. Congestive heart failure
4. Symptomatic cardiovascular disease
5. Major operative procedures
 - a. CABG
 - b. Abdominal aneurysm repair
6. Sepsis
7. Multiple organ system dysfunction
8. Iatrogenic causes
 - a. Inadequate fluid replacement

- b. Delayed treatment of sepsis
 - c. Nephrotoxic drugs or dyes
9. Hypotension

Etiology

1. Prerenal: (usually reversible with an improved circulatory status)
 - a. Absolute decrease in renal blood flow
 - i. Dehydration
 - ii. Acute hemorrhage
 - (1) Hypovolemia
 - (2) Hypotension
 - iii. Gastrointestinal (GI) fluid loss
 - iv. Trauma
 - v. Surgery
 - (1) Mechanical restriction of renal blood flow by aortic or renal artery clamping
 - vi. Burns
 - vii. Renal artery or vein thrombosis
 - viii. Excessive diuretic use
 - b. Relative decrease in renal blood volume
 - i. Septic shock
 - ii. Hepatic failure
 - iii. Allergic reaction/transfusion reaction
 - iv. Vasoconstriction
 - v. Congestive heart failure
 - vi. Decreased cardiac output
2. Renal: (most serious form, which often requires hemodialysis to treat)
 - a. Acute glomerulonephritis
 - i. Goodpasture's syndrome
 - ii. Wegener's granulomatosis
 - iii. Acute lupus nephritis with systemic lupus erythematosus
 - iv. Post-infectious glomerulonephritis
 - v. Berger's disease
 - vi. Henoch-Schonlein purpura
 - vii. Drugs
 - (1) Allopurinol
 - (2) Hydralazine
 - (3) Rifampin
 - b. Interstitial nephritis
 - i. Pyelonephritis
 - ii. Sarcoidosis

Table 48.1. Normal Values

Glomerular Filtration Rate (GFR)	Blood Urea Nitrogen (BUN)	10–20 mg/dL
	Creatinine (CR)	0.5–1.5 mg/dL
	Creatinine Clearance (CRCL)	85–125 mL/min (Women) 95–140 mL/min (Men)
Tubular Function	Specific Gravity (SG)	1.002–1.030
	Osmolality (mOsm/kg)	50–1400
	Urine Sodium (mEq/L/day)	130–260

Adapted from Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002; Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005; Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.

Table 48.2. Prerenal versus Renal Lab Values

	Prerenal	Renal
Urine Output (cc/kg/hr)	<0.5	<0.5
Urine Sodium (mEq/L)	<25	>35
Fractional Excretion of Sodium (%) (FENa)	<1	>1
Urine Osmolality (mOsm/kg)	>500	<350
Urine/Plasma Creatinine	>30:1	<10:1
Urine/Plasma Osmolality	>1.8:1	<1.1:1
Urine/Plasma Urea	>20:1	<3:1
BUN/CR	>20:1	<10:1

Adapted from Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002; Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005; Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.

iii. Allergic drug reaction (non-steroidal anti-inflammatory agents [NSAIDs], aspirin)

c. Acute tubular necrosis (most common in surgical patients)

i. Ischemia (50% of cases)

(1) Episode of hypotension

(2) Shock (cardiogenic, septic, hemorrhagic)

ii. Embolic event or aortic cross clamp

iii. Mechanical damage

(1) Trauma

iv. Nephrotoxic drugs (35% of cases)

(1) Radiographic contrast dyes

(2) Aminoglycoside antibiotics

(3) Anesthetic agents

(4) NSAIDs

(5) Chemotherapeutic agents

v. Solvents

vi. Myoglobinuria/rhabdomyolysis/hemolysis

vii. Transfusion reaction

viii. Hypoxia

d. Vasculitis

e. Chronic kidney disease

i. Diabetes

ii. Hypertension

f. Multiple myeloma

3. Postrenal: (found in <5% of the cases. It is usually reversible with the treatment of the obstruction. The obstruction is usually secondary to stones, strictures, infection, clots, tumors, surgical ligation, and edema. The obstruction can also be due to medications that interfere with normal bladder emptying).

a. Upper urinary tract obstruction

i. Renal pelvis

ii. Ureter

b. Lower urinary tract obstruction

i. Bladder outlet

ii. Foley catheter

iii. Urethral

iv. Prostatic hypertrophy or cancer

v. Cervical cancer

Complications of ARF

1. Neurologic

a. Confusion

b. Asterixis

c. Somnolence

d. Seizures

2. Cardiovascular

a. Hypertension/hypotension

b. Congestive heart failure

c. Pulmonary edema

d. Cardiac dysrhythmias

3. Gastrointestinal

a. Anorexia

b. Nausea/vomiting

- c. Ileus
- d. GI bleeding
- 4. Infection
 - a. Respiratory
 - b. Urinary tract
- 5. Metabolic derangements
 - a. Metabolic acidosis
 - b. Hyperkalemia



KO TREATMENT PLAN

The sample patient can be considered to be oliguric if this low urine output continues. A urine output of 15 mL/hr over 24 hours is only 360 mL/day (less than the designated 400 mL/day for the diagnosis of oliguria). It is important to diagnose and treat the cause of the ARF.

1. Check vital signs.
2. Determine whether the ARF is prerenal, renal, or postrenal.
3. Determine and treat the underlying cause.
 - a. Prerenal
 - i. Look for signs of decreased cardiac output, shock, or hypovolemia by assessing the intravascular volume status.
 - ii. Central venous pressure (CVP)
 - iii. Pulmonary artery catheter (PAC)
 - iv. Transesophageal echocardiography
 - v. Fluid challenge
 - b. Renal
 - i. Check urine for blood or myoglobin.
 - ii. Check the electrolytes.

- iii. Check BUN/CR.
- iv. Check FENa.
- v. Send a blood and urine specimen for sodium, osmolality, creatine, and urea.
- vi. Discontinue nephrotoxic drugs.
- c. Postrenal
 - i. Renal ultrasound is the best to diagnose postrenal obstruction.
 - ii. Check the Foley catheter.
 - iii. Palpate the bladder.
4. Resuscitate the patient until he or she is normotensive with a normal cardiac output.
 - a. Fluids
 - i. Guided by a Foley catheter and central venous pressure (CVP) monitoring
 - b. Inotropes
 - i. Norepinephrine IV
 - ii. Dobutamine IV
5. Mannitol/dopamine/furosemide 100–200 mg IV to maintain urine output, because a nonoliguric ARF is usually considered better than an oliguric acute renal failure.
6. Hemodialysis every 2–4 days as needed

BIBLIOGRAPHY

1. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 356–60.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2178–81 and 2187–9.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1017–8.

49. Chronic Renal Failure (CRF)^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 43-year-old male presents to the operating room for a revision of his arteriovenous dialysis graft. He missed his dialysis yesterday because he did not have transportation. His last dialysis was 4 days ago. Does he need dialysis prior to surgery? What lab values would you like? What if his potassium is 6.4 mEq/L? What would you do? Do you want an EKG? Why? What if his hematocrit is 27%? What would you do? Does he need a blood transfusion? When you do the surgery, what is your choice of anesthetic?

CLINICAL ISSUES

Definitions

1. Uremic syndrome
 - a. Most extreme form of CRF
 - b. Glomerular filtration rate (GFR) <10% of normal
 - c. Inability of the kidneys to regulate volume and composition of the extracellular fluid
 - d. Inability of the kidneys to effectively excrete waste products

49. Chronic Renal Failure

- e. Leads to multiple system organ dysfunction.
 - f. Requires frequent or continuous dialysis.
2. **End-stage renal disease (ESRD): a clinical syndrome characterized by multiple organ dysfunction that would prove fatal without dialysis.**
- a. GFR 10–25% of normal.
3. Renal insufficiency
- a. GFR 26–40% of normal
 - b. Abnormal creatinine and blood urea nitrogen (BUN) values
 - c. The only symptom is nocturia.
4. Decreased renal reserve
- a. GFR 41–75% of normal
 - b. Asymptomatic
 - c. Normal blood levels of creatinine and urea

Complications of Uremic Syndrome

1. Electrolyte abnormalities
 - a. Hyponatremia
 - b. Hyperkalemia
 - i. Peaked T waves, prolongation of the QRS complex and PR interval, heart block, and ventricular fibrillation can be seen on the EKG.
 - ii. If the serum potassium is greater than 6.5 mEq/L or if the EKG changes are present, the hyperkalemia should be treated.
 - iii. Treat the hyperkalemia with IV calcium, glucose, insulin, bicarbonate, and/or emergency dialysis.
 - c. Hyper- or hypocalcemia
 - d. Hyperphosphatemia
 - e. Hypermagnesemia
 - f. Metabolic acidosis
 - i. Treat a pH <7.3 with IV bicarbonate, assuming the patient has appropriate ventilation.
2. Cardiovascular
 - a. Heart failure secondary to hypervolemia
 - b. Hypertension
 - i. Treat with diuretics in predialysis patients.
 - ii. Treat with hemodialysis.
 - c. Pericarditis/cardiac tamponade
 - d. Myocardial dysfunction
 - e. Arrhythmias
 - f. Coronary artery disease
3. Respiratory
 - a. Pulmonary edema
 - b. Central hyperventilation
4. Hematologic
 - a. Normochromic, normocytic anemia
 - i. Treat with recombinant human erythropoietin (epoetin alpha/Epogen/Procrit).
 - ii. Parenteral iron
 - iii. Avoid blood transfusions if possible.
 - b. Platelet dysfunction
 - c. Coagulopathy
 - d. Uremic bleeding

- i. Treat with cryoprecipitate to provide factor VIII and/or DDAVP (desmopressin).
5. Gastrointestinal (GI)
- a. Delayed gastric emptying
 - b. Anorexia
 - c. Nausea/vomiting
 - d. Hemorrhage
6. Neuromuscular
- a. Encephalopathy
 - b. Seizures
 - c. Tremors
 - d. Myoclonus
 - e. Polyneuropathy
 - f. Autonomic dysfunction
7. Endocrine/metabolism
- a. Osteodystrophy
 - i. Treat with antacids to bind the phosphorus in the GI tract, oral calcium supplements, and vitamin D therapy.
 - b. Glucose intolerance
 - c. Increased atherosclerosis
 - d. Fatigue
 - e. Pruritus

Associated Causes

1. Systemic hypertension
2. Glomerulopathies
 - a. Primary
 - b. Associated with a disease
 - i. Diabetes mellitus
 - ii. Amyloidosis
 - iii. Systemic lupus erythematosus
 - iv. Wegener's granulomatosis
3. Tubulointerstitial disease
 - a. Sarcoidosis
4. Hereditary disease
 - a. Polycystic kidney disease
5. Renal vascular disease
6. Obstructive uropathy
7. Human immunodeficiency virus (HIV)



KO TREATMENT PLAN

Pre-operative

1. This surgery is not an emergency. He very likely has a temporary dialysis catheter in place.
2. This patient should have dialysis prior to this surgery. The hemodialysis should correct his elevated potassium.
3. While waiting for dialysis, the patient should have a 12-lead EKG to look for any changes secondary to the hyperkalemia. If he has EKG changes, his hyperkalemia should be treated immediately and not wait for his dialysis.
 - a. 10 mL of 10% calcium chloride IV infused over 10 minutes

- b. Glucose (D10W) IV and 5–10 units of regular insulin for every 25–50 grams of glucose given
 - c. Sodium bicarbonate 50–100 mEq IV over 5–10 minutes
 - d. β_2 agonist (albuterol)
4. His hematocrit may be low secondary to hemodilution from fluid overload due to his missing his dialysis. Review any old records. Repeat a hematocrit after his dialysis. These patients typically tolerate a lower hematocrit. I would not transfuse for a hematocrit of 27% if the patient is normally anemic.

Intra-operative

1. This is a low risk surgery. It should be able to be done under regional technique or local block with sedation.
2. If the patient requires general anesthesia, remember that he is considered “full stomach” secondary to his associated delayed gastric emptying. If he has had hemodialysis and has a normalized potassium level, a rapid sequence induction with succinylcholine can be safely done. His potassium will increase only about 0.5 mEq/L, the same as in normal healthy patients. If the patient was an emergent surgery and was not able to have his dialysis, then succinylcholine should be avoided.

TKO: Pick one induction agent and defend it. Do not give a list of all of the induction agents that can be used. Don't say “I could ...”, say “I would use this drug and this is why.” Just pick one drug and defend it.

3. Safe induction agents
 - a. You may choose etomidate because of the presumed associated cardiovascular issues with this patient and the fact that renal failure does not seem to affect its clinical effects.
 - b. You may choose propofol because of the uremia-associated nausea/vomiting, decreased gastric emptying,

and the fact that it also has an unchanged clinical profile in renal failure patients.

- c. Or, you may choose to explain why you would give a decreased dose of thiopental secondary to the CRF patient's reduced protein binding, and therefore increased free fraction of available thiopental.
 - d. Ketamine is also a good choice because poor renal function does not tend to alter its pharmacokinetic or clinical profile.
4. Intraoperative opioids
 - a. Fentanyl is an excellent choice and is recommended secondary to its primary liver metabolism and lack of active metabolites.
 - b. Remifentanyl is also good secondary to its rapid metabolism by blood and tissue esterases.
 - c. A single small dose of morphine is acceptable, but chronic dosing is not acceptable, secondary to the accumulation of its metabolite, 6- glucuronide, which leads to prolonged respiratory depression and sedation.
 - d. Meperidine has a metabolite, normeperidine, which is neurotoxic and can lead to convulsions when used in a renal failure patient.
 5. All volatile anesthetics can be used safely, but sevoflurane does have a theoretical risk secondary to the plasma inorganic fluoride concentrations approaching nephrotoxic levels (50 $\mu\text{mol/L}$) after prolonged inhalation. However, no evidence of gross changes in renal function has been found in humans.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2181–7.
2. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 344–9.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 197, and 1018–22.

50. Dialysis^{1,2,3,4,5}

Lisa L. Bethea, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 49-year-old male with type II diabetes mellitus, hypertension, end-stage renal disease, and chronic obstructive pulmonary disease presents for a laparoscopic hemicolectomy for colon polyps. The patient usually receives hemodialysis

three days per week. What are the major concerns in the pre-, intra-, and post-operative periods? If surgery is scheduled on the day of dialysis, should the patient receive dialysis prior to the surgery? How will this affect your anesthetic management before, during, and after surgery?

CLINICAL ISSUES

Definition: dialysis is used as a substitute for the kidney when the kidney cannot perform its usual functions, including fluid and electrolyte balance and toxin/drug removal.¹

Modalities

1. Hemodialysis
 - a. Removes both solutes and fluids from the body.
 - b. This is done by diffusive clearance and ultrafiltration, respectively.¹
 - c. Solute clearance is determined by the molecular size of the solutes, concentration gradient, membrane surface area, membrane permeability, and flow rate of blood and dialysate.¹
 - d. Ultrafiltration is determined by the transmembrane pressure.¹
2. Peritoneal dialysis
 - a. Removal of fluids and solutes through use of patient's own peritoneal membrane.¹
 - b. Solute removal is via diffusion across a concentration gradient whereas fluid removal is via osmotic ultrafiltration.¹

Indications for Dialysis^{1,5}

1. Absolute indications
 - a. Metabolic acidosis
 - b. Electrolyte abnormalities
 - i. Hyperkalemia
 - ii. Hypercalcemia
 - iii. Hyperphosphatemia
 - iv. Hypermagnesemia
 - v. Hyperuricemia
 - c. Intoxication/drug overdose: this is not a complete list – just examples of some of the more commonly used drugs for anesthesia that are dialyzable. Propofol, fentanyl, and rocuronium are not dialyzable.
 - i. Acetaminophen
 - ii. Aspirin
 - iii. Antibiotics
 - (1) Amoxicillin
 - (2) Ampicillin
 - (3) Cefazolin
 - (4) Cefepime
 - (5) Cefotaxime
 - (6) Cefoxitin
 - (7) Penicillin
 - iv. Acyclovir
 - v. Allopurinol
 - vi. Most β -blockers
 - (1) Atenolol
 - (2) Esmolol
 - (3) Metoprolol
 - vii. Most angiotensin-converting enzyme inhibitors (ACEIs)

- (1) Captopril
- (2) Enalapril
- (3) Lisinopril
- viii. Vitamins
 - (1) Ascorbic acid
 - (2) Edate calcium
 - (3) Folic acid
- ix. Mannitol
- x. Metformin
- xi. Nitroprusside
- xii. Phenobarbitol
- d. Fluid overload/pulmonary edema
- e. Uremia
2. Relative indications^{1,5}
 - a. Blood urea nitrogen (BUN) and creatinine (Cr) values should be used only as a guide.
 - i. BUN >100 mg/dL or Cr >10 mg/dL values are used when initiating chronic dialysis.
 - b. Glomerular filtration rate (GFR) less than 10 mL/min/1.73 m²
 - i. Less than 15 mL/min/1.73 m² in diabetics
3. General
 - a. Acute renal failure
 - b. Chronic renal failure
 - c. End-stage renal disease
 - d. Uncontrolled hypertension

Access

1. Hemodialysis
 - a. Vascular access
 - i. Native arteriovenous fistula (AVF)
 - (1) Connection between the radial artery or brachial artery and the cephalic or basilic veins¹
 - (2) This allows blood flow rates of 400 ml/minute.¹
 - (3) Matures in 6–8 weeks.¹
 - (4) Longest survival after maturation
 - (5) Fewest complications
 - ii. Artificial arteriovenous graft (AVG)
 - (1) Synthetic graft used when AVF cannot be placed.^{1,5}
 - (2) Easier to place
 - (3) Higher rate of complications, including infection, stenosis, and thrombosis^{1,5}
 - iii. Percutaneous double-lumen catheter
 - (1) Placed in the internal jugular or femoral vein and usually tunneled under the skin^{1,5}
 - (2) Catheters have been placed in the subclavian vein in the past but they were found to be associated with central venous stenosis.^{1,5}
2. Peritoneal dialysis
 - a. Access via catheter in the abdominal cavity, placed between the anterior abdominal wall and the omentum/bowel loops⁵.
 - b. Catheter is either a silicone or polyurethane material.
 - c. Catheter is cuffed.

Complications

1. Hemodialysis

- a. Vascular-access problems^{2,3}
 - i. Infection
 - ii. Bleeding from the puncture site
 - iii. Thrombosis
 - iv. Stenosis
 - v. Aneurysm
- b. Hypotension^{2,3}
 - i. Decreased intravascular volume
 - ii. Hemorrhage
 - iii. Septicemia
 - iv. Cardiogenic shock
 - v. Dysrhythmia
 - vi. Electrolyte abnormalities
 - (1) Hypo-/hyperkalemia
 - (2) Hypo-/hypercalcemia
 - (3) Hypermagnesemia
 - vii. Anaphylactoid reaction
 - viii. Air embolism
- c. Hemorrhage with resultant cardiac symptoms
 - i. Symptomatic angina
 - ii. Congestive heart failure
 - iii. Patients are typically treated with epoetin prior to dialysis to prevent severe anemia.^{2,3}
- d. Gastrointestinal bleed^{2,3}
 - i. Angiodysplasia
 - ii. Peptic ulcer disease
- e. Cardiovascular
 - i. Acute pericardial tamponade^{2,3}
 - ii. Chest pain
 - (1) Most dialysis patients have coronary artery disease; therefore, ischemic origin of chest pain must be ruled out.^{2,3}
- f. Electrolyte abnormalities
- g. Shortness of breath due to volume overload^{2,3}
- h. Neurologic dysfunction^{2,3}
 - i. Disequilibrium syndrome: headache, malaise, nausea, vomiting, and muscle cramps. This may progress to seizures and coma.
 - ii. Cerebrovascular accident
 - iii. Subdural hematoma
 - iv. Hypertensive encephalopathy
 - v. Electrolyte abnormalities
 - (1) Hypo-/hyponatremia
 - (2) Hypercalcemia
 - (3) Hypermagnesemia
 - (4) Hypo-/hyperglycemia

2. Peritoneal dialysis^{2,3}

- a. Peritonitis is due to bacterial contamination, usually from a *Staphylococcus* species.
- b. Catheter contamination
- c. Catheter leakage
- d. Abdominal wall or inguinal hernia due to the associated increased intra-abdominal pressure.
- e. Infection of catheter exit site

- f. Pneumoperitoneum from air that enters during exchange therapy.
- g. Mechanical problems
 - i. Inability to drain dialysate completely because of catheter kinking or obstruction
- h. Volume overload may lead to worsening blood pressure control or acute pulmonary edema.
- i. Volume depletion
 - i. Poor oral intake
 - ii. Increased gastrointestinal loss
- j. Metabolic problems (e.g., hyperglycemia) from absorption of glucose from the dialysate fluid (hyperosmolar solution)

TKO: Dialysis patients have an increased risk of morbidity and mortality.⁴ This risk is further enhanced in the setting of surgery.

Increased Risk of Morbidity and Mortality

1. Higher likelihood of coronary artery disease and cardiac abnormalities
2. **Electrolyte and fluid imbalances, especially in the presence of worsening kidney function**
 - a. Patients with renal failure tend to have fluid overload/pulmonary edema as well as increases or decreases in potassium, magnesium, calcium, and/or glucose levels.
3. Problems with drug excretion and metabolism, which interferes with anesthetic dosing
 - a. Drugs that are renally metabolized/excreted take longer to be removed from the body and therefore can build up and cause toxicity.
 - b. See following for specific details.
4. Bleeding complications
 - a. Impaired platelet function
 - i. Prolonged bleeding time
 - ii. Platelet dysfunction is most often seen in the presence of uremia because toxins are not properly removed from the body.
5. Blood pressure fluctuations, depending on when dialysis was last received
 - a. If too much fluid has been removed, then the blood pressure tends to be low from volume depletion.
 - b. If too little fluid has been removed or it has been days since the last treatment, then the blood pressure is elevated from volume overload.
6. **Require more medical management: prolonged ICU/hospital time and mechanical ventilation.**⁴



KO TREATMENT PLAN

Pre-operative

1. Start with a history.
 - a. Complaint of chest pain or shortness of breath
 - b. New or worsening headache, nausea/vomiting, malaise

50. Dialysis

- c. Medical problems and medications currently taking
 - d. Dialysis schedule and when last received
 - e. Dry weight
2. Physical exam
- a. Check vital signs: blood pressure, heart rate, respiratory rate, temperature, and oxygen saturation.
 - b. Listen to the patient's heart and lungs.
 - c. Look for signs of fluid overload: extremity edema, jugular venous distension, and use of accessory muscles of respiration.
 - d. Assess for signs of volume depletion: poor skin turgor, dry mucous membranes, hypotension, and tachycardia.
3. Labs
- a. Laboratory values must be monitored closely prior to any surgical intervention.⁴ The patient's baseline electrolyte, hemoglobin, coagulation panel, and platelet values are important.
 - i. Check prior lab values to assess patient trends. If there are significant increases or decreases, further treatment may be necessary.
 - ii. Potassium less than 6.0 mEq/L is usually acceptable unless there are cardiographic changes on the electrocardiogram.⁴
 - (1) Peaked T waves
 - (2) Prolonged PR interval
 - (3) Widened QRS complex
 - iii. Hemoglobin concentrations in the range of 12–13 g/dL are preferred.
 - (1) If the procedure is nonemergent, the patient can receive erythropoietin to help stimulate red blood cell production.
 - iv. Glucose
 - (1) Must consider NPO status, decreased activity level, and co-morbid conditions that might affect glucose metabolism.⁴
 - v. Bleeding problems must be corrected with blood transfusions, platelets, desmopressin, or cryoprecipitate.
 - (1) Heparin should be avoided in the days leading up to surgery.⁴
4. Cardiovascular evaluation will assess for coronary artery disease (CAD) and myocardial dysfunction.⁴
- a. No further evaluation is needed for low-risk patients; no history of CAD, myocardial ischemia, or chest pain.
 - b. An EKG can help determine whether cardiovascular effects of hyperkalemia are present as well as assess for arrhythmias.
 - i. Baseline EKG will help evaluate the patient for changes intra-operatively and post-operatively.
 - c. Echocardiogram and/or stress test is used to evaluate myocardial function and determine whether ischemia develops in the presence of stress.⁴
 - i. This is a non-invasive way of determining whether the patient can handle the stress of surgery and help determine whether further procedures (angiography, cardiac catheterization) are needed.
5. Nutrition status is the key to good healing.
- a. Check the patient's protein catabolic rate and/or albumin concentration.⁴
 - b. Eliminate drugs that hinder appetite.
 - c. Give drugs that inhibit or treat gastroparesis.⁴
6. Good blood pressure control leading up to the surgery is important. This will allow for less labile pressures, hopefully preventing instances of hypertension and hypotension during the surgery.
- a. Hypotension is usually due to too much fluid removed during dialysis or inappropriate dosages of anti-hypertensive medications.
 - i. Adjust medication dosages as needed.
 - ii. This may also be treated with judicious fluid boluses.
 - iii. If this does not help, vasopressors (phenylephrine, ephedrine) may be needed.
 - b. Hypertension should be treated with anti-hypertensive agents such as beta-blockers, hydralazine, diltiazem, and nitroglycerin.
7. A more intensive dialysis regimen prior to surgery may be beneficial.
- a. Patients receiving intermittent hemodialysis may benefit from daily dialysis several days prior to surgery (more beneficial for cardiac surgery).⁴
 - b. Those receiving peritoneal dialysis may benefit from an additional exchange every day for the entire week leading up to surgery.⁴
8. Dialysis should be provided the day prior to surgery.⁴
- a. Dry weight
 - b. Ensure normalization of electrolytes.
 - c. Optimize fluid status.
 - d. Having dialysis on the day of surgery could cause volume depletion if too much fluid is removed. This would cause significant hypotension during surgery, which is worsened in the presence of vasodilatation already produced by anesthesia.
9. Antibiotics, if necessary, should be adjusted for renal function.
10. Intravenous access⁴
- a. Avoid any sites that may be needed for dialysis access in the future.
 - b. Subclavian access should be avoided because of the increased risk for stenosis.
 - c. Awareness of the patient's vascular anatomy is important.
- ### Intra-operative
1. In patients with renal failure, drugs/anesthetic agents are not metabolized the same. Maintain caution so as to avoid overdosing and/or toxic levels.
- a. Pre-induction agents
 - i. Benzodiazepines have increased protein binding, which may lead to increased free fraction in the presence of renal failure.⁴
 - ii. Smaller doses should be used.
 - b. Induction agents

- i. Thiopental must be used with caution. Its free fraction is doubled in renal failure, which leads to enhanced clinical effects.⁴ Lower dosages are recommended.
- ii. Propofol is metabolized by the liver and is therefore not contraindicated in renal failure.
- c. Neuromuscular blockers
 - i. Depolarizing agents
 - (1) Succinylcholine can cause hyperkalemia, which can be a significant problem in patients with renal failure.⁴
 - (a) Succinylcholine raises serum potassium about 0.5 mEq/L.
 - (b) This increase is not significant in normal patients but can be potentially dangerous in the presence of renal failure.⁴
 - (c) Typically, if the serum potassium is less than 5 mEq/L, then succinylcholine is acceptable to use.⁴
 - ii. Neuromuscular blocking agents
 - (1) Pancuronium is a long-acting non-depolarizer and is renally excreted. It should be avoided in these patients due to the risk of prolonged blockade.⁴
 - (2) Atracurium is excreted via extra-renal mechanisms (Hoffman elimination) and is therefore a preferred neuromuscular blocking agent.⁴
- 2. Monitors
 - a. Standard ASA monitors including heart rate, blood pressure, pulse oximetry, and temperature
 - b. Arterial line
 - i. Watch for excessive alterations in blood pressure.
 - ii. Glucose monitoring helps maintain a glucose level within normal limits.
- 3. Maintenance
 - a. Anesthesia can be maintained with isoflurane, O₂, and air.
 - b. N₂O is contraindicated in laparoscopic procedures because it can diffuse into spaces and cause distension of the bowel. This would hinder visualization for the surgeon.
 - c. Isoflurane may decrease renal blood flow but not more or less than any other anesthetic agent. There are no contraindications in renal failure.
 - d. All volatile anesthetics can be used safely, but sevoflurane does have a theoretical risk secondary to the plasma inorganic fluoride concentrations approaching nephrotoxic levels (50 μmol/L) after prolonged inhalation. However, no evidence of gross changes in renal function has been found in humans.
 - e. Opiates can be used intra-operatively for pain control.
 - i. Fentanyl is preferred because of its short redistribution phase and lack of active metabolites.⁴
- 4. IV Fluids
 - a. Normal saline is preferred because it does not contain additional electrolytes like potassium, calcium, and magnesium, which are found in lactated Ringer's solution.
 - b. Providing fluids with additional electrolytes can cause electrolyte imbalances post-operatively.

c. Judicious amounts of fluids should be given to prevent fluid overload.

Post-operative

1. Recovery depends on the nature of the surgery. Patients do not need to go to the intensive care unit (ICU) unless the procedure itself requires this.
2. Blood pressure monitoring must continue in the post-operative period.
 - a. Treat hypotension with vasopressors (phenylephrine, norepinephrine, and vasopressin) as necessary.⁴
 - b. Avoid giving too much fluid.
 - c. If vasopressors are needed, an ICU setting is preferred.
 - d. Treat hypertension with anti-hypertensive agents such as hydralazine, beta-blockers, or nitroglycerin.
 - i. If the hypertension cannot be controlled, dialysis may be required to get rid of excess fluid.⁴
3. Labs should be checked immediately post-operatively and daily to assess electrolyte abnormalities. Follow glucose levels closely, especially if the patient has a NPO status.⁴
4. Fluid status can be evaluated by following the patient's dry weight and/or monitoring urine output if the patient still produces urine.
5. **Dialysis should be resumed post-operatively. The timing depends on patient fluid status, electrolyte balance, and hemodynamic stability. When dialysis is used in the post-operative period, heparin must be avoided for 24–48 hours.⁴**
6. Analgesics, like opiates and acetaminophen, may be used for post-operative pain relief.⁴
 - a. Acetaminophen has no renal effects and can be used without adjustments.
 - b. Because of its short redistribution phase and lack of active metabolites, fentanyl is a good choice.⁴
 - c. Morphine's sedative effects are prolonged in the presence of renal failure because its metabolites are renally excreted.⁴ Morphine should therefore be used with caution.
 - d. Meperidine is broken down to its active metabolite, normeperidine, whose half-life is prolonged in renal failure. Build-up of this metabolite can lead to myoclonic jerks, seizures, and respiratory depression.⁴ Therefore, meperidine should not be used.

BIBLIOGRAPHY

1. Goldman L, Ausillo D. *Cecil's Medicine*, 23rd ed. Philadelphia: Saunders Elsevier, 2007, pp 936–41.
2. Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 6th ed. Philadelphia: Mosby Elsevier, 2006, pp 1546–54.
3. Ferri FF. *Practical Guide to the Care of the Medical Patient*, 7th ed. Philadelphia: Elsevier Mosby, 2007, pp 682–6.
4. Soundararajan R, Golper TA. Medical management of the dialysis patient undergoing surgery. Uptodate.com. 2007.
5. Humes HD. *Kelley's Textbook of Internal Medicine*, 4th ed. Philadelphia: Lippincott Williams and Wilkins, 2000, pp 1290–7.

51. Transurethral Resection of the Prostate (TURP) and TURP Syndrome^{1,2,3}

Lisa L. Bethea, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 55-year-old male with hypertension, chronic obstructive pulmonary disease (COPD), and benign prostatic hypertrophy (BPH) presents for a transurethral resection of the prostate (TURP). Pre-operative vital signs include a blood pressure of 145/70 mm Hg, a pulse of 90, respiration rate of 20, and a temperature of 36.4°C. The procedure is performed under spinal anesthesia. After placement of the spinal and proper positioning of the patient, the procedure is begun without incident. About 1 hour into the procedure, the patient complains of a headache and difficulty breathing. The blood pressure is noted to be 90/50 mm Hg. Other vital signs include respirations of 25, heart rate of 65, and an oxygen saturation of 97% on 3L nasal cannula. What are your major concerns? What tests should you order? Should the procedure have been done under general anesthesia instead? How should the patient's headache and breathing difficulties be treated?

CLINICAL ISSUES

Definition

1. TURP is the resection of enlarged prostate tissue via the urethra using cystoscopy.¹ It is used for the treatment of BPH. The procedure involves distending the bladder with irrigation fluid and cauterizing the prostatic tissue with electrical current.² The irrigation fluid washes out the debris and helps keep the bladder distended for improved visualization of the prostate.³ Several irrigation fluids are available, including glycine, sorbitol, mannitol, glucose, and cytal.¹ The irrigating solution must be isotonic, sterile, nontoxic, and not conduct electricity.¹
2. TURP syndrome is a group of signs/symptoms that may occur during a TURP as result of the absorption of the irrigating fluids into the prostatic veins.³ This can lead to hyponatremia, hypo-osmolality, and metabolic acidosis.³ Incidence of TURP syndrome: 2% of patients undergoing TURP²

Risk Factors for TURP Syndrome (Increased Risk of Fluid Absorption²)

1. Prostate >45 grams

2. Prolonged resection time greater than 90 minutes
3. High inflow irrigating fluid pressure
4. Non-continuous irrigating fluid
5. Pre-operative hyponatremia
6. Smoking history

Triggers for TURP Syndrome

Absorption of irrigating fluids is the primary cause, which can lead to water intoxication and resultant hyponatremia.¹ The resection time has a profound affect on the amount of fluids absorbed (10–30 mL/min).¹

Clinical Features of TURP Syndrome

The symptoms usually present with prolonged procedures, increased irrigating fluid or bladder pressure, and/or a serum sodium concentration measured less than 120 mEq/L.²

1. Neurologic^{1, 3}
 - a. Early: headache, nausea, irritability, apprehension, confusion
 - b. Late: visual disturbances, somnolence, seizure, coma, death
2. Cardiovascular^{1, 3}
 - a. Early: hypertension, reflex bradycardia
 - b. Intermediate: negative inotropy, hypotension, dysrhythmias
 - c. Late: widened QRS, ST segment elevations, ventricular arrhythmias, congestive heart failure (CHF), cardiovascular collapse
3. Respiratory^{1, 3}
 - a. Early: tachypnea, oxygen desaturation
 - b. Late: hypoxemia, pulmonary edema, respiratory arrest
4. Metabolic^{1, 3}
 - a. Early: hyponatremia, hyperglycemia, hyperammonemia, hypo-osmolality
 - b. Late: metabolic acidosis
5. Other^{1, 3}
 - a. Renal failure
 - b. Hemolysis

TKO: TURP syndrome is characterized by shifts in intravascular fluid volume. This can lead to significant electrolyte

51. Transurethral Resection of the Prostate (TURP)

abnormalities, which can contribute to the above symptoms. If the patient becomes symptomatic, stop the procedure, provide supportive care, and check a serum sodium level as well as serum osmolality.

Other Complications of a TURP

1. Bladder perforation¹
 - a. Intraperitoneal versus extraperitoneal
 - b. Signs/symptoms include abdominal pain, shoulder pain, pallor, sweating, nausea/vomiting, and hypotension.
 - c. Decreased return of irrigating fluid from bladder is an early sign.
 - d. Diagnosis with cystourethrography
 - e. Treatment is a suprapubic cystostomy.
2. Bleeding¹
 - a. Dependent on the prostate size, 20–50 mL/g of prostate
 - b. Dependent on resection time, 2–5 mL/min of the resection time
3. Coagulopathy¹
 - a. Clinical (1% of cases) versus subclinical (6% of cases)
 - b. Causes: fibrinolysis, dilution of coagulation factors/platelets, disseminated intravascular coagulation (DIC)
4. Transient bacteremia and septicemia¹
 - a. Due to prostatic bacteria entering through the prostatic sinuses
 - b. Treat with antibiotics and supportive care.
5. Hypothermia¹
 - a. Occurs secondary to the use of irrigating fluids at room temperature.
 - b. Warming fluids can prevent this problem.
6. Toxicity of irrigating fluids^{1, 3}
 - a. Glycine toxicity: transient blindness
 - b. Ammonia toxicity: neurologic sequelae, nausea, vomiting, convulsions, coma



KO TREATMENT PLAN

Pre-operative

1. Formulate an appropriate anesthetic plan; regional anesthesia is preferred because
 - a. You are able to monitor the patient's mental status.¹
 - b. Early detection of hyponatremia and TURP syndrome¹
 - c. Early detection of other complications of the TURP¹
 - d. Decreased rate of deep venous thrombosis (DVT)¹
 - e. Decreased blood loss due to the sympathetic block and decreased central venous and peripheral venous pressures.¹
 - f. Better post-operative pain control and less narcotics required.¹
2. Correct pre-operative electrolyte abnormalities.

TKO: If regional anesthesia is performed, you must ensure at least a T9–10 level to have adequate coverage of the bladder

(T11–12) and prostate (T11–12) as well as coverage of the pelvic floor and perineum (S2–S4).¹ Blockage above T9 is not recommended because it will obscure any symptoms that may develop if a bladder perforation were to occur.¹

Intra-operative

1. Monitor the patient closely for any signs of mental status changes.
2. Note any significant alterations in vital signs and provide supportive measures.
3. Have the surgeon stop or expedite the procedure as soon as possible if any complications develop.
4. Restrict intravenous fluids.
5. Use loop diuretics (furosemide) to get rid of excess free water.¹
6. Check the serum sodium level. If severe hyponatremia develops with neurologic symptoms, treat with 3% saline IV. Replace the sodium deficit slowly as to avoid central pontine myelinolysis.
 - a. Correction of the sodium deficit: dose = weight (kg) × (140 – current sodium concentration) × 0.6¹
 - b. Correct at a rate of 0.6 to 1.0 mmol/L/hr until the serum sodium is 125 mEq/L.¹
 - c. Replace half of the deficit over the first 8 hours and the rest over the next 1–3 days.¹
 - d. Monitor the sodium level frequently.
7. Supplemental oxygen and/or tracheal intubation/mechanical ventilation as needed
8. Demeclocycline 150 mg qid or 300 mg bid if fluid restriction alone is inadequate.²

Post-operative

1. Follow all labs and vital signs closely, including serial serum electrolyte concentrations.
2. Monitor patient's urine output.
3. Monitor his mental status for changes.
4. Watch for signs of DIC, including thrombocytopenia, hemolysis, and abnormal bleeding.
5. Assess for metabolic acidosis with an arterial blood gas and treat as needed.
6. A TURP procedure is usually done on an inpatient basis. Following the procedure, the patient is admitted to a general floor for recovery. However, if the patient exhibits any signs/symptoms of TURP syndrome, the patient should be monitored overnight in the intensive care unit to allow for closer monitoring.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 514, 2189–94.
2. Leslie, SW. Transurethral resection of the prostate. *eMedicine*. 2006.
3. Hines RL, Marschall KE. *Stoelting: Anesthesia and Co-Existing Disease*, 5th ed. Philadelphia: Saunders, 2008, pp 345–6.

52. Emesis on Induction and Aspiration^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 35-year-old male is scheduled for a left knee arthroscopy. He denies anything to eat or drink since midnight. He is an otherwise healthy ASA 1. Your plan is for a general anesthetic with a laryngeal mask airway (LMA). After the placement of the LMA, the patient coughs and starts vomiting. What do you do? How will you treat the patient immediately? Do you secure the airway? Do you cancel the case? What medications will you give the patient? Does the patient need antibiotics? Steroids? Will you do a bronchial lavage?

CLINICAL ISSUES**Minimum Fasting Guidelines for Healthy Patients Undergoing Elective Procedures**

1. Clear liquids: 2-3 hours
 - a. Water
 - b. Fruit juice without pulp
 - c. Carbonated beverages
 - d. Clear tea
 - e. Black coffee
2. Breast milk: 4 hours
3. Infant formula and nonhuman milk: 6 hours
4. Light meals: 6 hours
 - a. Toast without butter
5. Regular meals: 8 hours
6. Fried and fatty foods or meat: >8 hours

Definitions

1. Aspiration: inhalation of gastric contents into the tracheo-bronchial tree
2. Aspiration pneumonia: aspiration of a substance containing bacteria
3. Aspiration pneumonitis: chemical injury of the lungs from sterile gastric contents and particulate antacids. The severity depends on the acidity and volume of the aspirate.
 - a. Occurs in 1 in 3,000 patients receiving general anesthesia.
 - b. Mortality is high.
 - c. 10-30% of all deaths related to anesthesia are due to aspiration.
 - d. Stage 1: symptoms peak after a few hours secondary to direct caustic effects of the aspirate.

e. Stage 2: after 6 hours the symptoms are due to an inflammatory process.

4. Mendelson syndrome: acute chemical aspiration pneumonitis. It presents with immediate respiratory distress with bronchospasm, cyanosis, tachycardia, and dyspnea. It is followed by a partial recovery and then a gradual return of respiratory dysfunction. It is associated with a very high mortality.
5. Chemical pneumonitis is more likely if the aspirate has a pH <2.5 or the volume of the aspirate is >0.4 mL/kg (about 25 mL in an adult). This results in atelectasis, edema, bronchospasm, and hypoxia within 10 minutes. The patient's chest X-ray may remain normal for hours before developing possible bilateral, perihilar, or basal atelectasis.

Predisposing Factors

1. Pregnancy
 - a. Increased gastric acid secondary to placental gastrin
 - b. Delayed gastric emptying and decrease in lower esophageal sphincter tone secondary to progesterone
 - c. Stomach contents after 4 hours in a non-pregnant patient and a non-laboring pregnant patient are the same.
 - d. A laboring patient can have solids in her stomach for up to 24 hours after eating.
2. Morbid obesity
 - a. Increased volume of gastric contents
 - b. Increased acidity of gastric contents
3. Emergency surgery
4. Decreased level of consciousness
5. Hiatal hernia
6. Scleroderma
7. Presence of a nasogastric tube
8. Diabetic gastroparesis
9. Uremia
10. Alcohol abuse
11. Drug abuse
12. Head trauma
13. Seizures
14. Neurologic disorders
15. Administration of sedatives
16. Esophageal cancer/incompetent gastroesophageal junction
17. Bowel obstruction
18. Repeated vomiting
19. Induction and recovery from anesthesia
20. Impaired laryngeal reflexes
21. Muscle weakness and paralysis

52. Emesis on Induction and Aspiration

22. Trauma

- a. Full stomach secondary to the ingestion of food or liquids prior to the trauma
- b. Full stomach secondary to swallowed blood from an oral or nasal injury
- c. Delayed gastric emptying secondary to the stress of the trauma
- d. Liquid contrast medium given for CT scanning
- e. Distracting concerns
- f. Potential difficult airway/cervical collar
- g. Pain
- h. An emergency has a four times greater risk of aspiration than an elective case.

23. Acute intoxication

24. ASA 4 and 5

- a. 1.1% risk with ASA 1 versus a 29% risk with an ASA 4 or 5.

25. Bedridden patients/prolonged hospitalization

Signs/Symptoms

1. Arterial hypoxemia is the earliest and most consistent clinical manifestation.
2. Visualization of gastric contents
3. Increased peak inspiratory pressures
4. Copious tracheal secretions
5. Pulmonary hypertension
6. Hypercarbia and acidosis
7. Wheezing/rales/rhonchi
8. Dyspnea
9. Apnea
10. Tachypnea
11. Coughing
12. Cyanosis
13. Bronchospasm/laryngospasm
14. Pulmonary edema
15. Shock
16. Radiographs
 - a. Most commonly show infiltrates and atelectasis in the dependent areas of the lung, especially the right lower lobe.
 - b. 15–20% have unremarkable chest X-rays.
 - c. Often takes 6 to 12 hours to develop.

Differential Diagnosis

1. Hypoxia secondary to an obstructed endotracheal tube
2. Bronchospasm
3. Pulmonary edema
4. Pulmonary embolism
5. Pneumonia
6. Acute respiratory distress syndrome (ARDS)



KO TREATMENT PLAN

Pre-operative

1. Pre-operative fasting
2. Avoid general anesthesia.

3. Delay non-emergent surgeries until NPO for 6–8 hours.

4. Pre-treatment medications

- a. Non-particulate antacid
 - i. Sodium citrate or sodium bicarbonate
 - ii. Reduces the level of acidity.
 - iii. Does not reduce the gastric volume.
 - iv. Decreases the severity of the aspiration.
 - v. Effectiveness is lost by 30–60 minutes after the ingestion.
- b. H2 antagonists (ranitidine)
 - i. Works by competitive inhibition of histamine binding to parietal cells.
 - ii. Decreases gastric volume.
 - iii. Decreases gastric acidity.
 - iv. Works best if given the night before and again 2 hours before surgery.
- c. Metoclopramide (10–20 mg IV)
 - i. Onset 3–5 minutes
 - ii. Increases lower esophageal sphincter tone.
 - iii. Enhances gastric emptying.
 - iv. Decreases volume of gastric fluid.
 - v. No effect on acidity
- d. Transdermal scopolamine patch
 - i. Best for motion sickness
 - ii. Must be placed several hours prior to the surgery.
 - iii. Side effects include dry mouth, somnolence, mydriasis, and dizziness.
5. Optimize the airway.
6. Effective cricoid pressure
7. Intubate the trachea and inflate the cuff as quickly as possible.
8. Consider an awake tracheotomy with local anesthetics or an awake fiber optic intubation.

Intra-operative Treatment of Aspiration

1. Trendelenburg: head of the bed down 30 degrees with a right lateral tilt
2. Apply cricoid pressure once the patient is finished vomiting. Do not apply cricoid pressure until the patient has lost consciousness.
3. Suction the airway before intubation.
4. Secure the airway with a cuffed endotracheal tube (ETT).
5. Suction the ETT before giving positive pressure ventilation.
6. Minimize exposure of the aspirate to the lungs.
7. Ensure adequate oxygenation and ventilation.
8. Supportive treatment for hypoxia and cardiovascular instability: give 100% oxygen, positive end expiratory pressure (PEEP), and inotropes as needed.
9. Bronchoscopy with irrigation only if particulate aspiration of the obstructive type occurs. Sample the aspirate for pH, gram stain, and culture.
10. Antibiotics if fecal material is aspirated and bacteria identified on a culture.
11. Steroids are not recommended. They have no proven benefit and can impair long-term healing.
12. Cancel elective cases.

13. Bronchodilators
14. Send an arterial blood gas (ABG).
15. Nasogastric suction prior to extubation
16. Extubate only after the recovery of the protective laryngeal reflexes.

Post-operative

1. A patient with suspected aspiration requires at least 24 hours of observation. The symptoms will likely worsen over the next few hours.

2. The treatment is purely symptomatic.
3. Follow serial chest X-rays.
4. Continuous pulse oximetry is recommended.

BIBLIOGRAPHY

1. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 580-1.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2456, 2595-2601, 2720-1.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1054-6.

53. Post-Operative Jaundice^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

Three days after a reportedly uneventful bilateral mastectomy, the patient's bilirubin is 7 mg/dL and she appears jaundiced. The patient is told that her jaundice is due to her anesthesia, and you are called to evaluate. What do you say to the patient? What do you say to the surgeon? What tests or exams do you order?

CLINICAL ISSUES

Definition of Jaundice

1. Hyperbilirubinemia >1.5 mg/dL
2. Yellow appearance of the skin, sclerae, and mucous membranes

Incidence of Post-operative Jaundice

1. 25-75% of patients undergoing surgery experience post-operative hepatic dysfunction. This can range from mild elevations of liver biochemical tests to hepatic failure.³
2. Jaundice has been reported in almost 50% of patients with underlying cirrhosis in the post-operative period.³
3. Patients undergoing upper abdominal surgeries are at the highest risk of post-operative liver dysfunction.³

Causes of Post-operative Jaundice

1. Pre-hepatic
 - a. Anything that causes an increased rate of hemolysis
 - b. Laboratory findings

- i. Normal to increased total bilirubin
- ii. Increased serum unconjugated/indirect bilirubin
- iii. Serum alkaline phosphatase and alanine transaminase (ALT) are often normal.
- c. Examples
 - i. Breakdown of transfused erythrocytes (hemolysis) from multiple transfusions
 - ii. ABO incompatibility
 - iii. Resorption of hematomas
 - iv. Hemolytic anemia: drug-induced
 - v. Glucose-6-phosphate dehydrogenase deficiency (G6PD)
 - vi. Malaria
 - vii. Sickle cell anemia
 - viii. Spherocytosis
 - ix. Kidney diseases leading to hemolytic uremic syndrome
2. Decreased hepatic clearance secondary to hepatic hypoperfusion
 - a. Non-cardiogenic shock/bacteremia/infection/sepsis
 - b. Cardiogenic shock (heart failure)
 - c. Endotoxemia
3. Hepatic
 - a. Direct hepatocellular injury
 - b. Laboratory findings
 - i. Increased total bilirubin
 - ii. Predominant serum ALT elevation
 - iii. Conjugated bilirubin present in the urine
 - c. Examples
 - i. Acute viral hepatitis and other unrecognized chronic liver diseases
 - ii. Acute post-transfusion hepatitis

53. Post-Operative Jaundice

- iii. Alcoholic liver disease
- iv. Hypotension
- v. Hypovolemia
- vi. Peri-operative hypoxia
- vii. Ischemic hepatitis: shock liver
- viii. Hepatic allograft rejection
- ix. Hepatic artery thrombosis
- x. Primary biliary cirrhosis
- xi. Gilbert's syndrome
- xii. Crigler-Najjar syndrome
- xiii. Metastatic carcinoma
- xiv. Hepatotoxicity
 - (1) Hepatotoxic drugs:
 - (a) Tetracyclin
 - (b) Rifampin
 - (c) Cephalosporins
 - (d) Penicillin
 - (e) Inhalational anesthetics: halothane
 - (f) Sodium thiopental
 - (g) Ranitidine
 - (h) Insulin
 - (i) Hydralazine
 - (j) Labetalol
 - (k) Procainamide
 - (l) Phenothiazine
 - (m) Alpha-methyl dopa
 - (n) Heparin
 - (o) Salicylates
 - (p) Acetaminophen
 - (q) Ibuprofen/non-steroidal anti-inflammatory drugs (NSAIDs)
 - (r) Steroids
 - (s) Alcohol
 - (t) Oral contraceptives
 - (u) IV contrast media

4. Post-hepatic

- a. Obstructive jaundice/cholestatic jaundice
- b. Caused by an interruption to the drainage of the bile in the biliary system.
- c. Patient may present with pale stools, dark urine, and severe itching.
- d. Laboratory findings
 - i. Increased total bilirubin
 - ii. Increased direct/conjugated bilirubin
 - iii. Elevated serum alkaline phosphatase
- e. Examples
 - i. Mechanical obstruction of the common bile duct
 - (a) Gallstone
 - (b) Stricture
 - (c) Biliary atresia
 - (d) Duct ligation
 - (e) Surgical injury
 - (f) Tumor: pancreatic cancer at the head of the pancreas, pancreatitis, pancreatic pseudocysts, ductal carcinoma
 - (g) "Liver fluke" parasites (Trematoda)

- ii. Sepsis
- iii. Prolonged cardiopulmonary bypass
- iv. Acalculous cholecystitis
- v. Benign post-operative cholestasis
- vi. Cholangitis
- vii. Biliary sludge
- viii. Prolonged total parenteral nutrition (TPN)
- ix. Dubin-Johnson syndrome
- x. Drugs
 - (a) Amoxicillin-clavulanate
 - (b) Erythromycin
 - (c) Trimethoprim-sulfamethoxazole
 - (d) Chlorpromazine
 - (e) Warfarin

Clinical and Laboratory Signs and Symptoms of Post-operative Jaundice

1. Most often manifests within 3 weeks of the surgery.
2. Anemia
3. Yellowish discoloration of the skin, sclerae, and mucous membranes
4. An associated fever, rash, and eosinophilia are often present with halothane anesthetic toxicity.
5. Hypotension is often present in patients with ischemic hepatitis.

Inhalational Anesthetic-Induced Jaundice

1. Halothane
 - a. Rare syndrome of acute hepatotoxicity, usually after repeat exposures.
 - i. First exposure incidence: 0.3–1.5 per 10,000³
 - ii. Incidence after multiple exposures: 10–15 per 10,000³
 - b. Halothane is rarely used in the United States but is commonly seen in other countries.
 - c. Presents with fever, eosinophilia, jaundice, myalgias, ascites, gastrointestinal hemorrhage, and hepatic necrosis a few days to weeks after the anesthesia.
 - d. More frequent in females (2:1)
 - e. Mortality rate is 10–80% in the pre-transplantation era. Much lower mortality with a liver transplant.
 - f. Associated risk factor
 - i. Age 40 years or older
 - ii. Female
 - iii. Repeat exposures
 - iv. Obesity
 - v. Family predisposition
 - g. Spontaneous recovery of symptoms can occur.
2. Isoflurane, sevoflurane, and desflurane
 - a. The quantity of metabolized volatile anesthetic contributes to hepatotoxicity.
 - b. Hepatotoxicity is extremely rare because these volatile anesthetics exhibit a much lower degree of metabolism compared to halothane.


KO TREATMENT PLAN
Post-operative Evaluation
Conversation with the Patient and Surgeon

The important thing to know is that the jaundice is unlikely secondary to the anesthesia. Halothane is the usual anesthetic cause of jaundice but is rarely used today. The jaundice could very likely be secondary to an acute peri-operative event. Tell the patient that you will carefully examine her and her peri-operative records to help to determine a possible cause of her jaundice.

1. Perform a history and physical exam.
 - a. Age
 - b. Sex
 - c. Latency/onset of the jaundice
 - d. Does the patient have a fever?
 - e. Does the patient have a rash?
 - g. History of hepatomegaly?
 - h. History of any pre-existing liver problems?
 - i. Coagulopathy?

2. Review the peri-operative record for events that can lead to jaundice.
 - a. Did the patient receive a transfusion?
 - b. Make a list of all of the medications given in the peri-operative period that may be hepatotoxic.
 - c. Note any hypoxic events.
 - d. Note any significant hypotensive events.
3. Order appropriate labs and tests.
 - a. Liver function tests
 - b. Check for serum eosinophilia.
 - c. Hemoglobin and hematocrit to check for hemolysis
 - d. Coagulation panel
 - e. Send a urine and stool sample. Check the urine for signs of hemolysis.
 - f. Abdominal ultrasound: liver, gallbladder
4. Consult gastroenterology.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2219-22.
2. Goldman L, Ausiello D. *Cecil Medicine*, 23rd ed. Philadelphia: Saunders Elsevier, 2008, chapters 150, 151, 153, and 159.
3. Feldman M, Friedman LS, Brandt LJ. *Sleisenger & Fordtran's Gastrointestinal and Liver Disease*, 8th ed. Philadelphia: Saunders Elsevier, 2006, pp 1853-5.

54. Ascites^{1,2,3}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 50-year-old man with a history of liver cirrhosis secondary to alcohol abuse presents for an exploratory laparotomy for a suspected bowel obstruction. His blood pressure is 100/50 mm Hg and his heart rate is 92. His abdomen is severely distended with ascites. How does his liver cirrhosis and ascites affect your anesthetic plan? Would you like any pre-operative tests? Should his ascites be drained prior to the surgery?

CLINICAL ISSUES

Definition of Ascites

1. Accumulation of fluid in the peritoneal cavity
2. Common sequela of many forms of cirrhosis
3. Factors that contribute to its formation
 - a. Portal hypertension

- b. Hypoalbuminemia
- c. Sodium and water retention

Theories of Pathogenesis of Ascites in Cirrhosis

1. Underfilling hypothesis
 - a. Cirrhosis-related hepatic venous drainage block and portal hypertension leads to ascitic fluid formation.
 - b. This decreases the effective intra-vascular volume, leading to further sodium and water retention by the kidney.
2. Overflow hypothesis
 - a. Portal hypertension leads to sodium and water retention despite an overfilled vasculature, which increases the total plasma volume.
 - i. Expanded intra-vascular volume
 - (1) Increased portal hydrostatic pressure, which leads to portal hypertension.

54. Ascites

(1) Be prepared for circulatory collapse after the drainage of the ascites.

(a) Give concomitant intravenous plasma expander solutions during the ascites drainage.

(b) Re-equilibration of the intra-vascular volume will occur 6–8 hours after the removal of a large volume of ascitic fluid.

Intra-operative

1. Standard ASA monitors with consideration given to the placement of an arterial line and possible central line

a. Arterial line

i. Immediate measurement of the patient's hemodynamic status in the setting of large volume shifts.

(1) Similar hemodynamic changes to those seen during large volume paracentesis may be anticipated in the setting of emergent laparotomy in the face of tense ascites.

ii. Frequent blood draws

b. Central line

i. Provides an indication of cardiac filling pressures.

ii. Placement may be difficult in patients with coagulopathy and known pulmonary dysfunction.

iii. Monitoring of intra-vascular fluid volume, especially in situations of

1. Underlying cardiovascular instability

2. Hepatorenal syndrome

3. Dehydration

4. Anticipated large intercompartmental fluid shifts

c. Foley catheter

2. A rapid-sequence intubation should be performed to minimize the possibility of aspiration.

a. In this sample case, the ascites increases intra-abdominal pressure, while the bowel obstruction also independently increases the probability of aspiration.

b. However, renal dysfunction or elevated potassium may preclude the use of succinylcholine.

3. Maintenance drugs

a. Any maintenance regimen may be used as part of a balanced anesthetic technique.

b. Muscle relaxants

i. Because of the altered volume of distribution, coupled with the altered hepatic metabolism in patients with end-stage liver disease (ESLD), train-of-four stimuli need to be monitored.

ii. Succinylcholine or mivacurium are acceptable, but severe liver disease may decrease plasma cholinesterase activity and prolong the duration of action.

1. Plasma potassium levels may preclude the use of succinylcholine.

iii. Cisatracurium and rocuronium may be beneficial in order to avoid issues with hepatic metabolism. (Please refer to the liver transplant chapter for further information.)

Post-operative

1. Post-operative care is guided by the patient status before and during the case.

2. Most likely, however, this patient will need intensive care unit monitoring to prevent cardiac decompensation in the subsequent 6–12 hours post-surgery, as intercompartmental fluid shifts occur.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1093–7.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 760–3, 2220–3.
3. Hines, Marschall. *Stoelting's Anesthesia and Co-existing Disease*, 5th ed. New York: Churchill Livingstone, 2008, chapter 11.

55. Physiologic Changes of Pregnancy^{1,2,3}

Jessica Lovich-Sapola, MD

SAMPLE CASE

A 24-year-old female presents for a repeat cesarean section. She is otherwise healthy. She is deathly afraid of needles and demands a general anesthetic. It is important that you give her an informed consent for her anesthetic. Please explain to her your anesthetic of choice, and why. Discuss with her the physiologic changes that occur in pregnancy, and why they affect your anesthetic plan.

CLINICAL ISSUES**Cardiovascular System**

1. Increases
 - a. Cardiac output (CO)
 - i. Increases from the fifth week of pregnancy.
 - ii. Maximum level at 32 weeks of pregnancy
 - iii. Increases by 30–50% during pregnancy.
 - iv. CO increases secondary to an increase in stroke volume and heart rate
 - v. The cardiac output continues to increase by 60–85% during labor and in the postpartum period secondary to autotransfusion and the removal of aortocaval compression by evacuating the uterus.
 - vi. The cardiac output will return to non-pregnant values over the next 12 weeks postpartum.
 - b. Stroke volume (SV)
 - i. Increases by 20–50% at term from non-pregnant values.
 - c. Heart rate (HR)
 - i. An increase in resting HR can be seen by 4 weeks gestation and rises as much as 20% over the gestational period.
 - d. Blood volume
 - i. Increases by 45% over non-pregnant values.
2. Decreases
 - a. Systemic vascular resistance (SVR)
 - b. Systemic blood pressure
 - c. Pulmonary vascular resistance
3. No change
 - a. Central venous pressure (CVP)
 - b. Pulmonary capillary wedge pressure
 - c. Pulmonary artery pressure
 - d. Left ventricular function
 - e. Ejection fraction

4. Variable
 - a. Contractility
5. EKG changes associated with pregnancy
 - a. Sinus tachycardia
 - b. Left axis deviation
 - c. Ectopic beats
 - d. Inverted or flattened T waves
 - e. Q waves in lead III
6. Minor changes in the cardiovascular system are considered normal, further investigation is warranted if the patient has
 - a. Chest pain
 - b. Syncope
 - c. Severe arrhythmias
 - d. Systolic murmur more than grade 3
 - e. Diastolic murmur

Hematologic System

1. Physiologic anemia
 - a. Plasma volume increases by a higher percentage (50%) compared to cell volume (30%) which will decrease hematocrit.
 - i. Oxygen transport is not compromised by this physiologic anemia due to the patient's
 - (1) Increase in cardiac output
 - (2) Increased partial pressure of arterial oxygen
 - (3) Rightward shift of the oxyhemoglobin dissociation curve
 - b. This physiologic anemia occurs to
 - i. Allow adequate perfusion to vital organs, including the fetus.
 - ii. Prepare for blood loss associated with parturition.
2. Average hemoglobin of 11.6 g/dL
3. Average hematocrit of 35.5 %
4. Platelet count is usually unchanged.
5. Hypercoagulable state
 - a. Increased levels of most coagulation factors including VIII, IX, X, and XII
 - b. Largest increase in factor VII and fibrinogen
6. Plasma proteins
 - a. Albumin levels decrease.
 - b. Decrease in the colloid oncotic pressure

Respiratory System

1. Increases
 - a. Respiratory minute volume

55. Physiologic Changes of Pregnancy

- b.** Minute ventilation: due to increased respiratory center sensitivity and drive
 - i.** Increase in tidal volume with a constant respiratory rate
 - ii.** The increased respiratory drive is believed to be due to the elevation in serum progesterone, a direct respiratory stimulant.
 - c.** Work of breathing
 - d.** Tidal volume increases by 50%.
 - i.** The patient has an increased oxygen demand and requirement for carbon dioxide elimination.
 - e.** Pulmonary blood flow with increased pulmonary capillary blood volume
 - i.** Pulmonary edema
 - f.** Oxygen carrying capacity
 - g.** Oxygen consumption: increased metabolic demands
- 2.** Decreases
 - a.** Functional residual capacity (FRC)
 - b.** Expiratory reserve volume
 - c.** Chest wall compliance: enlarging uterus
 - 3.** No change
 - a.** Respiratory rate
 - b.** Closing capacity
 - c.** Vital capacity
 - d.** Forced expiration
 - i.** Forced expiratory volume in 1 second (FEV₁) is not affected by pregnancy.
 - 4.** Compensated respiratory alkalosis
 - 5.** Rapid hypoxia secondary to
 - a.** Decreased FRC
 - b.** Decreased FRC/closing capacity (CC) ratio resulting in faster small airway closure when the lung volume is reduced
 - c.** Increased oxygen consumption
 - 6.** Capillary engorgement of the mucosa is due to increased blood volume and increased estrogen.
 - a.** Edema of the oropharynx, larynx, and trachea
 - b.** Increased risk of difficult intubation
 - i.** Use a smaller endotracheal tube than usual for this patient.
 - c.** Any airway manipulation may result in
 - i.** Edema
 - ii.** Bleeding
 - iii.** Upper airway trauma
 - d.** The nasopharynx mucosa is particularly friable; therefore, all instrumentation of the nose should be avoided.
 - 7.** Normal chest X-ray in a pregnant patient
 - a.** Mild cardiomegaly
 - b.** Widened mediastinum
 - c.** Increased anterior-posterior diameter
 - d.** Prominence of the pulmonary vasculature

Gastrointestinal System

- 1.** Associated with an increased risk of aspiration with anesthesia.

- 2.** Progesterone
 - a.** Relaxes smooth muscle
 - b.** Impairs esophageal and intestinal motility
 - c.** Reduces lower esophageal sphincter tone
 - d.** Increases the placental production of gastrin: increases gastric acidity.
 - e.** Delayed gastric emptying and increased emesis
 - i.** Laboring patients/painful labor
 - (1)** Placentally derived gastrin
 - ii.** Patients undergoing anesthesia
 - (1)** Increased risk of aspiration when sedated after about 16 weeks gestational age
 - iii.** Patients receiving parenteral opioids
 - iv.** Recent studies show that gastric emptying is likely not decreased in the healthy, term, non-obese, non-laboring patient.
 - v.** Epidural analgesia with local anesthetics and not opioids does not seem to affect the patient's gastric emptying.
 - (1)** Small doses of epidural fentanyl also do not have an effect on gastric emptying.
- 3.** Lab values in pregnancy
 - a.** Increased: alkaline phosphatase levels
 - b.** Decreased: transaminase and bilirubin levels

Renal System

- 1.** Affected by the increase in progesterone and the mechanical effects of compression by the enlarging uterus
- 2.** Increases
 - a.** Urea clearance
 - b.** Creatinine clearance
 - c.** Uric acid clearance
 - d.** Renal plasma flow (RPF): secondary to the increase in CO
 - e.** Glomerular filtration rate (GFR): secondary to the increase in CO
 - f.** Risk of urinary tract infection, nephrolithiasis, and pyelonephritis
 - g.** Frequency, urgency, and incontinence
- 3.** Decreases
 - a.** Plasma creatinine and urea
 - i.** Secondary to
 - (1)** Dilutional effect of plasma volume expansion
 - (2)** Increase in GFR
 - ii.** "Normal" renal indices in pregnancy are lower than in the non-pregnant state.

Central Nervous System

- 1.** Increased sensitivity to both regional and general anesthetics.
 - a.** Decreased minimum alveolar concentration (MAC) for volatile anesthetics.
 - b.** Mechanism is unclear.
 - i.** Possibly due to the increased progesterone levels

- ii. Possibly due to the increased concentrations of endorphins and dynorphins which results in an altered pain threshold
- iii. Mechanical effects of the enlarging uterus
 - (1) It is likely that the true mechanism is multifactorial.



KO TREATMENT PLAN

Pre-operative

1. Regional anesthesia is always the preferred choice for a cesarean section in an otherwise healthy patient.
 - a. Rapid hypoxia
 - b. Capillary engorgement of the mucosa
 - i. Edema of the oropharynx, larynx, and trachea
 - ii. Increased risk of difficult intubation
 - iii. Any airway manipulation may result in
 - (1) Edema
 - (2) Bleeding

- (3) Upper airway trauma
- vi. Avoid instrumentation of the nose.
- c. Increased risk of aspiration

Intra-operative

1. Understand that a patient refusal is the number one contraindication to a regional anesthetic.
 - a. If after your long explanation of why you would choose a regional anesthetic, she still chooses a general anesthetic, put her to sleep.
 - i. Assuming she has a normal airway exam, perform a rapid sequence induction/intubation.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2307–11.
2. Hill CC, Pickinpaugh J. Physiologic changes in pregnancy. *Surgical Clinics of North America* 2008;88(2):391–401.
3. Wise RA, Polito AJ, Krishnan V. Respiratory physiologic changes in pregnancy. *Immunol Allergy Clin North Am* 2006;26(1):1–12.

56. Pre-Eclampsia¹⁻⁷

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 26-year-old female, G1P0 at 34 3/7 weeks, presents with complaints of severe right upper quadrant pain. She has had no prior issues with this pregnancy. Her blood pressure (BP) is 160/90 mm Hg, heart rate (HR) 75, respiratory rate (RR) is 20, and temperature 36.6°C. On exam, the patient has some tenderness in her right upper abdominal quadrant and moderate pedal edema. She has a Mallampati (MP) class III airway. Laboratory analysis reveals platelets 100, AST 156, ALT 174, and creatinine 1.0. She is scheduled for an urgent cesarean section. What are your concerns? Do you want any more laboratory tests? Would you use a regional or general anesthetic? If a general anesthetic was required, how would you induce anesthesia? What monitors would you use?

CLINICAL ISSUES

Definition

1. Pre-eclampsia
 - a. This is a disease process in which the basic underlying etiology is unclear, but it may be related to an excess

production of thromboxane versus prostacyclin by the placenta.

- b. Thromboxane leads to vasoconstriction, platelet aggregation, increased uterine activity, and decreased uteroplacental blood flow.
- c. In a normal pregnancy, these effects are equally opposed by the actions of prostacyclin.
- d. In pre-eclampsia the widespread arteriolar vasoconstriction causes hypertension, tissue hypoxia, and endothelial damage.
- e. Placental ischemia leads to a release of thromboplastin with subsequent deposition on the already constricted glomerular vessels, which in turn leads to proteinuria.
- f. Renal damage may continue with decreased renal blood flow and a decreased glomerular filtration rate.
- g. Right upper quadrant pain may be secondary to a subcapsular hematoma of the liver (rare). Rupture of this liver hematoma carries an 80% mortality rate.
- h. Cerebral edema and small foci of degeneration may develop.

56. Pre-Eclampsia

2. Mild pre-eclampsia is usually accompanied by 1, 2, or 3 of the following:

- a. Hypertension: BP >140/90 mm Hg, two elevated BP's taken four hours apart
- b. Proteinuria >300 mg over 24 hours
- c. Non-pedal edema

3. Severe pre-eclampsia

- a. Severe hypertension: systolic blood pressure (SBP) >160 mm Hg or a diastolic blood pressure (DBP) >110 mm Hg
- b. Severe proteinuria >5 gm over 24 hours
- c. Evidence of severe end-organ damage with refractory oliguria
- d. Cerebral headache or visual changes
- e. Pulmonary edema
- f. Epigastric pain
- g. Intrauterine growth retardation
- h. The HELLP syndrome is a form of severe pre-eclampsia characterized by hemolysis, elevated liver enzymes, and low platelets.
- i. Low platelets may occur due to adherence of the platelets at sites of endothelial damage, resulting in a consumptive coagulopathy.

4. Eclampsia

- a. The occurrence of convulsions and/or coma unrelated to pre-existing neurologic disease
- b. Seizures may occur before, during, or after the delivery.
- c. 25% of all postpartum eclampsia-related seizures occur between 48 hours and 4 weeks postpartum.
- d. Once a seizure occurs, it should be stopped with 50 to 100 mg of sodium thiopental. A bolus of IV magnesium sulfate, 4 to 6 gm, should then be administered, followed by an initial infusion of 2 gm/hr.
- e. An airway must be established.
 - i. Administer supplemental oxygen
 - ii. Bag and mask ventilate as needed
- f. Pulse oximetry, EKG, and blood pressure should be monitored.
- g. The only "cure" for pre-eclampsia/eclampsia is delivery of the fetus and placenta.
- h. Typically in pre-eclampsia and HELLP syndrome, the obstetrical management strategies should focus on the stabilization of the mother until the fetus is mature. With severe pre-eclampsia, if the patient is stable, she may be followed until the fetus is viable or at least until 48 hours of steroid benefit can be achieved. Progression to eclampsia or fetal deterioration mandates immediate delivery of the fetal/placental unit.

Thrombocytopenia

1. Circulating platelets adhere to the damaged or activated endothelium.
2. The thrombocytopenia is usually moderate, and clinical bleeding is rare unless disseminated intravascular coagulation (DIC) develops.

3. The choice of anesthetic technique depends upon the mode of delivery, fetal gestational age, coagulation status, history of recent/current bleeding, and other medical issues.

4. The risk of an epidural hematoma is estimated to be about 1:150,000 after epidural analgesia.

5. A platelet count of 100,000 is considered safe for performing an epidural block, although there are no supporting data.

6. Hematologists suggest that a platelet count of over 50,000 is safe for surgery and neuraxial blockade, provided the function of the existing platelets is normal.

7. Many anesthesiologists would agree that a platelet count of 80,000 would be safe for a central neuraxial block, as the benefits of regional anesthesia far outweigh the risks of a general anesthetic. It is important to follow the trend of the patient's platelets.

8. Available platelet function tests include

- a. Thromboelastogram (TEG)
- b. Aggregometry
- c. Flow cytometry
- d. Platelet function assay (PFA)-100
 - i. The most rapid and simple assessment of platelet aggregation
 - ii. Uses epinephrine and ADP.

Magnesium

1. This is the first-line therapeutic drug used for controlling pre-eclampsia and eclampsia.

2. Properties of magnesium include

- a. Central nervous system depressant and anticonvulsant effects
- b. Vasodilation with increased uterine and renal blood flow
- c. Bronchodilator
- d. Decreased platelet aggregation
- e. Tocolysis
- f. Decreased fetal heart rate variability
- g. Generalized muscle weakness
- h. Decreased muscle tone and lower APGAR scores in neonates
- i. Increased maternal sensitivity to both depolarizing and nondepolarizing muscle relaxants
 - i. Magnesium inhibits the release of acetylcholine at the neuromuscular junction.
 - ii. Decreases the sensitivity of the motor endplate to acetylcholine
 - iii. Decreases the muscle membrane excitability

3. Therapeutic levels of magnesium are between 4 and 6 mEq/L.

a. Deep tendon reflexes are lost at 10 mEq/L.

b. Sinoatrial and atrioventricular block occurs concurrent with respiratory paralysis at 15 mEq/L.

c. Cardiac arrest occurs around 20 mEq/L.

4. Calcium gluconate counteracts the cardiac effects of magnesium.

5. Treatment of magnesium toxicity

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- a. Stop the magnesium infusion.
- b. Intravenous calcium gluconate 1 gm or calcium chloride 300 mg
- c. Ventilate and intubate if respiratory failure occurs.



KO TREATMENT PLAN

Be prepared to defend your choices. The following is representative of how the authors might manage this patient. However, be prepared to be taken down a total/high spinal pathway or a difficult airway pathway as alternatives. Multiple anesthetic techniques are acceptable, but be prepared to defend your choice.

Pre-operative

1. Prepare for surgery with consideration of a 500 mL fluid bolus of crystalloid to expand her intravascular volume depending on a current assessment of her volume status.
 - a. Remember that a patient who is currently on a magnesium drip has a tenuous fluid status and an increased risk of pulmonary edema.
2. Order a repeat platelet count to determine the trend of the platelet count, and with what rapidity the platelets are falling.
3. Given that this is an urgent cesarean section, spinal anesthesia would be appropriate.
4. In the face of a difficult airway, it is defensible to perform a neuraxial anesthetic technique down to a platelet count of about 80,000, assuming no recent rapid decrease in the platelet number.
5. Provide aspiration prophylaxis with an H₂-blocker and administer 30 mL of a nonparticulate antacid.
6. Consider an arterial line prior to the placement of the neuraxial technique if the severely pre-eclamptic patient has uncontrolled blood pressure, in order to better monitor the blood pressure changes.

Intra-operative

1. Be prepared to treat large swings in blood pressure that may occur due to the spinal anesthetic.
2. Treat hypotension with oxygen via face mask, left uterine displacement, hydration, and IV phenylephrine or ephedrine.
3. Remember that the pre-eclamptic patient is more sensitive to the effects of pressors.
4. Raise the lower extremities to facilitate venous return.

The Airway

1. Difficulty intubating occurs at a higher rate in pregnant than non-pregnant women, and patients with pre-eclampsia have additional considerations.

2. Often, upper airway narrowing occurs in these patients during sleep.
3. Reduced plasma proteins due to proteinuria and marked fluid retention make the tongue larger and less mobile.
4. Multiple direct laryngoscopy attempts may lead to laceration and bleeding of the pharyngeal structures.
5. Swelling can be severe enough to cause total airway obstruction.

TKO: Please review the difficult airway section to consider the various scenarios examiners may present in the obstetric patient.

Epidural versus Spinal Anesthesia

1. There has been concern that the intravascular volume contraction that has been noted in pre-eclamptic patients could result in severe hypotension with the onset of a sympathectomy.
2. Historically, epidural anesthesia has been used in these patients in order to better control the sympathectomy with a more gradual onset. Recent studies, however, are showing no significant difference in the incidence of hypotension in severely pre-eclamptic women with spinal or epidural anesthesia.
3. It is felt that spinal anesthesia in stable, noncoagulopathic, severely pre-eclamptic women is a reasonable alternative to an epidural block, especially in emergency situations, and in an attempt to avoid general anesthesia.

General Anesthetic

1. If there is a contraindication for a regional anesthetic, a general anesthetic must be performed.
2. Give aspiration prophylaxis.
 - a. Oral nonparticulate antacid
 - b. H₂-blocker
 - c. Metoclopramide
3. Preoxygenate and denitrogenate with 100% oxygen.
4. Labetalol or nitroglycerin can be given pre-induction to blunt the sympathetic response to direct laryngoscopy and intubation.
5. Perform a rapid-sequence induction with sodium thiopental and succinylcholine, assuming the patient does not have a "difficult airway."
 - a. This patient's airway exam revealed a Mallampati III score; therefore, an awake fiber optic intubation should be strongly considered.
6. Have smaller endotracheal tubes available secondary to the associated airway edema.
7. Remember that the magnesium sulfate will potentiate the effects of both depolarizing and nondepolarizing muscle relaxants.
8. Consider a pre-induction arterial line in any patient with severe pre-eclampsia.

Post-operative

1. Be aware of the postoperative development of eclampsia.
2. Continue magnesium therapy in the post-operative setting for 24–48 hours.
3. Monitoring must be continued for 24–48 hours after delivery due to the potential development of eclampsia as well as the risks associated with magnesium therapy. The location of management: labor and delivery, ICU or floor, is at the discretion of the managing physician, guided by the patient's clinical status.
4. An additional 24 hours of BP monitoring in the postpartum unit once the magnesium drip is completed is also recommended.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1164–7.
2. Kam PCA, Thompson SA, Liew ACS. Thrombocytopenia in the parturient. *Anaesthesia* 2004;59:255–64.
3. Mandal NG, Surapenemi S. Regional anaesthesia in pre-eclampsia: advantages and disadvantages. *Drugs* 2004;64(3):223–36.
4. Munner U, deBoisblanc B, Suresh MS. Airways problems in pregnancy. *Crit Care Med* 2005;33(10):S259–68.
5. Chestnut DH, ed. *Obstetric Anesthesia: Principles and Practice*, 2nd ed. New York: Churchill Livingstone, 1999, p 911.
6. Hughes SC, Levinson G, Rosen MA, eds. *Shnider & Levinson's Anesthesia for Obstetrics*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2001, pp 297–315.
7. Yao FSF, Fontes ML, Malhorta V. *Yao and Artusio's Anesthesiology: Problem-Oriented Patient Management*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 917–18 and 923.

57. Obstetrical Antepartum Hemorrhage^{1,2,3,4}*Jessica A. Lovich-Sapola, MD***PLACENTAL ABRUPTION^{1,2,4}****SAMPLE CASE**

A 22-year-old female presents to labor and delivery at 34 weeks gestation. She reports bleeding for several hours. She has mild abdominal tenderness. A toxicology screen is positive for cocaine. Would you recommend regional or general anesthetic for her cesarean section?

CLINICAL ISSUES**Definition**

1. Partial or complete separation of the placenta from the uterus before delivery of the fetus
2. Occurs in 0.2–2.4% of all pregnancies.
3. Usually occurs in the final 10 weeks of gestation.

Pre-existing Conditions

1. Chronic hypertension
2. Pregnancy-induced hypertension
3. Pre-eclampsia
4. Maternal cocaine use
5. Excessive alcohol intake

6. Smoking
7. Previous history of abortion
8. Advanced age
9. Premature rupture of membranes
10. Trauma: especially blunt trauma to the abdomen

Presentation

1. Vaginal bleeding of dark, clotted blood
2. Uterine tenderness
3. Increased uterine activity/hypertonus
4. Blood loss can be concealed behind the placenta and therefore underestimated.
5. A greater than 2 L blood loss can result in maternal hypotension and tachycardia.
6. Fetal bradycardia and death

Associated Complications

1. Up to a 30% risk of disseminated intravascular coagulation (DIC)
2. Fetal demise secondary to hypoxia
3. Acute renal failure
4. Hemorrhage and shock
5. Anterior pituitary necrosis: Sheehan's syndrome
6. Couvelaire uterus: extravasated blood dissects between the myometrial fibers

57. Obstetrical Antepartum Hemorrhage

7. 2–11% maternal mortality
8. Up to 50% fetal mortality

Obstetrical Management

1. Large bore IV access
2. Draw baseline blood samples: hematocrit, coagulation studies, blood typing, and cross-matching.
3. Continuous monitoring of the fetal heart rate
4. Supplemental oxygen
5. Placement of a Foley catheter
6. Left uterine displacement
7. Deliver the fetus and placenta.
8. Correct the patient's blood volume and coagulation with blood components.
 - a. Fresh frozen plasma
 - b. Cryoprecipitate
 - c. Platelets
 - d. Packed red blood cells
9. Mild cases, with no evidence of fetal distress and a favorable cervix, can be managed by the induction of labor with oxytocin and the artificial rupture of the amniotic membranes.
10. Non-reassuring fetal status warrants an emergency cesarean section.
11. Mild cases with a pre-term fetus may warrant a delay of delivery and observation in the hospital to allow time for fetal lung maturation.



KO TREATMENT PLAN

Labor and Vaginal Delivery

1. A continuous epidural anesthetic has been successfully used in patients with normal coagulation studies and no intravascular volume deficit.

Intra-operative

1. General anesthesia is usually preferred in the setting of coagulopathies, uncontrolled hemorrhage, and hemodynamic instability.
 - a. IV ketamine (1 mg/kg) or etomidate are the preferred choices for the induction of anesthesia. Thiopental may precipitate hypotension in a patient with unrecognized hypovolemia.
2. Depending on the severity of the abruption, an arterial line and/or central venous pressure (CVP) monitor may be necessary to guide volume resuscitation.

PLACENTA PREVIA^{1,2,4}

SAMPLE CASE

A 33-year-old patient presents to labor and delivery with painless vaginal bleeding. This is her fifth pregnancy, and two

were delivered by cesarean section. She is awake and alert. All of her blood labs are normal. She is hemodynamically stable. Since she is 37 weeks pregnant, her obstetrician decides to perform an elective cesarean section. Would you do regional or general anesthesia?

CLINICAL ISSUES

Definition

1. Low implantation of the placenta into the uterus
2. The placenta either is overlying or encroaching on the cervical os.
3. Present in about 0.1–1% of all pregnancies

Diagnosis

1. Painless, bright red vaginal bleeding
2. The bleeding usually occurs after the 7th month of pregnancy.

Increased Risk

1. Multiparous women
2. Previous cesarean section
3. Prior uterine trauma
4. Advanced maternal age
5. Previous uterine surgery
6. Previous placenta previa

Obstetrical Management

1. Diagnosed by ultrasound
2. Avoid a vaginal exam, secondary to the possibility of inducing bleeding.
3. If the bleeding stops spontaneously, conservative treatment is recommended.
 - a. Fetal demise is uncommon with the first episode of bleeding.
 - b. Tocolysis: medications used to suppress premature labor
 - i. Magnesium sulfate
 - ii. Terbutaline
 - iii. Indomethacine
 - iv. Nifedipine
 - c. Blood transfusions as needed
 - d. Serial ultrasounds
 - e. Nonstress test and biophysical profile
 - f. Fetal lung maturity studies should be performed if leaning toward an early delivery (i.e., less than 39 weeks).
4. Persistent bleeding, active labor, or a mature fetus require delivery of the fetus by cesarean section.
 - a. Send a type and cross for packed red blood cells (PRBC).
 - b. Place at least one large-gauge IV.
 - c. Send labs for hematocrit.

57. Obstetrical Antepartum Hemorrhage

Diagnosis

1. Non-specific symptoms
2. Fetal bradycardia, variable and late decelerations
3. Maternal hypotension
4. Loss of function of the uterine pressure monitors
5. Constant abdominal pain (possibly concealed by a labor epidural)
6. Change in the uterine shape
7. Cessation of contractions
8. Hematuria
9. Vaginal bleeding
10. Suspected clinically, but confirmed surgically with a laparotomy
11. Loss of station of fetal presenting parts

Obstetrical Management

1. Repair of the uterus
2. Arterial ligation: O'Leary or hypogastric artery ligation
3. Hysterectomy



KO TREATMENT PLAN

Intra-operative

1. Emergency laparotomy
2. Regional anesthesia can be used if a good working epidural is already in place and the patient is hemodynamically stable.
3. If you do not have an epidural in situ, general anesthesia is usually indicated.
4. You must explain to the patient that it is likely that she may still require general anesthesia depending on the findings of the laparotomy. Uterine rupture can lead to significant damage to the surrounding organs, especially the bladder.
5. Aggressive volume replacement
6. Maintain urine output.

VASA PREVIA^{1,4}

CLINICAL ISSUES

Definition

1. Velamentous insertion of the umbilical vessels so that they run through the amniotic membranes traversing between fetal presenting part and the cervical os.¹

2. These vessels are very fragile and susceptible to trauma during labor.
3. The associated bleeding is fetal in origin.
4. Uncommon (1 in 3,000 deliveries)
5. High fetal mortality secondary to the fetus's small blood volume (50–75%)
6. No threat to the mother

Diagnosis

1. Vaginal bleeding immediately on rupture of the amniotic membranes
2. Fetal heart rate abnormalities

Increased Risk

1. Multiple gestation, especially with triplets

Obstetrical Management

1. Early diagnosis and treatment
2. Immediate delivery



KO TREATMENT PLAN

1. Emergency cesarean section
2. Use the fastest technique possible for the induction of anesthesia. This is usually general anesthesia. If you believe that you could get a spinal in the lateral position faster than inducing general anesthesia, do that. If you already have a continuous spinal or epidural, use that. Use whatever is the fastest and safest technique.
3. Remember, the bleeding is fetal and not maternal. The mother should not be hypotensive from the blood loss. She also should not require significant volume replacement.
4. The neonate will require rapid volume replacement after delivery. The physician may transfuse some of the baby's own blood drawn from the umbilical-placental vessels into a heparinized syringe.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2333–6.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1167–8.
3. Gabbe SG, Niebyl JR, Simpson JL. *Obstetrics: Normal and Problem Pregnancies*, 5th ed. Philadelphia: Churchill Livingstone & Elsevier, 2007, chapters 15 and 18.
4. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 119 and 662–76.

58. Obstetrical Postpartum Hemorrhage^{1,2,3}

Jessica A. Lovich-Sapola, MD

PLACENTA ACCRETA¹

CLINICAL ISSUES

Definition

1. The placenta becomes abnormally adherent to the implantation site.
2. Potentially life threatening
3. Classified by the degree of placental penetration of the myometrium
 - a. Placenta accreta: the placenta is adherent to the myometrium.
 - b. Placenta increta: the placenta invades the myometrium.
 - c. Placenta percreta: the placenta extends through the myometrium and may adhere to the surrounding structures.

Associated risk factors

1. Previous cesarean section
2. Current placenta previa

Diagnosis

1. Ultrasonography
2. Magnetic resonance imaging (MRI)
3. Often not recognized until the obstetrician has difficulty separating the placenta from the uterus

Obstetric Management

1. Arterial embolization under radiographic guidance
2. Cesarean hysterectomy



KO TREATMENT PLAN

Intra-operative

1. Good IV access
2. Arterial line
3. Possible central line with central venous pressure (CVP) monitoring to help guide fluid therapy
4. Type and cross for at least four units of packed red blood cells.

5. Although controversial in the parturient, one can consider cell salvage secondary to the associated massive blood loss.
6. General anesthesia is usually required for patient comfort and adequate operating conditions.
7. A continuous epidural can be used, but the timing of the epidural catheter removal is controversial secondary to the possibility of an associated massive hemorrhage and transfusion-precipitated disseminated intravascular coagulation (DIC).

UTERINE ATONY^{1,2,3}

CLINICAL ISSUES

Definition

1. Most common cause of early postpartum hemorrhage
2. Ineffective uterine muscle contraction
3. Occurs in 1 in 20 deliveries.

Risk Factors

1. Prolonged or rapid labor
2. Prolonged oxytocin use
3. Over-distended uterus: fetal macrosomia, multiple gestation, polyhydramnios
4. Infection: chorioamnionitis
5. Grand multiparity
6. Placenta previa
7. Administration of drugs that relax the uterus
 - a. Halogenated anesthetics
 - b. β -sympathetic agonists
 - c. Magnesium sulfate
 - d. Nitroglycerin
 - e. Tocolytic therapy

Diagnosis

1. Rapid uterine bleeding
2. Lack of myometrial tone



KO TREATMENT PLAN

1. Bimanual uterine massage
2. Uterotonic therapy

58. Obstetrical Postpartum Hemorrhage

- a. Oxytocin (Pitocin)
 - i. First-line therapy
 - ii. Given intravenous (IV) or intramuscular (IM)
 - iii. Side effects: hypotension, nausea, emesis, water intoxication
 - iv. Usually given by continuous intravenous infusion
 - b. Prostaglandins
 - i. $F_{2\alpha}$ (hemabate/carboprost)
 - (1) IM or intrauterine (IU)
 - (2) Side effects: nausea, vomiting, flushing, chills and bronchospasm
 - (3) Contraindicated in patients with active cardiac, pulmonary, renal, or hepatic disease
 - ii. E (misoprostol)
 - (1) Rectal (PR) or oral (PO)
 - (2) Side effects: tachycardia and fever
 - c. Methylergonovine (methergine)
 - i. IM or IU
 - ii. Blood vessel constrictor and smooth muscle agonist
 - iii. Side effects: profound hypertension, nausea, and vomiting
 - iv. Contraindicated in patients with chronic hypertension, pre-eclampsia, peripheral vascular disease, and ischemic heart disease.
3. When the uterine atony is due to a tocolytic therapy such as magnesium sulfate, treat with 1 g of calcium gluconate IV.
 4. Uterine tamponade
 5. Selective arterial embolization
 6. Surgical intervention
 - a. Arterial ligation
 - b. Uterine suturing
 - c. Hysterectomy

UTERINE INVERSION^{1,2,3}

CLINICAL ISSUES

Definition

1. Rare
2. Uterine fundus inverts through the cervix into the vagina.

Risk Factors

1. Uterine atony and over-distension
2. Fetal macrosomia
3. Prolonged labor
4. Uterine malformations
5. Short umbilical cord
6. Umbilical cord traction
7. Ehlers-Danlos syndrome
8. Inappropriate fundal pressure

Diagnosis

1. Hypotension is usually seen before significant blood loss.
2. Sudden onset of vaginal bleeding
3. Diagnosed with a bimanual exam and ultrasound



KO TREATMENT PLAN

1. IV fluid therapy.
2. Provide uterine relaxation.
 - a. β -sympathomimetic agents
 - b. Magnesium
 - c. Nitroglycerin
 - d. The agent of choice depends solely on the patient's hemodynamic status.
3. Restore the uterus to its normal position.
4. If these initial attempts fail, rapid-sequence induction with cricoid pressure, endotracheal intubation, and the administration of a halogenated anesthetic may be necessary to provide sufficient uterine relaxation.
5. Once the uterus is in place, oxytocin should be given to induce uterine contraction.

RETAINED PLACENTA AND PRODUCTS OF CONCEPTION^{2,3}

CLINICAL ISSUES

Definition

1. Placental tissue and amniotic membranes remain in the uterus after delivery and can inhibit the uterus from contracting adequately, resulting in hemorrhage.
2. Occurs in about 0.5–1.0% of deliveries

Associated Risk Factors

1. Mid-trimester delivery
2. Chorioamnionitis
3. Accessory placental lobe



KO TREATMENT PLAN

1. This usually only requires manual exploration and ultrasound diagnostics. This can be done in the birthing room if the patient has an epidural in place.
2. Some patients will require a uterine curettage in the operating room.
3. A spinal anesthetic is a good choice for patients who are not bleeding and did not have an epidural placed for labor.
4. In some cases, 50% nitrous oxide, or small doses of ketamine (10 mg boluses) or fentanyl (50–100 mcg), are adequate for the exploration and extraction.

5. The surgeon may require complete uterine relaxation. This can be accomplished with general anesthesia or nitroglycerin (IV or sublingual).

- a. Perform rapid-sequence induction of general anesthesia followed by high-dose volatile anesthetic to relax the uterus.
- b. Risk of failed intubation and aspiration

Non-uterine Causes of Postpartum Hemorrhage^{1,2}

1. Genital tract lacerations
2. Vulvar and vaginal hematoma

3. Retroperitoneal hematoma
4. Maternal coagulopathy

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2333–6.
2. Gabbe SG, Niebyl JR, Simpson JL. *Obstetrics: Normal and Problem Pregnancies*, 5th ed. Philadelphia: Churchill Livingstone & Elsevier, 2007, chapters 15 and 18.
3. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 119 and 662–76.

59. Other Obstetrical Complications^{1,2,3,4,5}

Jessica A. Lovich-Sapola, MD

UMBILICAL CORD PROLAPSE^{1,4}

SAMPLE CASE

A 25-year-old expectant female's cervix is 6 cm dilated. You have placed an epidural and are verifying the level when the obstetrical resident comes in to rupture the patient's membranes. Upon rupture, the fetal heart rate drops suddenly. The obstetrical resident notes that she can now feel the umbilical cord. What is your anesthetic plan for this emergent cesarean section?

CLINICAL ISSUES

Definition

1. The umbilical cord prolapses through the cervix.
2. This leads to significant cord compression. This can lead to sudden fetal bradycardia.⁵

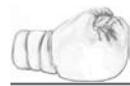
Incidence

1. Rare
2. 0.4% of cephalic term pregnancies
3. Increased risk with breech (6% risk) and transverse presentations (20% risk)
4. Increased risk with polyhydramnios

Diagnosis

1. Suspect cord prolapse when there is a sudden drop in the fetal heart rate immediately after the membranes are ruptured.

2. Confirm by palpation of the umbilical cord on vaginal exam.



KO TREATMENT PLAN

Pre-operative

1. Manual replacement of the cord
2. Fetal head elevation until operative delivery
3. Avoidance of cord compression by the presenting fetal parts
4. Urgent cesarean section

Intra-operative

1. Use a regional technique if an epidural catheter is in situ. In this patient, I would use my recently placed catheter. It is important to ask the obstetrical resident to monitor the fetal heart rate by palpating the cord.
2. General anesthesia is usually the first choice if there is associated fetal compromise. If the obstetrical resident noted a loss of fetal heart tones prior to achieving a proper level with the epidural catheter, I would induce general anesthesia. Always remember that the mother is your first priority. If you believe that she will be a difficult intubation, wait for your epidural level, or if this is inadequate, an awake intubation prior to general anesthesia may be required.

FETAL HEART RATE DECELERATIONS²

SAMPLE CASE

A 22-year-old female is scheduled for an emergency cesarean section at 37 weeks' gestation. She has pregnancy-induced hypertension. She has had a recent development of late decelerations of the fetal heart rate. Her blood pressure is 170/100 mm Hg and her hemoglobin is 9.5 gm/dL. What is a late deceleration? Is it significant? Are there other tests that can be done to assess fetal well-being?

CLINICAL ISSUES

1. Early decelerations

a. Definition:

- i. Shallow, symmetric, uniform decelerations with a gradual onset and return to baseline, resulting in a U-shaped deceleration²
- ii. They begin early in the contraction; their nadir coincides with the peak of the contraction and returns to baseline by the time the contraction is over.
- iii. Usually occurs when the patient's cervix is dilated between 4 and 6 cm
- iv. They do not indicate fetal hypoxia.
- v. Occur in 5–10% of all labors

b. Cause

- i. Fetal head compression by the uterine cervix as it overrides the anterior fontanel of the cranium
- ii. This results in altered fetal cerebral blood flow, precipitating a vagal reflex, with resultant slowing of the fetal heart rate.

2. Late Decelerations

a. Definition

- i. Gradual onset and return to baseline
- ii. U-shaped
- iii. Delayed in timing relative to the contraction
- iv. Begin about 30 seconds after the onset of the contraction or even at or after its peak
- v. The nadir is after the peak of the contraction.
- vi. Late decelerations *always* indicate fetal hypoxia.

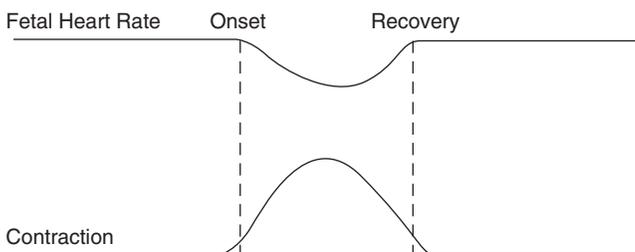


Figure 59.1. Early deceleration. Figure credit: J. Lovich-Sapola, MD.

b. Causes

- i. Uteroplacental insufficiency
- ii. Uteroplacental perfusion is temporarily interrupted during the peak of a strong contraction.
- iii. Any compromise in delivery, exchange, or uptake in fetal oxygen, other than umbilical cord compression, can result in a late deceleration if the insult is sufficient.¹
- iv. Excessive uterine contractions (seen with IV oxytocin)
- v. Spinal or epidural secondary to either systemic or local hypoperfusion/hypotension
- vi. Post-maturity
- vii. Maternal hypertension
- viii. Collagen vascular disease
- ix. Advanced diabetes mellitus
- x. Placental abruption
- xi. Severe maternal anemia or hypoxemia
- xii. Chronic fetal anemia

c. Diagnosis

i. Fetal scalp pH

- (1) Used to determine fetal acidosis
- (2) Technically difficult
- (3) Used infrequently
- (4) The patient must be at least 4–5 cm dilated, vertex, and –1 station.
- (5) A pH <7.20 is consistent with fetal acidosis and requires an emergent cesarean section.
- (6) A pH of 7.20–7.25 is considered borderline and should be repeated if the late deceleration pattern persists.
- (7) A pH greater than 7.25 is considered adequate for the fetus. Continue to closely monitor the mother and fetus, and repeat the pH as needed.

d. Treatment

- i. Call for help.
- ii. Administer oxygen through a tight fitting face mask.
- iii. Change the maternal position: lateral or knee-chest.
- iv. IV fluid bolus with lactated Ringer's solution
- v. Consider tocolysis to treat uterine tetany or hyperstimulation.
- vi. Discontinue the oxytocin infusion.
- vii. Consider an amnioinfusion.
- viii. Determine whether a cesarean section is required.
 - (1) In situ epidural, spinal, or general anesthesia are all viable options for the induction of anesthesia depending on the pattern and depth of decelerations.

3. Variable Decelerations

a. Definition

- i. Most common decelerations
- ii. Variable size, shape, depth, duration, and timing relative to the contraction
- iii. Caused by umbilical cord compression

59. Other Obstetrical Complications

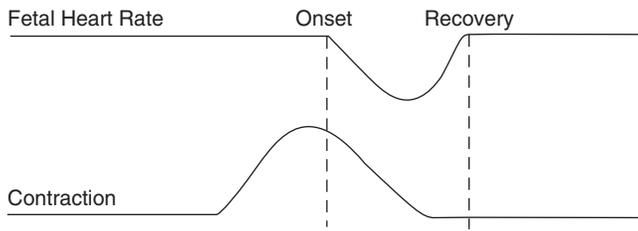


Figure 59.2. Late deceleration. Figure credit: J. Lovich-Sapola, MD.

- iv. Continued cord compression leads to a progressive increase in fetal CO₂ and a resulting respiratory acidosis. This can worsen over time into a combined respiratory and metabolic acidosis.
- v. Usually has a favorable outcome.
- b. Causes
 - i. Umbilical cord compression and stretch
 - ii. Initially a reflex response to changes in pressure and not hypoxia
 - iii. Can be seen in fetuses with no change in oxygen saturation.
 - iv. Oligohydramnios in early labor
 - v. Nuchal cord at 8–9 cm dilated
 - vi. Umbilical cord prolapse

MOLAR PREGNANCY³

SAMPLE CASE

A 32-year-old female presents with a 2 week history of lower abdominal pain, nausea, vomiting, and vaginal bleeding. She has a 16 week-sized uterus. She is scheduled for an evacuation of her molar pregnancy. What potential problems could be anticipated? Three hours after the evacuation, she complains of chest pain, cough, and tachypnea. What is the differential diagnosis? How would you diagnose and manage her care?

Definition

1. Vesicular swelling of placental (choroid) villi
2. Absence of an intact fetus
3. Gestational trophoblastic disease that originates in the placenta

Diagnosis

1. Ultrasound
2. Abnormally elevated human chorionic gonadotrophin (hCG) levels for gestational age

Symptoms and Signs

1. Abnormal bleeding in early pregnancy
2. Can mimic an incomplete or threatened abortion.
3. Large uterus for dates
4. Nausea and vomiting
5. Late first and early second trimester signs of toxemia (prior to 24 weeks)
6. Laboratory manifestations of hyperthyroidism
7. Lower abdominal pain
8. Absent fetal heart tones or fetal parts

Associated Complications

1. Anemia
2. Pregnancy-induced hypertension
3. Pulmonary insufficiency
 - a. Acute dyspnea
 - b. Cyanosis
4. Congestive heart failure
5. Hyperthyroidism/thyrototoxicosis
 - a. hCG is identical to a sub-unit of the thyroid stimulating hormone (TSH) but has a weak thyroid-stimulating effect in normal pregnancies.
 - b. Molar thyrotropin is responsible for the associated thyrototoxicosis with molar pregnancies.
6. Disseminated intravascular coagulation (DIC)
7. Pulmonary embolism (trophoblastic embolization)

Obstetrical Management

1. The molar pregnancy must be evacuated from the uterus using suction curettage.



KO TREATMENT PLAN

Pre-operative

1. Complete history and physical
2. Laboratory test
 - a. CBC: high risk for anemia secondary to vaginal bleeding
 - b. Electrolytes
 - c. Arterial blood gas
 - d. Thyroid function tests: high risk for thyrototoxicosis
 - e. Chest X-ray
3. Treat any associated hyperthyroidism with intravenous iodine and β -adrenergic receptor blockers.
 - a. It is not possible to get the patient into a euthyroid state prior to the surgical procedure.
 - b. Focus on controlling the sympathetic activity.
4. Avoid excessive fluid resuscitation to prevent pulmonary edema.

Intra-operative

1. General anesthesia is recommended.
 - a. Secure the airway with a rapid-sequence intubation.
 - b. Regional anesthesia is not recommended secondary to the associated DIC.
2. An arterial line is recommended for continuous monitoring of the patient's blood pressure and frequent blood draws.

Post-operative

1. The patient should be monitored closely in the intensive care unit (ICU) for 12-24 hours.
2. Observe for pulmonary embolus, congestive heart failure, DIC, and thyrotoxicosis.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2333-6.
2. Gabbe SG, Niebyl JR, Simpson JL. *Obstetrics: Normal and Problem Pregnancies*, 5th ed. Philadelphia: Churchill Livingstone & Elsevier, 2007, chapters 15 and 18.
3. Katz VL, Lentz GM, Lobo RA, et al. *Comprehensive Gynecology*, 5th ed. Philadelphia: Mosby Elsevier, 2007, chapter 35.
4. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 119 and 662-76.
5. Erol DD, Cevryoglu AS, Usulan I. Preoperative preparation and general anesthesia administration with sevoflurane in a patient who develops thyrotoxicosis and cardiogenic dysfunction due to a hydatidiform mole. *Internet J Anesthesiology (ISSN 1092-406X)* 2004;8(1).

60. The Pregnant Trauma Patient^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 25-year-old woman is brought to the trauma bay after injury in a motor vehicle accident (MVA). She was the restrained passenger in a head-on collision. She tells her caregivers she is 7 months pregnant and repeatedly asks "Is my baby ok?" She is tachycardic to 120 and moderately hypotensive at 92/56 mm Hg, with complaints of abdominal tenderness. What concerns do you have in the trauma bay? How do you want to proceed?

CLINICAL ISSUES**Incidence**

1. Approximately 5% to 10% of all pregnant women experience some type of trauma, with 8% occurring in the first trimester, 40% in the second trimester, and the remaining 52% in the third trimester.
2. The primary cause of fetal death is maternal death.
3. Ensuring adequate maternal care is the best method of ensuring adequate care of the fetus.

Issues Specific to the Care of a Pregnant Trauma Patient

1. Presence of the obstetrical (OB) team immediately in trauma bay

2. Need for fetal monitoring
 - a. Viable fetuses (estimated gestational age 24 weeks) are placed on continuous electronic fetal heart rate (FHR) monitoring in order to assess fetal heart rate variability.
 - i. This requires continuous monitoring by the OB team.
 - b. Fetal well-being is a reflection of adequate maternal blood flow and oxygenation.
 - i. Proportional to maternal mean arterial pressure
 - ii. Inversely proportional to uterine vascular resistance
 - c. Even if the estimated fetal age is nonviable, FHR tracings may provide information regarding adequate fetal perfusion and may indicate the need for more intensive maternal management.
 - d. Prolonged decreased FHR or late decelerations may indicate the need for an immediate cesarean section.
3. Radiologic studies
 - a. X-rays should be ordered on the basis of clinical need for information pertinent to diagnosis.
 - b. Radiation effects are most deleterious during organogenesis from weeks 2 to 7.
 - c. Avoid computerized tomography (CT) scans of the abdomen. If they are truly necessary, then it is advised to limit the area studied or the number of cuts.
4. Major obstetrical hemorrhage may be induced by trauma. (Please see the obstetrical hemorrhage chapters: Chapters 57 and 58.)
 - a. Includes

- i. Placental abruption
- ii. Uterine rupture
- b. Also consider non-trauma-related hemorrhage such as
 - i. Placenta previa
 - ii. Placenta accreta/increta/percreta
 - iii. Vasa previa
 - iv. Postpartum bleeding
 - (1) Retained placenta
 - (2) Uterine atony
 - (3) Cervical/vaginal lacerations



KO TREATMENT PLAN

Pre-operative

1. A complete understanding of the hematologic, cardiovascular, pulmonary, and gastrointestinal system alterations secondary to pregnancy is necessary. (Please read the appropriate chapter on physiology of pregnancy prior to proceeding with this review: Chapter 55.)
2. Consistent with review of all trauma patients, assessment begins with the ABCDs followed by a secondary survey. Special considerations in pregnant patients include

- a. Increased risk for aspiration due to lower esophageal sphincter relaxation and cephalad displacement of the stomach. It may be necessary to intubate the trachea quickly to avoid aspiration.
- b. Increased risk of difficult airway secondary to
 - i. Weight gain
 - ii. Increased breast tissue
 - iii. Airway edema
 - iv. Possible cervical neck and spine injuries from the trauma
- c. Need for lateral decubitus positioning to relieve aorto-caval compression by the gravid uterus.
 - i. Aortocaval compression decreases maternal systolic blood pressure by up to 30 mm Hg and decreases stroke volume by 30%
 - ii. If cardiopulmonary resuscitation is needed
 - (1) Supine positioning is necessary.
 - (2) Manual displacement of the gravid uterus to the left decreases uterine compression on the abdominal aorta and inferior vena cava.
 - (3) Delivery of fetus may need to be emergently performed in order to apply effective CPR and save the life of the mother.

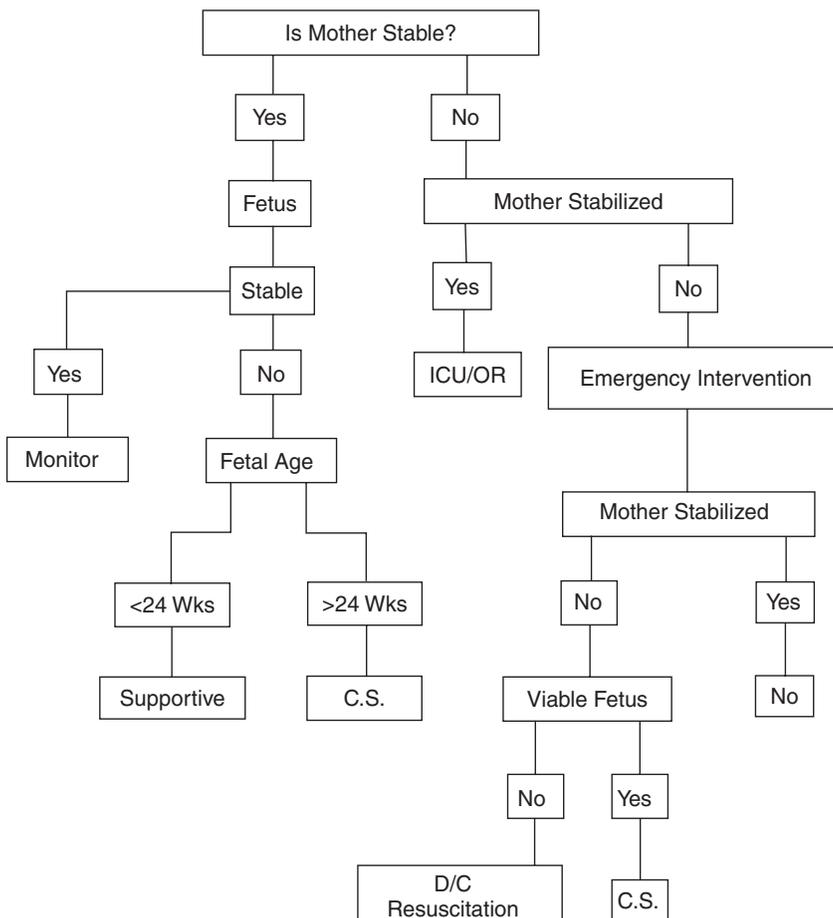


Figure 60.1. A treatment algorithm of trauma in pregnancy. Source: This figure has been reprinted with permission from *Emergency Medicine Clinics of North America*, Volume 5, JD Neufeld, EE Moore, JA Marx, and P Rosen, Trauma in Pregnancy, Page 623, Copyright Elsevier (1987).

3. No difference between pregnant and non-pregnant patients exists in the decision to proceed with a trauma-related emergency surgery.

Intra-operative

1. Consider regional techniques when feasible.
2. Goals are to maintain uteroplacental circulation and fetal oxygenation, with prevention of fetal distress.
3. Review intra-operative management of pregnant patients for review of the best drugs to use or avoid in pregnant surgical patients. Of note:
 - a. Hemodynamically unstable patients may be unable to tolerate volatile anesthetic agents during surgery.
 - b. Ketamine
 - i. Increases uterine tone and decreases uteroplacental perfusion, thus making it a sub-optimal drug, even in the situation of hemodynamic instability.
 - c. Avoid nitrous oxide if risk for pneumothorax exists.
4. Need for fetal resuscitation
 - a. FHR monitoring should be continued throughout the surgery if the location of the surgical site allows.

Post-operative

1. Fetal-maternal hemorrhage must be considered in cases of abdominal trauma.
 - a. Women who are Rh-negative must be treated with Rh (D)-immunoglobulin (Rhogam) within 72 hours to prevent maternal isoimmunization.
2. Continue FHR monitoring in the post-operative period.
3. Continue obstetrical team involvement.
 - a. Increased risk of preterm labor
 - b. Tocolytic administration may be necessary to prevent contractions leading to pre-term delivery.

BIBLIOGRAPHY

1. Hull SB, Bennett S. The pregnant trauma patient: Assessment and anesthetic management. *Int Anesthesiol Clin* 2007;45(3):1-18.
2. Neufeld JD, Moore EE, Marx JA, et al. Trauma in pregnancy. *Emerg Med Clin North Am* 1987;5:623.
3. Mercier FJ, Van de Velde M. Major obstetric hemorrhage. *Anesthesiology Clin* 2008;26:53-66.
4. Prentice-Bjerkeseeth R. Perioperative anesthetic management of trauma in pregnancy. *Anesth Clin North Am* 1999;17(1):278-295.

61. Difficult Obstetrical Airway^{1,2,3}

Kasia Petelenz Rubin, MD

SAMPLE CASE

An obese, 25-year-old woman, G1P0, presents for an elective cesarean section secondary to breech presentation of the fetus. She has a medical history significant for asthma and morbid obesity, with a BMI of 42. She has previously had a laparoscopic cholecystectomy, at which time she states that she awoke with an incredibly sore throat and a chipped front tooth. She states that she had been told there was some difficulty in putting her to sleep, but is unable to provide further details. What is your plan? Which would you recommend: regional anesthesia, or a general anesthetic approach? Assuming you have to induce general anesthesia, how will you secure the airway?

CLINICAL ISSUES

Difficult Intubation

1. Failed intubation in the general population is 1 in 2,330.
2. In the obstetric population it is quoted as 1 in 280.

Characteristics of an Obstetrical Airway Which Lead to an Increased Incidence of Difficult Intubations

1. There is capillary engorgement of the mucosa throughout the entire respiratory tract.
2. The Mallampati score changes due to increased pharyngeal edema and fatty infiltration of the pharyngeal tissue.
3. Associated morbid obesity
4. Pre-eclampsia (see Chapter 56)
5. Increased breast size

Aspiration

1. Patients are at high risk for pulmonary aspiration.
2. A delay in gastric emptying can be demonstrated by the end of the first trimester.
3. Lower esophageal sphincter tone is generally reduced.
4. The angle of the gastroesophageal junction is changed because the position of the stomach is shifted by the gravid uterus.

61. Obstetrical Difficult Airway

5. A rapid-sequence induction of anesthesia with proper cricoid pressure and use of a cuffed endotracheal tube is required for any general anesthetic after the 12th week of gestation.



KO TREATMENT PLAN

Intra-operative

1. Because of the airway problems that are associated with general anesthesia, regional anesthesia should be used when possible.
2. It is recommended that early intervention with the placement of a functioning regional anesthetic (labor epidural) will minimize emergency induction of general anesthesia in parturients with difficult airways.
3. In the event of an urgent or emergent cesarean section, establishment of a surgical block is often quicker than establishing adequate anesthesia via an awake intubation, or worse, securing an airway after a failed intubation.

Labor and a Known Difficult Airway

1. A functioning epidural block avoids the need for general anesthesia and resultant airway instrumentation.
2. In the case of an accidental dural puncture, thread a catheter into the spinal space, but use extreme caution in its dosing to avoid a high or total spinal anesthetic and its associated risk of emergent intubation.

Cesarean Section with a Known Difficult Airway

1. Aspiration prophylaxis in the form of H₂-blockers and/or non-particulate antacid should be provided to all patients prior to cesarean section.

2. An awake intubation allows for preservation of the natural airway and airway reflexes.
3. A nasal fiber optic intubation should be avoided due to the mucosal engorgement and the high risk of epistaxis, leading to a compromised airway.
4. With adequate psychological and pharmacological preparation, patients can generally tolerate an awake intubation.
5. Regional anesthesia is the best possible choice in most cases of an anticipated difficult airway.
6. In a non-urgent situation, slow titration of an epidural block allows for the avoidance of any major hemodynamic and respiratory compromise.
7. In a more time-pressed situation, spinal anesthesia allows for rapid establishment of surgical anesthesia.
8. Additional equipment should be immediately available to establish an airway in case a need arises. Equipment may include
 - a. Endotracheal tubes in smaller sizes, with stylets in place
 - b. Short handle laryngoscope
 - c. Laryngeal mask airway (LMAs) of varying sizes
 - d. Immediate availability of secondary airway equipment, based on practitioner familiarity, such as a fiber optic scope, Glidescope, and/or a tracheotomy tray.

Cesarean Section and an Unknown Difficult Airway

1. In an urgent situation when perhaps there is not enough time to administer a regional anesthetic, a general rapid sequence induction is performed.
2. If direct laryngoscopy fails, the return to spontaneous ventilation and awakening of the mother should be considered.
3. In most situations except for maternal hemorrhage and fetal distress, the mother can be reawakened.

Table 61.1. Failed Obstetric Intubation Recommendations

Failed intubation	Inadequate ventilation	Adequate ventilation
<ol style="list-style-type: none"> 1. Call for help. 2. Ventilate with 100% oxygen. <ol style="list-style-type: none"> a. Face mask and cricoid pressure b. LMA and cricoid pressure 	<ol style="list-style-type: none"> 1. Non-surgical airway <ol style="list-style-type: none"> a. LMA with cricoid b. Combitube c. Transtracheal jet ventilation 2. Surgical airway <ol style="list-style-type: none"> a. Cricothyrotomy b. Tracheotomy 3. Deliver the baby. 	<ol style="list-style-type: none"> 1. Assess the fetus 2. Fetal distress <ol style="list-style-type: none"> a. Spontaneous ventilation with sevoflurane and 100% oxygen b. LMA plus cricoid pressure c. Intubation through the LMA 3. No fetal distress <ol style="list-style-type: none"> a. Wake the patient b. Attempt an awake intubation or regional anesthesia

Adapted from Obstetric Difficult Airway Algorithm. In Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006, p 905.

4. If the surgery needs to proceed despite failed intubation, mask ventilation with cricoid pressure should be attempted. In the presence of fetal distress, the surgery may proceed.

5. As this situation progresses, the ASA Difficult Airway Algorithm should be followed, with progression from supra-glottic airway such as the LMA, the intubating LMA, to a potential “cannot ventilate, cannot intubate” situation when transtracheal jet ventilation, cricothyroidotomy, and surgical tracheotomy need to be considered.

6. None of these are popular techniques, but well thought-out algorithms and equipment must be available to deal with

airway emergencies during failed intubations of the obstetric patient.

BIBLIOGRAPHY

1. Munner U, deBoisblanc B, Sursh MS. Airway problems in pregnancy. *Crit Care Med*, 2005;33(10):SS259–68.
2. Mandal NG, Surapenei S. Regional anaesthesia in pre-eclampsia: Advantages and disadvantages. *Drugs*, 2004;64(3):223–36.
3. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006, p 905.

62. Anesthesia for a Vaginal Delivery^{1,2,3}

Alma Hoxha, MD

SAMPLE CASE

A 26-year-old female G₃P₂, 39 weeks pregnant comes to labor and delivery complaining of 6/10 pain with contractions and is asking for an epidural. Her medical history is only significant for the recent diagnosis of pre-eclampsia. Vital signs on admission include a blood pressure of 165/82 mm Hg, a heart rate of 98, a respiratory rate of 19, and a temperature of 36.3°C. What kind of analgesia would you offer to this patient? Do you have any concerns?

CLINICAL ISSUES

Stages of Labor

1. First stage of labor
 - a. Uterine contractions
 - b. Complete dilation of the cervix
 - c. Stretching of the lower uterine segment
 - d. Pain impulses are carried in visceral afferent C-fibers accompanying the sympathetic nerves.
 - i. Early stage: T11–L2
 - ii. Later stage: T10–L1
2. Second stage of labor
 - a. Full cervical dilation to the delivery of the fetus
 - b. Additional pain impulses
 - i. Distension of the vaginal vault and perineum
 - ii. Impulses are carried via the pudendal nerves (S2–S4).
3. Third stage
 - a. Delivery of the placenta

Well-Conducted Obstetrical Analgesia

1. Pain relief
2. Reduced anxiety
3. Many benefits for the mother
 - a. Blunts the increase in heart rate, cardiac output, and blood pressure.
 - b. Blunts the release of catecholamines: epinephrine, norepinephrine.
 - c. Pain relief may improve uterine blood flow.
 - d. Eliminates maternal hyperventilation.
 - i. Indirectly benefits the fetus.

Mechanisms to Treat Labor Pain

1. Psychologic techniques
 - a. Psychoprophylaxis
 - i. Lamaze: prepared childbirth
 - (1) Positive conditioning and education
 - ii. Works under the concept that a lack of knowledge, fear, and anxiety can increase a patient’s perception of pain.
 - b. Hypnosis
 - c. Acupuncture
 - d. Transcutaneous electrical nerve stimulation
2. Systemic medications
 - a. Opioids
 - i. Meperidine
 - (1) Given intravenously (IV): 25–50 mg
 - (a) Peak effect 5–10 minutes after administration
 - (2) Intramuscularly (IM): 50–100 mg

62. Anesthesia for a Vaginal Delivery

- (a) Peak effect 40–50 minutes after administration
 - (3) Most commonly used
 - (4) Good for the first stage of labor
 - (5) Side effects
 - (a) Nausea and vomiting
 - (b) Dose-related depression of ventilation
 - (c) Orthostatic hypotension
 - (d) Neonatal depression
 - (e) May decrease beat-to-beat variability of the fetus.
 - (f) The placenta transfers the active metabolite normeperidine, which has a long elimination half-life in neonates: 62 hours
 - (g) Fetal exposure to the drug is highest at 2–3 hours after the administration.
 - ii. Fentanyl and Remifentanyl
 - (1) Potent medications
 - (2) Limited use due to their short duration of action
 - (3) Patient-controlled analgesia (PCA) is a better choice than bolus administration, but PCA has the potential for accumulation and increased risk of neonatal depression.
 - iii. Morphine
 - (1) IV or IM administration
 - (2) Produces more respiratory depression than meperidine in equianalgesic doses
 - (3) Rarely used
 - (4) Used very early in labor, if used at all
 - iv. Butanorphanol and nalbuphine: given IV or IM
 - (1) Opioid agonist-antagonist
 - (2) Decreased incidence of nausea and vomiting
 - (3) Decreased dysphoria
 - (4) Ceiling effect on the depression of ventilation
 - (5) Side effects: high incidence of maternal sedation
 - b. Ketamine
 - i. Potent analgesic
 - ii. Side effects: unacceptable amnesia
 - iii. If used at 0.2–0.4 mg/kg, it gives adequate analgesia with no neonatal depression.
 - c. Sedatives
 - i. Recommended only in the very early phase of labor when delivery is not anticipated for more than 12–24 hours secondary to the associated significant fetal respiratory depression
 - (1) Promethazine
 - (a) Relieves anxiety
 - (b) Decreases the opioid requirements
 - (c) Controls emesis
 - (2) Benzodiazepines
 - (a) Rarely used
 - (b) Sedation and anxiolysis
 - (c) Should be given in very small doses
 - (d) Usually recommended for use after the delivery of the fetus
 - d. Inhaled anesthetics
 - i. Nitrous oxide: rare
 - 3. Regional
 - a. Most effective techniques
 - b. Allows the mother to be awake and enjoy her labor.
 - c. Complications
 - i. Hypotension
 - ii. Cardiac arrest
 - iii. Nerve damage
 - iv. Total spinal anesthesia
 - v. Postdural puncture headache
 - d. Contraindications
 - i. Absolute
 - (1) Patient refusal
 - (2) Bacteremia/sepsis
 - (3) Increased intra-cranial pressure
 - (4) Infection at the needle insertion site
 - (5) Shock or severe hypovolemia
 - (6) Coagulopathy or therapeutic anticoagulation
 - ii. Relative
 - (1) Pre-existing neurologic disease
 - (2) Severe psychiatric disease or dementia
 - (3) Aortic stenosis
 - (4) Left ventricular outflow tract (LVOT) obstruction
 - (5) Various congenital heart conditions (Please see the related Chapter 65.)
 - (6) Deformities or previous surgery of spinal column
 - e. Patient preparation
 - i. Start an intravenous line: 500 ml saline bolus.
 - ii. Have resuscitation equipment available.
 - iii. Medications
 - (1) Benzodiazepines/thiopental: seizures
 - (2) Ephedrine/phenylephrine: hypotension
 - (3) Naloxone: respiratory depression
 - iv. Complete history and physical examination
 - v. Understanding of the obstetric plan
 - vi. Understanding of the fetal status
 - vii. Appropriate monitors
 - viii. Informed consent
- ### Spinal Analgesia
- 1. Rarely used for a vaginal delivery
 - a. More commonly used for a cesarean section
 - 2. Fast and reliable onset
 - 3. Bolus or continuous
 - 4. Opioid alone or in combination with bupivacaine
 - a. Most commonly lidocaine, bupivacaine, or tetracaine is used.
 - b. Lidocaine
 - i. Quickest onset and shortest duration of action
 - ii. Transient neurologic symptoms (TNS) reported in 13–36% of the patients receiving spinal lidocaine especially for procedures performed in the lithotomy position.

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- c. Low-dose opioids can produce analgesia without a motor block or systemic opioid effects.
 - i. Morphine
 - ii. Sufentanil
 - iii. Fentanyl
 - iv. Meperidine
5. Risk of postdural puncture headache
6. The physiologic effects of a spinal anesthetic depend on the level of anesthesia, which itself depends on
 - a. Volume of solution injected into the subarachnoid space
 - b. The concentration of the agent in the solution
 - c. The speed of injection of the solution
 - d. The site of injection
 - e. The specific gravity of the solution
 - f. Position of the patient
 - i. A saddle block (sacral nerve root block) occurs when the patient is allowed to sit up after the spinal anesthetic is placed so that the sacral nerve roots are blocked resulting in anesthesia of the buttocks, perineum, and inner thighs. This can be used for an instrumental delivery.
 - g. Barbotage
 - i. Repeated injection and aspiration of a spinal anesthetic
 - h. Presence of increased intra-abdominal pressure
 - i. Height of the patient
7. **The interruption of the spinal cord function begins caudally and proceeds in a cephalic direction.**
 - a. It starts with the loss of the autonomic function followed by sensory and then motor function.
8. Physiological effects of spinal anesthesia
 - a. Cardiovascular
 - i. There is a 15–20% decrease in mean arterial pressure (MAP), central venous pressure (CVP), and total peripheral resistance in normal nonmedicated volunteers having spinal anesthesia.
 - ii. Minimal reduction in cardiac output (CO), stroke volume (SV), and heart rate (HR)
 - b. Respiratory
 - i. If the phrenic nerve is intact, resting ventilation is not impaired.
 - ii. If all spinal nerve roots are blocked, inspiratory capacity is decreased by 20%, and the expiratory reserve volume (ERV) is decreased to zero.
 - c. Cerebral blood flow (CBF)
 - i. CBF is maintained due to autoregulation during spinal anesthesia as long as the MAP is kept above 60 mm Hg; the MAP should be higher in patients with hypertension and atherosclerotic disease.
 - d. Renal and genitourinary
 - i. Decrease in renal blood flow (RBF) secondary to arterial hypotension
 - ii. With a high spinal there is a 5–10% decrease in the glomerular filtration rate (GFR) and RBF.
- iii. The bladder is the last organ to recover from spinal anesthesia, making urinary retention very common.

Epidural Analgesia

1. Most common regional anesthetic used for a vaginal delivery
2. Blocks pain from the first stage (T10–L1) and second stage (S2–S4) of labor.
3. Benefits
 - a. Effective pain relief
 - b. Reduction in maternal catecholamines
 - c. The technique can be used for surgical anesthesia if the patient suddenly requires a cesarean section.
4. Epidural test dose
 - a. Used to verify the proper placement of the epidural
 - b. Injected after a negative aspiration test
 - c. It consists of 45 mg of lidocaine and a 1:200,000 concentration of epinephrine.
 - i. Subarachnoid injection results in leg weakness and evidence of an anesthetic block.
 - (a) A high spinal can occur even with this low concentration of lidocaine if the patient lies flat.
 - (b) Be prepared to treat a high spinal.
 - ii. Intravascular injection presents with tachycardia, tinnitus (ringing in the ears), metallic taste in the mouth, and perioral numbness.
5. Epidural opioids
 - a. Meperidine: 100 mg provides good analgesia that lasts for 2.5 hours.
 - b. Fentanyl: 100–200 mcg provides analgesia that lasts for 1–2 hours with few side effects.
6. A local anesthetic should be selected on the basis of
 - a. Speed of onset
 - b. Degree of motor blockade
 - c. Duration of the surgical procedure
7. Bupivacaine, ropivacaine, lidocaine, and 2-chloroprocaine are the most commonly used local anesthetics.
 - a. The dose should be 1–1.5 mL per segment blocked, with a reduced dose in parturient, elderly, and obese patients.
 - b. Adding epinephrine can prolong the duration of a lidocaine nerve block by 50%.
 - i. Less dramatic results occur with bupivacaine.
8. Local anesthetics
 - a. Bupivacaine
 - i. Most commonly used
 - ii. Relatively long duration of action
 - iii. Lack of tachyphylaxis
 - iv. Low placental passage secondary to its being highly protein bound
 - v. Effective at low concentrations, especially if combined with an opioid
 - vi. Risk: cardiotoxicity
 - b. Lidocaine
 - i. Used more often for a cesarean section than for labor pain relief

62. Anesthesia for a Vaginal Delivery

- c. 2-chloroprocaine
 - i. Rapid onset
 - ii. Brief duration of action
 - iii. Very little drug crosses the placenta.
 - iv. Has significant antagonism with subsequently injected epidural opioids or bupivacaine
 - d. Ropivacaine
 - i. Amide local anesthetic similar to bupivacaine
 - ii. Less cardiotoxic than bupivacaine
9. Complications
- a. Accidental dural puncture
 - b. Intravascular injection
 - c. Hypotension
 - d. High spinal
 - e. Epidural abscess and hematoma

Combined Spinal-Epidural Analgesia (CSE)

1. The CSE is the placement of a catheter into the epidural space along with a single shot administration of intrathecal anesthetics/analgesics.
2. Benefits
 - a. Rapid onset
 - b. Reliability
 - c. Minimal drug toxicity
 - d. Flexibility of dosing
 - e. Duration
 - f. Analgesic level of control
3. There is a 30% reduction of medication used with a CSE when compared to an epidural.
4. Caution should be taken when placing a CSE in an obese patient and patients with poor airways: high spinal risk.
5. Complications are the same as those with epidural and spinal anesthesia.

Paracervical Block

1. Relieves pain during the first stage of labor.
2. Associated with a high incidence of fetal asphyxia and poor neonatal outcomes
 - a. Uterine artery constriction
 - b. Increased uterine tone

Paravertebral Lumbar Sympathetic Block

1. Can be used if there is a contraindication to a central neuraxial technique.
2. Good for the first stages of labor
3. Decreased risk of fetal bradycardia when compared to the paracervical block
4. Increased risk of intravascular injection

Pudendal Nerve Block

1. Pudendal nerves are derived from the lower sacral roots (S2–S4).

- a. Vaginal vault, perineum, rectum, and bladder
2. Good for a forceps delivery or an episiotomy repair



KO TREATMENT PLAN

Pre-operative

1. Once the diagnosis of pre-eclampsia is made it is important to get laboratory values.
 - a. Platelet count
 - b. Liver function tests
 - c. Coagulation panel
2. Vaginal delivery may be performed if the fetus is not distressed.
3. Lumbar epidural analgesia provides pain relief and a method to help control the patient's blood pressure during labor.
 - a. Rule out coagulopathy.
 - b. Optimize the patient's fluid status.
 - i. Bupivacaine, fentanyl, and dilute epinephrine are given as an initial bolus, and then as a continuous infusion.
4. With regional anesthesia there is
 - a. Less increase in blood pressure
 - b. Less need for opioid analgesics
 - c. Improvement in placental and renal blood flow

Intra-operative

(See the pre-eclampsia chapter, Chapter 56, for more information.)

1. Cesarean section is indicated for delivery of a distressed fetus.
2. If an epidural catheter has been previously placed, it may be used to provide surgical anesthesia, assuming the patient's volume status has been optimized.
3. Although previously considered controversial, spinal anesthesia has been proven to be a safe technique for cesarean section for severely pre-eclamptic patients.
4. General anesthesia is an acceptable way to manage pre-eclamptic patients, but there are associated risks of pulmonary aspiration, airway compromise from edema, and acute blood pressure elevation during laryngoscopy.
 - a. This can lead to a significant risk of cerebral hemorrhage and pulmonary edema.
 - b. A rapid-sequence induction technique should be used.
 - i. Intravenous hydralazine, lidocaine, sodium nitroprusside, nitroglycerine, and/or esmolol can be used to attenuate the hypertensive response to laryngoscopy.
 - ii. Sodium thiopental (4 mg/kg) plus succinylcholine (1.5 mg/kg) intravenously
 - iii. Anesthesia can be maintained with a volatile agent, N₂O/O₂, and neuromuscular blockade as needed, guided by the peripheral nerve stimulator.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1158–60.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2315–23.
3. Faust RJ, Cucchiara RF. *Anesthesiology Review*, 3rd ed. Philadelphia: Churchill Livingstone, 2002, pp 430–31, 438–40, 444–45.

63. Anesthesia for a Cesarean Delivery^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 30-year-old patient with a history of asthma presents for an urgent cesarean section due to fetal breech positioning. She is in active labor, and 6cm dilated. Would you perform general or regional anesthesia?

CLINICAL ISSUES¹

Incidence of Cesarean Delivery

1. Most common hospital-based surgical procedure in the United States
2. 25% of all live births

Indications for a Cesarean Delivery

1. Fetal distress
2. Elective repeat cesarean sections
3. Failure to progress (i.e., arrest of active phase of labor)
4. Non-reassuring fetal status
5. Cephalopelvic disproportion
6. Malpresentation
7. Prematurity
8. Previous uterine surgery

REGIONAL ANESTHESIA^{1,2,3}

Advantages

1. Decreased risk of failed intubation and aspiration
2. Avoidance of depressant agents
3. Ability of the mother to remain awake and enjoy the birth of her child
4. Earlier attempts at breast-feeding
5. Reduced blood loss

SPINAL ANESTHESIA^{1,2,3}

Advantages

1. Used for most straightforward cesarean sections.
2. Rapid onset
3. Superior, dense block
4. Cost-effective
5. Little risk of anesthetic toxicity secondary to the low doses used
6. Minimal drug transfer to the fetus
7. Failure rate/patchy blocks are very rare.
8. Decreases risk of gastric aspiration.
9. Mother can be awake for the delivery of her baby.

Disadvantages

1. Duration is limited.
2. High incidence of hypotension

Technique

1. Hyperbaric bupivacaine (0.75%, 1.6–1.8 mL)
 - a. Duration is approximately 1.5–2.5 hours.
 - b. Usually use a set dose of 12–15 mg.
 - c. Patient's height, weight, and body mass index do not tend to correlate with the height of the block.
 - d. Doses greater than 15 mg are not recommended secondary to the increased risk of complications, including a high block.
 - e. The addition of 10–20 mcg of fentanyl to the spinal can decrease intra-operative nausea and decrease the visceral pain associated with the exteriorization of the uterus and visceral traction.⁴
 - f. 0.1–0.25 mg of preservative-free morphine can also be added to the spinal for post-operative pain control.

63. Anesthesia for a Cesarean Delivery

2. Prehydration with up to 20 mL/kg of crystalloid solution
3. Position
 - a. Sitting
 - i. Optimal for obese patients
 - b. Lateral
4. Goal is a bilateral T4 level

EPIDURAL ANESTHESIA^{1,2,3}

Advantages

1. Offers flexibility
 - a. Good for a prolonged cesarean section
 - b. The anesthesiologist is able to titrate the dose to the desired level.
2. A previously placed epidural catheter can be used for labor pain.

Disadvantage

1. Requires a large dose of local anesthetic, compared to a spinal, which can be potentially toxic
2. Catheter location can migrate into the intra-thecal space or a blood vessel.
3. Slower onset of action

Technique

1. Agents: 3% 2-chloroprocaine, 2% lidocaine with epinephrine, 0.5% bupivacaine, and 0.5% ropivacaine
2. Adjunct agents added to improve the quality of the block
 - a. Bicarbonate: shortens the onset time (not effective for bupivacaine).
 - b. Epinephrine (1:200,000 or 1:400,000): decreases the vascular absorption of the local anesthetic, improves the quality of the anesthesia, prolongs the duration of the block and shortens the onset time (not effective for bupivacaine).
 - c. Fentanyl 50–100 mcg or sufentanil 10–20 mcg improves operative conditions by decreasing the patient's sensitivity to visceral stimulation.

COMBINED SPINAL-EPIDURAL TECHNIQUE (CSE)^{1,3}

Advantages

1. Rapid onset of a dense surgical anesthetic while allowing the ability to prolong the block with an epidural catheter
2. The block can be supplemented at any time.

Disadvantages

1. Difficulty interpreting a “test dose” in the catheter
2. Possibility of a failed epidural catheter

3. Risk of enhanced spread of the previously injected spinal drug after the use of the catheter
4. Some consider it to be cumbersome and time-consuming.

CONTINUOUS SPINAL ANESTHESIA¹

Advantages

1. Small doses can be given in an incremental fashion.
2. Good for patients with cardiac disease, respiratory disease, morbid obesity, and neuromuscular disease

Disadvantages

1. Postdural puncture headache

GENERAL ANESTHESIA^{1,2,3}

Indications

1. Acute maternal hemorrhage and hypovolemia
2. Overt coagulopathy
3. Life-threatening fetal compromise may occur without an emergent cesarean section in a patient without an existing labor epidural.
4. Patient refusal of regional anesthesia or inadequate regional anesthesia

Advantages

1. Rapid speed of induction
2. Control of the airway
3. Good when intense uterine relaxation is necessary: breech and transverse presentation

Disadvantages

1. Increased mortality compared to a regional anesthetic for a cesarean section
2. Risk of failed intubation
3. Pulmonary aspiration of gastric contents
4. Neonatal depression
5. Maternal awareness
6. Increased risk in patients with asthma, upper respiratory infections, obesity, or a history of difficult intubation.

FAILED INTUBATION IN PREGNANCY^{1,2,3}

Increased Risk of Failed Intubation in Pregnancy Is Secondary to

1. Weight gain
2. Enlarged breasts
3. Oropharyngeal edema
4. Pre-eclampsia

Failed Intubation Treatment Algorithm

1. Induce general anesthesia.
2. Failure to intubate
3. Can you mask ventilate?
 - a. No: ASA emergency airway algorithm (laryngeal mask airway (LMA), surgical airway, or awaken the patient).
 - b. Yes:
 - i. Fetal distress: mask or LMA with cricoid pressure, surgical airway, or if ventilation is still difficult, attempt to awaken the patient. (Remember that the mother's life is always your first priority.)
 - ii. No fetal distress: awaken the patient and perform a regional anesthetic or an awake fiber optic intubation.

Induction of General Anesthesia

1. Verify good IV access and open IV to run full speed.
2. Non-particulate antacid
3. Pre-oxygenate (3–5 minutes)
4. Left uterine displacement
5. IV Metoclopramide (10 mg) and Ondansetron (4 mg)
6. Apply all standard ASA monitors.
7. Have the surgeon perform a sterile preparation and draping of the patient so that he or she is ready to cut as soon as the patient is intubated.
8. Assuming the airway exam appears normal, a rapid-sequence induction is performed using thiopental (4 mg/kg), propofol (2 mg/kg), or ketamine (up to 1 mg/kg) followed by succinylcholine (1–1.5 mg/kg).
9. Rocuronium is a suitable alternative if succinylcholine is contraindicated (0.6 mg/kg).
10. Apply cricoid pressure and maintain it until the endotracheal tube is secured and its placement is verified.
11. Maintenance anesthesia can be provided with 50% nitrous oxide and 50% oxygen plus 0.5 MAC of a volatile anesthetic.

This can be increased to 2 MAC temporarily just prior to the delivery to aid in uterine relaxation.

12. After delivery, increase the percentage of nitrous oxide to 70 and decrease the volatile anesthetic. (High doses of volatile anesthetics can lead to uterine relaxation and bleeding post-delivery.) Add an IV opioid and a benzodiazepine as needed.



KO TREATMENT PLAN

1. For this patient with a history of asthma and an emergent cesarean section for breech positioning, I would perform a spinal. She is otherwise healthy, so you can assume that she has no coagulopathy issues. A spinal can be done quickly in the sitting or lying position.
2. Remember, since she has asthma, she may be more sensitive to the decreased sensation of breathing with the T4 spinal level. Monitor her oxygen saturation. Treat with albuterol as needed.
3. General anesthesia could be done safely if you are unable to place a spinal/epidural, or if she refuses regional anesthesia.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2323–7.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1160–2.
3. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 421–38.
4. Manullang GTR, Christopher MV, Pace NL. Intrathecal fentanyl is superior to intravenous ondansetron for the prevention of perioperative nausea during cesarean delivery with spinal anesthesia. *Anesth Anal*, 2000;90:1162–6.

64. Complications of Regional Anesthesia^{1,2,3,4,5,6}

Jessica A. Lovich-Sapola, MD

HYPOTENSION^{1,2,6}

SAMPLE CASE

You have just placed an epidural in a 19-year-old pregnant female with no prior medical history. After a negative test dose, the epidural is bolused with a bupivacaine and fentanyl mixture. After 10 minutes, the patient's blood pressure is 80/40 mm Hg. The fetal monitor is showing late decelerations. What do you do?

CLINICAL ISSUES

Definition of Hypotension

1. Systolic blood pressure decrease to less than 100 mm Hg or to more than 20–25% less than baseline readings.
2. The incidence and severity depend on the height of the block and the position of the patient.

Causes

1. Increased venous capacitance and pooling of a major portion of the blood volume in the lower extremities and splanchnic bed⁶
2. Decreased systemic vascular resistance

Prophylactic Actions

1. IV fluid bolus up to 20 mL/kg of lactated Ringer's solution prior to the placement of the spinal or epidural
2. Left uterine displacement to avoid aortocaval compression
3. Vigilant monitoring of the blood pressure at frequent intervals after the placement of the regional anesthetic
4. Monitor the fetal heart rate.
5. Prophylactic IV vasopressors as needed: ephedrine or phenylephrine



KO TREATMENT PLAN

1. Rapid IV fluid bolus
2. Give 100% oxygen by mask.
3. Increased left uterine displacement

4. Trendelenburg positioning
5. IV bolus of phenylephrine (100–200 mcg increments) or ephedrine (5–10 mg increments)
6. Evaluate the level of the block.
7. Call the obstetrical service for assessment of the mother and fetus.

ACCIDENTAL DURAL PUNCTURE^{1,2}

CLINICAL ISSUES

Definition

1. Accidental puncture of the dura mater
2. "Wet tap"
3. Incidence is 3%.
4. Can lead to postdural puncture headaches (PDPH) in 50–70% of cases.



KO TREATMENT PLAN

1. The traditional method was to reposition the epidural at a different interspace.
2. A recent recommendation is to pass the epidural catheter into the spinal space and use a continuous spinal technique.
 - a. Rapid and effective pain relief
 - b. Significant reduction in the PDPH and the need for an epidural blood patch
 - c. Excellent labor analgesia
 - d. Almost instant onset for anesthesia for a cesarean section

Techniques to Decrease the Risk of PDPH after an Accidental Dural Puncture

1. Inject CSF from the epidural syringe back into the subarachnoid space through the epidural needle.
2. Insert the epidural catheter into the subarachnoid space.
3. Administer continuous intrathecal labor analgesia.
4. Leave the catheter in for a total of 12–24 hours.
5. Inject preservative-free normal saline through the catheter prior to its removal.

POSTDURAL PUNCTURE HEADACHE (PDPH)^{1,2}**CLINICAL ISSUES****Incidence**

1. Pregnant women are at a higher risk of developing a PDPH secondary to their age and gender.
2. Incidence decreases with increasing age.
3. Incidence decreases with the use of a small diameter spinal needle.
4. Women are at a greater risk than men.

Postpartum Headache Differential Diagnosis

1. Headaches occur in 15% of pregnant patients who do not receive an epidural.
2. Headaches occur in 12% of pregnant patients who receive an epidural but show no sign of a dural puncture.
3. Nonspecific headache
4. Migraine
5. Hypertension
6. Pneumocephalus can occur if air is used for the loss of resistance (LOR) technique during the epidural placement.
7. Infection (sinusitis, meningitis)
8. Cortical vein thrombosis
9. Intracerebral pathology
10. Caffeine withdrawal
11. PDPH

Definition of PDPH

1. Headache after a dural puncture
2. Headache is worsened by standing or sitting.
3. Headache is relieved by lying down.
4. The headache is characteristically fronto-occipital.
5. Can have associated cranial nerve symptoms (diplopia, tinnitis)
6. Some patients have nausea and vomiting.
7. The headache results from the loss of CSF through the meningeal needle hole resulting in decreased support for the brain. When upright, the brain sags and puts traction on the cranial nerves and pain-sensitive structures.

**KO TREATMENT PLAN**

Conservative treatment: (indicated for mild to moderate discomfort. The symptoms usually resolve in a few days to a week if untreated.)

1. Bed rest
2. Hydration
3. IV caffeine (500 mg) or oral caffeine (300–500 mg)
 - a. Cerebral vasoconstrictor
 - b. Transient relief

c. Side effects: unable to sleep, anxious, seizures, and cardiac arrhythmias

4. Analgesics
5. Vasopressin
6. Theophylline
 - a. Cerebral vasoconstrictor
7. Sumatriptan
 - a. Serotonin agonist
 - b. Cerebral vasoconstrictor
8. Adrenocorticotrophic hormone (ACTH)

Epidural Blood Patch

1. Used if the patient's symptoms are severe enough to limit her activity, if cranial nerve involvement is noted, or if conservative management has failed
2. Most effective treatment for PDPH
3. Success rate of 75%
4. Immediate relief occurs in almost all patients, but only 61% have a permanent cure.
5. About 90% of patients who fail the first blood patch have good results with the second.
6. Possible mechanism
 - a. Clotted blood obstructs the dural tear, thereby decreasing the further CSF leak.
 - b. CSF pressure and cerebral vasoconstriction increase.

Technique

1. Sterile epidural
2. Draw 20 ml of the patient's blood using a sterile technique.
3. Most practitioners inject 10–20 ml of blood and stop injecting if the patient has pain.
4. Place the blood patch as close as possible to the original dural puncture.
5. The patient should lie flat for at least an hour after the procedure.
6. Avoid heavy lifting for the next week.
7. The procedure may be repeated as necessary.

Side Effects of an Epidural Blood Patch

1. Backache
2. Radicular pain
3. Transient bradycardia
4. Cranial nerve palsies

TOTAL SPINAL BLOCK^{1,2,6}**CLINICAL ISSUES****Definition**

1. Rare
2. Very serious complication

64. Complications of Regional Anesthesia

3. Excessive cephalad spread of the local anesthetic in the subarachnoid space
4. The local anesthetic spreads high enough to block the entire spinal cord and occasionally the brainstem.

Signs and Symptoms

1. Profound hypotension (complete sympathetic block)
2. Bradycardia (complete sympathetic block)
3. Respiratory arrest
4. Complete sensory and motor blockade
5. Hypotension
6. Unconsciousness
7. Loss of protective airway reflexes

Causes

1. Can result from a single-shot spinal or inadvertent intrathecal spread after an accidental dural puncture or catheter migration.
2. Subdural spread can also result in a high block.
 - a. High sensory level
 - b. Sacral sparing
 - c. Incomplete or absent motor block
3. Single-shot spinal anesthetic after a failed spinal or patchy epidural; therefore a spinal should not be performed after a failed spinal/epidural.



KO TREATMENT PLAN

1. Rapidly secure the airway with an endotracheal tube.
2. Positive pressure ventilation with 100% oxygen
3. Treatment of the associated hypotension
 - a. Left uterine displacement
 - b. Trendelenburg positioning
 - c. IV fluids
 - d. Vasopressors (ephedrine or epinephrine)
 - e. Atropine

ACCIDENTAL INTRAVASCULAR INJECTION²

SAMPLE CASE

An 18-year-old healthy patient is receiving an epidural for labor pain relief. You identify the epidural space, place the epidural catheter, and give a test dose. Despite your negative test dose, the patient has a grand mal seizure after the initial bolus of bupivacaine and fentanyl. What is your differential diagnosis? How will you treat this problem? Would you recommend a cesarean section?

CLINICAL ISSUES

Consequences of an Accidental Intravascular Injection of Local Anesthetic

1. Life-threatening convulsions
2. Cardiovascular collapse (increased risk with bupivacaine)

Differential Diagnosis of Seizures in Pregnancy

1. Eclampsia
2. History of previous seizures
3. Intravascular local anesthetic toxicity
4. Syncope
5. Hypoglycemia



KO TREATMENT PLAN

1. Primarily supportive
2. Stop any injection of local anesthetic.
3. Secure the airway and maintain oxygenation.
4. Treat the seizure with IV thiopental (50–100 mg), midazolam (2–5 mg), diazepam (5–10 mg), or propofol (1 mg/kg).
5. Succinylcholine can terminate the muscular activity to facilitate ventilation, but remember that it does not terminate the seizure.
6. Mild cardiovascular depression caused by lidocaine that leads to hypotension and bradycardia can be treated with IV ephedrine (10–30 mg) and IV atropine (0.4 mg).
7. If cardiovascular collapse occurs, likely secondary to bupivacaine, an emergent cesarean section may be necessary to relieve aortocaval compression and improve the efficacy of cardiac massage.
8. Resuscitation may require large doses of epinephrine, vasopressin, and amiodarone.
9. Intravenous lipids have been recommended. Use a 20% lipid solution at a dose of 4 mL/kg followed by a 0.5 mL/kg/minute infusion for 10 minutes.
10. Some patients have required cardiopulmonary bypass until the local anesthetic can be metabolized.

NEUROLOGIC COMPLICATIONS^{1,2,6}

CLINICAL ISSUES

Incidence

1. Rare
2. Neurologic complications are five times more common after childbirth itself than after regional blockade.
3. Most neurologic injuries that occur after delivery are obstetric in origin.
4. Postpartum back pain is equally as common in patients who receive neuraxial anesthesia and those who do not.

64. Complications of Regional Anesthesia

Causes

1. Related to anesthesia
 - a. Epidural hematoma
 - b. Epidural abscess
 - c. Localized tenderness
 - d. Nerve root irritation (may last weeks or months)
2. Unrelated to anesthesia
 - a. Obstetrical instrumentation
 - b. Nonanatomic positioning during labor
 - i. Lithotomy
 - c. Compression of sacral nerve roots by the fetal head during delivery

Compression of the Lumbosacral Trunk (L4–5)

1. Presents with foot drop
2. Weakness with ankle dorsiflexion
3. L5 sensory deficits
4. Causes
 - a. Cephalopelvic disproportion
 - b. Prolonged labor
 - c. Difficult vaginal delivery

Obturator Nerve Palsy

1. Presents with weakness of hip adduction and internal rotation
2. Sensory deficit over the inner thigh

Femoral Nerve Palsy

1. Presents with inability to climb stairs
2. Causes
 - a. Prolonged flexion
 - b. Prolonged abduction
 - c. Prolonged external rotation of the hips
 - d. Excessive lithotomy position

Meralgia Paresthetica

1. Neuropathy of the lateral femoral cutaneous nerve
2. It is a purely sensory nerve.
3. It presents with numbness, tingling, and/or burning over the anterolateral aspect of the thigh.
4. Causes
 - a. Pregnancy (increased intra-abdominal pressure)
 - b. Retractors during pelvic surgery

Sciatic Nerve Palsy

1. Presents with a loss of sensation below the knee with sparing of the medial side and an absence of movement below the knee
2. Causes
 - a. Excessive sitting in one position

Peroneal Nerve Palsy

1. Presents with foot drop, but the plantar flexion and inversion is preserved unlike an L4–5 lesion.⁶
2. Causes
 - a. Regional anesthesia
 - b. Lithotomy position
 - c. Improper or prolonged positioning in the stirrups
 - d. Compression of the lateral knee against any hard object



KO TREATMENT PLAN

1. Go to see the patient.
2. Thorough history and physical
3. Neurological assessment
4. Rule out an epidural hematoma or abscess.
5. Neurology consult may be necessary.

LUMBAR EPIDURAL HEMATOMA^{1,2,3,4,6}

SAMPLE CASE

A 23-year-old female requested a lumbar epidural for labor pain relief. The patient's labor was progressing rapidly, so your colleague placed an epidural without any pre-procedure labs. The next morning you are called to see the patient. She is complaining of severe back pain and leg weakness and numbness that has not resolved since her epidural was removed. What should you do?

Clinical Issues

1. Rare complication (1 in 150,000)
2. A hemostatic abnormality was present in 68% of the reported patients with an epidural hematoma.
3. Significantly increased risk with associated anticoagulant medications
4. Difficult or bloody placement of needles and catheters occurred in 25% of the reported cases of epidural hematoma.
5. Usually develops hours after the placement of the catheter
6. The incidence of a vessel puncture with the placement of the catheter is 1–10%.
7. Signs and symptoms
 - a. Sciatic pain
 - b. Persistent pain after the removal of the catheter
 - c. Severe back pain, sometimes radiating to the legs
 - d. Leg weakness
 - e. Paraplegia
 - f. Numbness
 - g. Significant delay in normal recovery, or deterioration of lower limb or bladder function should signal the need for emergency imaging.⁶

64. Complications of Regional Anesthesia

8. Increased risk
 - a. Scoliotic patients
 - b. Elderly with a narrow spinal canal
 - c. Coagulation defect



KO TREATMENT PLAN

Pre-operative Diagnosis

An epidural hematoma is a serious condition that requires immediate diagnosis and treatment to try to prevent long-term neurological complications. Any complaints of back pain after a spinal or epidural should be taken seriously. If an epidural hematoma is suspected, after a quick history and physical, neurosurgery should be consulted and MRI/CT should be arranged.

Pre-operative Evaluation to Determine a Regional Anesthetic Candidate

1. An epidural/spinal is approved for patients taking non-steroidal anti-inflammatory drugs.
2. An epidural/spinal is also approved for patients on subcutaneous unfractionated heparin for deep vein thrombosis (DVT) prophylaxis.
3. It is contraindicated in patients taking thienopyridine derivatives (ticlopidine and clopidogrel) and GP IIb/IIIa antagonists.
 - a. Ticlopidine should be discontinued for 2 weeks and clopidogrel for 1 week prior to the placement of an epidural/spinal.
 - b. GP IIb/IIIa should be stopped for 24 to 48 hours before an epidural/spinal.
4. For the patient receiving low-molecular-weight heparin (LMWH), a neuraxial technique should be deferred for 10–12 hours in a patient who has received a low dose, and 24 hours for a patient that has received a higher dose of LMWH. An epidural catheter should not be removed until 10–12 hours after the last dose of LMWH, and the subsequent dose should not be given for at least 2 hours after the catheter removal.

Intra-operative Treatment

1. Decompressive laminectomy within 8 hours of the symptoms

LUMBAR EPIDURAL ABSCESS^{1,5,6}

SAMPLE CASE

A 23-year-old female has an epidural placed for labor pain relief. On post-procedure day two, the patient complains of a severe headache, fever, and back pain. What should you do?

Clinical Issues

1. Rare complication
2. Usually presents 24–72 hours after the performance of an epidural block, but can take up to 4–10 days
3. Can develop spontaneously in up to 1 in 10,000 patients admitted to the hospital in the United States.
4. 30% of patients have a history of back trauma.
5. Signs and symptoms
 - a. Severe headache
 - b. Local tenderness/severe back pain
 - c. Paraspinal muscle spasm
 - d. Fever
 - e. Leukocytosis
 - f. Meningismus
 - g. Elevated erythrocyte sedimentation rate
 - h. Localized infection
 - i. Neck stiffness
 - j. Headache
 - k. Sensory and motor deficits in the legs
 - l. Bladder complications
6. Sensory findings
 - a. Absent or paresthesias only
7. Motor findings
 - a. Flaccid skeletal muscle paralysis



KO TREATMENT PLAN

Pre-operative Prevention

1. Hand washing
2. Sterile “prep and drape”
3. Limit the number of people present in the room for the procedure.
4. Face mask and hair net for everyone present for the procedure
5. Some physicians are even recommending sterile gowns.
6. Be especially careful to maintain the sterility of the epidural kit and the catheter.

Diagnosis

An epidural abscess is a serious condition that requires immediate treatment to try to prevent long-term complications. Any complaints of back pain after a spinal or epidural should be taken seriously. If an epidural abscess is suspected, after a quick history and physical, neurosurgery should be consulted and MRI/CT should be arranged.

1. MRI is the most sensitive modality.
2. CT is useful for determining the presence of extradural compression of the spinal cord.

Intra-operative Treatment

1. A decompressive laminectomy should be performed promptly to minimize the likelihood of permanent neurological deficits.
2. Continued antibiotic treatment.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2326–9.

2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 466–7, 711–4, 1163.
3. Sollmann WP, Gaab MR, Panning B. Lumbar epidural hematoma and spinal abscess following peridural anesthesia. *Reg Anaesth*, 1987 Oct;10(4):121–4.
4. Gilbert A, Owens BD, Mulroy MF. Epidural hematoma after outpatient epidural anesthesia. *Anesth Analg*, 2002;94:77–8.
5. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 577–8.
6. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 425–7, 583–4, 588–94.

65. Cardiovascular Disease in Pregnancy^{1,2}

Jessica A. Lovich-Sapola, MD

CARDIOVASCULAR DISEASE

CLINICAL ISSUES

1. The prevalence of cardiovascular (CV) disease in pregnancy is about 0.4–4.1%.
2. Maternal outcome correlates with the New York Heart Association (NYHA) criteria.
 - a. Class I or II have a <1% mortality.
 - b. Class III or IV have a 5–15% maternal mortality rate and a 20–30% perinatal mortality rate.
3. Patients with cardiovascular disease should be followed closely by a cardiologist prior to pregnancy to determine a plan. This plan may include medication modifications, valve surgery, or a plan to avoid pregnancy altogether.

NYHA Classification of Cardiovascular Disease

1. Class I: the patient is not limited by her CV disease in her physical activity. Ordinary physical activity does not precipitate the occurrence of symptoms such as fatigue, palpitations, dyspnea, or angina.
2. Class II: the patient's cardiac disease causes a slight limitation in physical activity. Patient is comfortable at rest.
3. Class III: the patient's CV disease results in a marked limitation of physical activity. She is comfortable at rest. Less than ordinary activity precipitates symptoms.
4. Class IV: cardiac disease results in the inability to carry out physical activity without discomfort. Symptoms may be present at rest. Discomfort is increased with physical activity.

LEFT-TO-RIGHT SHUNTS

CLINICAL ISSUES

Left-to-right shunts are usually well tolerated in pregnancy.

Causes

1. Small atrial septal defect (ASD)
2. Ventricular septal defect (VSD)
3. Patent ductus arteriosus (PDA)



KO TREATMENT PLAN

1. Avoid intravenous (IV) infusion of air bubbles.
2. Use loss-of-resistance with saline (rather than air) technique for the placement of an epidural.
3. An early placement of an epidural is desirable. Pain can cause increased maternal catecholamines and increased systemic vascular resistance (SVR), which may increase the severity of the left-to-right shunt, resulting in pulmonary hypertension and right ventricular failure.
4. Slow onset of the epidural anesthesia is recommended because a rapid decrease in SVR could result in shunt flow reversal and maternal hypoxemia.
5. Supplemental oxygen should be given. Even mild hypoxemia can reverse the shunt flow.
6. Avoid hypercarbia and acidosis.

COARCTATION OF THE AORTA

CLINICAL ISSUES

1. Congenital lesion
2. Fixed obstruction to the forward ejection of the left ventricular stroke volume
3. An increase in cardiac output can be achieved by increasing the heart rate.
4. If the condition has been successfully surgically treated and she has normal arm and leg pressures, she does not need any special treatment or monitoring during pregnancy.
5. Patients with uncorrected coarctation are at a high risk for left ventricular failure, aortic rupture or dissection, and endocarditis.
6. Fetal mortality in uncorrected patients may approach 20%.



KO TREATMENT PLAN

1. Maintain a normal to slightly elevated SVR, heart rate (HR), and intravascular volume status
2. In uncorrected patients avoid neuraxial anesthesia. Systemic medications and inhalation anesthesia can be used, as well as a pudendal block.
3. General anesthesia is preferred for a cesarean section.
4. Invasive hemodynamic monitoring (arterial line, central venous (CVP)) can help guide the administration of IV fluids.
5. The vasopressors of choice are dopamine and ephedrine.

TETROLOGY OF FALLOT

CLINICAL ISSUES

1. The lesion has four components.
 - a. VSD
 - b. Right ventricular (RV) hypertrophy
 - c. Pulmonic stenosis with RV outflow tract obstruction
 - d. Overriding aorta
2. Right-to-left shunt
3. Patients usually present with cyanosis.
4. Patients usually have had corrective surgery by childbearing age.
5. Even if the patient has been asymptomatic, an echocardiogram should be done before or during early pregnancy.
6. The increased blood volume, increased cardiac output (CO), and decreased SVR of pregnancy may unmask any previously asymptomatic complications from the earlier repair.



KO TREATMENT PLAN

1. 12-lead EKG
2. Avoid a decrease in SVR, which would increase right-to-left shunt
3. Maintain adequate intravascular volume and venous return.

4. Perform a neuraxial block early in labor.
5. Cesarean delivery should be done with a slowly titrated regional anesthetic. A single dose spinal is not recommended secondary to the abrupt decrease in SVR, which can lead to shunt reversal and hypoxemia.
6. General anesthesia is also an option for a cesarean section. An arterial line should be placed to determine the PaO₂ and discover early arterial hypoxemia, which can occur with increased right-to-left shunting. Pulse oximetry can also be used to reflect the changes in arterial oxygenation.

EISENMENGER SYNDROME

CLINICAL ISSUES

1. A chronic and uncorrected left-to-right shunt can produce RV hypertrophy, elevated pulmonary artery pressures, RV dysfunction, and ultimately Eisenmenger syndrome.¹
2. A reversal of the shunt flow occurs when the pulmonary artery pressure exceeds the level of the systemic pressure.
3. The primary left-to-right shunt becomes a right-to-left shunt.
4. The pulmonary vasculature does not respond to vasodilator therapy.
5. This combination of problems is not amenable to surgical correction.
6. These patients do not tolerate pregnancy well.

Clinical Manifestations When Not Pregnant

1. Arterial hypoxemia
2. RV failure
3. Dyspnea
4. Clubbing of the nails
5. Polycythemia
6. Engorged neck veins
7. Peripheral edema

During Pregnancy

1. Decreased SVR associated with pregnancy exacerbates the right-to-left shunt.
2. These individuals are unable to respond to the increased demand for oxygen during pregnancy.
3. Maternal hypoxemia leads to decreased oxygen to the fetus and a high incidence of fetal demise.
4. Maternal mortality is 30–50%.
5. Maternal deaths can occur as late as 4–6 weeks postpartum.



KO TREATMENT PLAN

1. The patient should initially be counseled to terminate the pregnancy.
2. Maintain SVR.

65. Cardiovascular Disease in Pregnancy

3. Maintain intravascular volume and venous return.
4. Avoid aortocaval compression.
5. Prevent pain, hypoxemia, hypercarbia, and acidosis which can lead to an increase in pulmonary vascular resistance.
6. Avoid myocardial depression during general anesthesia.
7. Avoid the infusion of air through the tubing secondary to the increased possibility of paradoxical air embolus.

Labor

1. Give supplemental oxygen at all times.
2. Continuous pulse oximeter to look for changes in shunt flow
3. Arterial line
4. CVP monitor and a pulmonary artery catheter (PAC) monitoring are recommended by some, but you must weigh the risks of complications including
 - a. Air emboli
 - b. Infection
 - c. Hematomas
 - d. Pneumothorax
5. Also, the PAC rarely yields useful clinical information secondary to the presence of severe, fixed, pulmonary hypertension.
6. Pain control
 - a. First stage: intrathecal opioids
 - b. Second stage:
 - i. An epidural or intrathecal dose of a local anesthetic and an opioid. Titrate slowly to avoid decreases in SVR. Avoid epinephrine in the epidural solution.
 - ii. If the patient has coagulation defects, then an intravenous infusion of remifentanyl may be the next best option.

Cesarean Section

1. Epidural anesthesia is the technique of choice.
2. The important thing is to employ incremental dosing while carefully correcting any hemodynamic changes.
3. Avoid aortocaval compression.
4. Maintain SVR, preload, and oxygen saturation.
5. General anesthesia may be required.
 - a. Positive-pressure ventilation results in decreased venous return and compromised CO.
 - b. Volatile anesthetics can cause myocardial depression and decreased SVR.
 - c. Propofol and thiopental boluses during a rapid sequence intubation (RSI) can cause a decrease in contractility and SVR and therefore an exacerbation of the right-to-left shunt. RSI is usually avoided.
 - d. Ketamine is often listed as the induction drug of choice.
 - e. Conversely, a slow induction predisposes the patient to aspiration.
 - f. Replace any large blood loss with crystalloid or the appropriate blood products.

PRIMARY PULMONARY HYPERTENSION (PPH)

CLINICAL ISSUES

1. Markedly elevated pulmonary artery pressure in the absence of an intra-cardiac or aortopulmonary shunt.
2. Reactive pulmonary vasculature that can respond to vasodilator therapy.
3. Maternal mortality rate may be as high as 30–50%. Most deaths are due to congestive heart failure and occur in the early postpartum period.
4. High incidence of fetal loss and preterm delivery



KO TREATMENT PLAN

1. Prevent pain, hypoxia, acidosis, and hypercarbia because they can cause an increase in pulmonary vascular resistance (PVR).
2. Maintain intravascular volume and venous return.
3. Maintain adequate SVR.
4. Avoid myocardial depression during general anesthesia.
5. Routine supplemental oxygen
6. Arterial line
7. CVP
8. PAC when the patient's pulmonary vasculature is responsive to vasodilators.
9. Transesophageal echocardiography can be used during a cesarean section under general anesthesia.
10. Medical treatment includes
 - a. Inhaled nitric oxide
 - b. Nitroglycerine
 - c. Calcium channel blocking agents
 - d. Prostaglandins
 - e. Endothelin antagonists

Labor

1. An early placement of a continuous epidural is recommended.
 - a. A slow induction of anesthesia with the epidural is important.
 - b. Treat the associated hypotension with IV fluids.
 - c. Vasopressors such as ephedrine should be used with caution because they can further increase the pulmonary artery pressure.
2. Avoid single dose spinal.

Cesarean Section

1. Avoid single dose spinal.
2. An epidural or a spinal catheter has been used successfully.
3. General anesthesia is often recommended, but it has associated risks.

65. Cardiovascular Disease in Pregnancy

- a. Increased pulmonary artery pressure during direct laryngoscopy and intubation.
 - b. Positive pressure ventilation has adverse effects on the venous return.
 - c. The volatile anesthetics have a negative inotropic effect.
4. Use a narcotic-based induction and maintenance anesthetic.
 5. The patient requires at least one week of continued monitoring post-delivery secondary to the high incidence of sudden death during this period.

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

CLINICAL ISSUES

1. Left ventricular hypertrophy
2. Decreased left ventricular chamber size
3. Left ventricular dysfunction
4. Sudden death is the major cause of mortality, secondary to ventricular arrhythmias.
5. Some patients tolerate pregnancy well, secondary to the increased blood volume of pregnancy.
6. Pregnancy-induced increase in heart rate, myocardial contractility, and decreased SVR may worsen the outflow obstruction.



KO TREATMENT PLAN

1. Women who are symptomatic or have a history of syncope should have a pacemaker or automatic implantable cardioverter defibrillator (AICD) placed before conception.
2. Slow the heart rate with a β -blocker throughout the pregnancy and delivery.
3. Modest expansion of intravascular volume to ensure adequate ventricular filling
4. Avoid increases in myocardial contractility.
5. Avoid decreases in SVR, since it tends to worsen the degree of outflow obstruction.
6. Avoid aortocaval compression.

Labor

1. Most tolerate labor well.
2. Give oxytocin slowly. Avoid boluses.
3. Methylergoline may be a better alternative to oxytocin.
4. A combined spinal-epidural using intrathecal opioids is a good choice for pain relief.
5. Treat hypotension with phenylephrine.

Elective Cesarean Section

1. Epidural anesthesia
2. A single dose spinal is contraindicated since the rapid onset of a sympathectomy is hazardous.

3. These patients generally tolerate general anesthesia well, if needed.
 - a. Volatile anesthetics decrease myocardial contractility, which is advantageous.

AORTIC STENOSIS (AS)

CLINICAL ISSUES

1. The lesion does not become symptomatic until the valve diameter is one-third of its normal size. (Normal aortic valve area is 2.6 to 3.5 cm².)
2. The onset of angina, dyspnea, or syncope is ominous and signals a life expectancy of less than 5 years.
3. Associated with a coarse systolic murmur that radiates to the neck
4. The diagnosis should be made with echocardiography and should be used to evaluate the lesion throughout the pregnancy.
5. Women with mild AS usually tolerate pregnancy well due to the associated increase in blood volume.
6. Women with severe AS should have corrective surgery before conception.



KO TREATMENT PLAN

1. Maintain a normal heart rate and a sinus rhythm.
2. Maintain an adequate SVR.
3. Maintain the intravascular volume and venous return.
4. Avoid aortocaval compression.
5. Avoid myocardial depression during general anesthesia.

Labor

1. Place an arterial line early in labor.
2. Many anesthesiologists like to place a PAC, but others are concerned with the increased risk of ventricular arrhythmias and cardiovascular collapse. It is usually adequate to monitor the CVP only. Maintain the CVP and/or pulmonary capillary wedge pressure at high-normal levels.
3. A continuous spinal or epidural can be safely done. Titrate the anesthetic level slowly. Remove epinephrine from the epidural/spinal solution to avoid the risk of an inadvertent intravascular injection and the precipitated tachycardia.

Cesarean Section

1. Moderate to severe AS is a relative contraindication to a single dose spinal.
2. A continuous spinal or epidural can be safely done.
3. General anesthesia can be safely established using etomidate and an opioid for induction. Thiopental is usually avoided secondary to its ability to cause myocardial depression, and ketamine is avoided because of its associated tachycardia.

MITRAL STENOSIS (MS)

SAMPLE CASE

You are called to evaluate a 22-year-old female for the placement of a labor epidural. She has a recent diagnosis of mitral stenosis. Should a labor epidural be used in a patient with mitral stenosis? What are the risks? What are the benefits? How do you minimize cardiac decompensation if a cesarean section is required? Would you use general or regional anesthesia for a cesarean section?

CLINICAL ISSUES

1. Develops when the mitral valve surface area is less than 2 cm².
2. Normal valve area is 4–6 cm².
3. Less than 1 cm² is considered severe and requires surgical intervention.
4. MS prevents the filling of the left ventricle, which results in a decreased stroke volume and cardiac output.

Diagnosis

1. 25% of women are first diagnosed during pregnancy.
2. Symptoms include dyspnea, hemoptysis, chest pain, right heart failure, and thromboembolism.
3. Diastolic murmur with auscultation
4. An EKG may show left atrial enlargement, paroxysmal atrial tachycardia, or atrial fibrillation.
5. Diagnosed by echocardiography
6. Increased incidence of pulmonary edema

Pregnancy

1. Women with severe MS do not tolerate pregnancy.
2. They are at an increased risk for pulmonary embolism.
3. The risk of maternal death is greatest during labor and in the postpartum period.



KO TREATMENT PLAN

1. β -blocker to prevent tachycardia.
2. Aggressive treatment of atrial fibrillation with digoxin and β -blockers or cardioversion.
3. Significant MS should be treated surgically prior to pregnancy. This can be done with a mitral commissurotomy or a bioprosthetic valve replacement.
4. If symptoms become severe during the pregnancy, a mitral commissurotomy should be performed in the second trimester. A balloon valvuloplasty can also be done during pregnancy if the patient's valve is pliable enough.

Labor and Cesarean Section

1. Patients with symptomatic MS require hemodynamic monitoring.
2. Plan for a low forceps delivery or cesarean section if the patient has obstetrical conditions that require it.
3. Give supplemental oxygen for labor and delivery.
4. Left uterine displacement
5. Place a pulmonary artery catheter before the induction of labor or in early labor.
6. Restrict fluids and maintain a pulmonary artery wedge pressure of approximately 14 mm Hg.
7. Prevent tachycardia. Maintain a sinus rhythm and treat atrial fibrillation aggressively.
8. Maintain the SVR.
9. Prevent pain by placing an epidural and maintaining it until the immediate postpartum period to reduce preload and prevent postpartum pulmonary edema. A combined spinal-epidural can also be placed with an intrathecal dose of opioid given initially and a continuous infusion of a dilute solution of local anesthetic as a slow infusion for the later stages of labor.
10. Prevent hypoxia, hypercarbia, and acidosis.
11. Phenylephrine is the preferred vasopressor.
12. An epidural is recommended for a cesarean section.
 - a. IV fluid administration
 - b. Slow induction of anesthesia
 - c. Small boluses of phenylephrine
 - d. Avoid tachycardia by avoiding atropine, ketamine, meperidine, and pancuronium.
 - e. A β -blocker and a dose of an opioid should be given pre-operatively.

TRANSPLANTED HEART

CLINICAL ISSUES

1. The transplanted heart has no afferent or efferent autonomic or somatic innervation.
2. Lack of vagal innervation causes the baseline heart rate to be 100–120 beats per minute.
3. Reflex slowing of the heart rate does not occur. Atropine and neostigmine have no cardiac effect.
4. Isoproterenol can be used to produce chronotropic or ionotropic effects.



KO TREATMENT PLAN

1. Pre-anesthetic evaluation
 - a. Exercise tolerance
 - b. Cardiac catheterization reports
 - c. Echocardiograms
2. Stress dose of steroids
3. Strict aseptic technique

4. Avoid aortocaval compression.
5. Maintain adequate intravascular volume and venous return.
6. Slow induction of an epidural anesthetic can be done using a dilute solution of local anesthetic and opioid.
7. Treat the associated hypotension with phenylephrine or isoproterenol.
8. A CVP is rarely placed secondary to the risk of catheter-related sepsis.
9. An epidural is the preferred technique for a cesarean section.
 - a. Give additional crystalloid to maintain adequate volume.
 - b. Both a spinal and general anesthesia have been used successfully, though.
 - c. Use ketamine instead of thiopental for induction of general anesthesia.

CARDIOPULMONARY RESUSCITATION DURING PREGNANCY

CLINICAL ISSUES

Causes of Cardiac Arrest

1. Amniotic fluid embolus
2. Pulmonary embolus
3. Arrhythmia
4. Hemorrhage
5. Myocardial infarction
6. Congestive heart failure
7. Intracranial hemorrhage
8. Local anesthetic toxicity
9. High spinal anesthesia
10. Hypermagnesemia
11. Hypoxemia



KO TREATMENT PLAN

1. Begin cardiopulmonary resuscitation (CPR).
2. Intubate the patient.
3. Prior to 24 weeks gestation, the only concern should be saving the mother.
4. After 24 weeks gestation, the team must consider both lives.
5. Maintain left uterine displacement.
6. Place the patient on a hard surface for more efficient cardiac compression.
7. Use all standard resuscitative measures, medications, and procedures without modification.

8. If the initial resuscitative efforts are unsuccessful in a pregnant woman greater than 24 weeks, deliver the fetus. This should be done within the first 4–5 minutes of the resuscitation. This may save the fetus and facilitate resuscitation of the mother. Evacuation of the uterus allows for relief of aortocaval compression and restoration of venous return to the heart.
9. A cesarean section may be necessary even if the fetus is not viable.
10. If delivery of the fetus does not facilitate successful resuscitation, consider thoracotomy, open-chest cardiac massage, and cardiopulmonary bypass (CPB).
11. CPB is especially helpful in patients who have bupivacaine-induced toxicity, pulmonary embolus, or require rewarming.

PERIPARTUM CARDIOMYOPATHY

CLINICAL ISSUES

1. Rare form of heart failure
2. Onset during the last month of pregnancy, or in the first 5 months postpartum
3. Presents with symptoms of a mild upper respiratory infection, chest congestion, and fatigue
4. Progresses to cardiac failure and low cardiac output
5. 20% chance of relapse with a subsequent pregnancy

Risks

1. Multiple gestation
2. Pre-eclampsia
3. Obesity
4. Advanced maternal age
5. Women who breast-feed



KO TREATMENT PLAN

1. Provide supportive treatment with diuretics and digitalis.
2. Prompt vaginal delivery or cesarean section
3. Continuous labor epidural is recommended for labor and vaginal delivery.
4. Perform general anesthesia with remifentanyl and propofol for maintenance, or a slow induction of epidural anesthesia guided by pulmonary artery measurements.

BIBLIOGRAPHY

1. Chestnut DH. *Obstetric Anesthesia: Principles and Practice*, 3rd ed. Philadelphia: Mosby, 2004, pp 707–28.
2. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 664–8.

66. The Basics of Pediatrics^{1,2,3,4,5}

Kasia Petelenz Rubin, MD and Kanwaljit Rani Sidhu, MD

CLINICAL ISSUES

Airway

1. Anatomic differences
 - a. Larger occiput. Neck flexion is not required to attain the “sniffing” position.
 - b. Hypertrophied tonsil and adenoid tissue
 - c. A more cephalad larynx
 - i. C2–3 in the premature infant
 - ii. C3–4 in the term infant
 - iii. C4–5 in the adult
 - d. A child has a narrower and shorter epiglottis, angled into the lumen of the airway, making the epiglottis more difficult to displace anteriorly with direct laryngoscopy.
 - e. The tongue is relatively larger in proportion to the oral cavity.
 - f. The cricoid cartilage is the narrowest part of the upper airway (compared to the vocal cords in adults).
 - g. Infants are obligate nasal breathers through 6 months of age

TKO: Despite these characteristics, unless syndromic facial anomalies exist, it is EXTREMELY RARE that tracheal intubation cannot be accomplished in the prepubertal child.

2. Endotracheal tubes (ETT)
 - a. A variety of formulas exist for estimating the proper size ETT.
 - i. The most common to use with an uncuffed oral endotracheal tube is internal tube diameter (mm) = $(16 + \text{age (yrs)})/4$
 - b. Historically, uncuffed tubes have been used in children under the age of 8.
 - i. Controversy still exists regarding the use of cuffed tubes in children, although trends are changing to the use of cuffed tubes in younger children.
 - ii. Be prepared to defend a choice of cuffed versus uncuffed tubes if you intend to address this topic during your examination. These arguments are outside the scope of this review book.

Physiology

1. Pulmonary system
 - a. Infants are prone to peri-operative hypoxemia.
 - i. Increased oxygen consumption

TKO: Desaturation occurs quickly in infants secondary to their increased oxygen consumption coupled with a relatively decreased FRC, leading to hypoxia and bradycardia.

- ii. High closing volumes
 - (1) Lung volumes at which the alveoli close
- iii. High MV/ FRC ratio
 - (1) This leads to a rapid uptake of volatile anesthetics.
 - (a) Faster inhalational induction
 - (i) Increased MV/ FRC ratio
 - (ii) Higher percentage of the neonate’s body weight consists of vessel-rich tissues.
 - (iii) Greater cardiac output per kg of body mass
- iv. Lower blood gas partition coefficient for volatile anesthetics
- v. Pliable rib cage
 - (1) The diaphragm is the primary contributor to ventilation.
 - (2) If the child has an increased oxygen demand, he or she responds by
 - (a) Increasing the respiratory rate
 - (b) Increasing respiratory excursion by diaphragmatic contraction
 - (c) This leads to negative intra-thoracic pressure and retractions.
 - (d) This is an inefficient form of ventilation with a very high energy expense, especially if the patient develops an airway obstruction.
2. Cardiovascular system
 - a. Immature
 - b. Decreased ability to increase myocardial contractility
 - i. The stroke volume (SV) is relatively fixed secondary to the pediatric noncompliant left ventricle.

TKO: The infant’s cardiac output (CO) is rate-dependent: $\text{CO} = \text{heart rate (HR)} \times \text{SV}$

- c. Causes of decreased heart rate in infants
 - i. Hypoxemia
 - ii. Vagal stimulation
3. Temperature regulation
 - a. Temperature regulation is maintained via brown fat metabolism (nonshivering thermogenesis), crying, and movement.
 - b. Infants do not shiver.

Table 66.1. Vital Signs

	Newborn	3 mos–2 yrs	2 – 10 yrs	> 10 yrs
Respirations (breaths/minute)	45–60	30	25	20
Heart Rate (beats/minute)	100–180	80–150	70 – 110	50–90
Minimum Acceptable Blood Pressure	Mean BP \geq PCA	SBP $\geq 70 + 2 \times$ age	SBP $\geq 70 + 2 \times$ age	SBP \geq approx 90

Abbreviations: PCA: Post-conception age (weeks); SBP: systolic blood pressure; age in years.

Table adapted from information in www.cchmc.org and Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*, 7th ed. Philadelphia: Mosby Elsevier, 2006, pp 798–9.

Table 66.2. Pediatric Pulmonary System

Parameter	Infant	Adult
Tidal volume (ml/kg)	7	7
Dead space (ml/kg)	2–2.5	2.2
Alveolar ventilation (ml/kg/min)	100–150	60
FRC (ml/kg)	27–30	30
Oxygen consumption (L/min)	7–9	3

Abbreviation: FRC: functional residual capacity

Table adapted from Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1181–1202.

- c.** It is difficult to maintain normothermia secondary to a disproportionately large body surface area, increased metabolic rate, and a thinner layer of subcutaneous insulating body fat.
- d.** This means that the room temperature must be kept higher, and warming blankets and fluid warmers must be used whenever possible.
- 4. Renal system**
- a.** Decreased glomerular filtration rate (GFR) and renal blood flow (RBF)
- These values increase rapidly by 3 months of age.
 - This may clinically affect the clearance of certain medications.
 - Aminoglycosides
 - Muscle relaxants
- b.** Renal tubular function is also immature at birth.
- Limited ability to concentrate urine
- 5. Fluids and electrolytes**
- a.** Maintenance fluid requirements
- 4 ml/kg/hr for 1st 10 kg, plus
 - 2 ml/kg/hr for 2nd 10 kg, plus
 - 1 ml/kg/hr for each remaining kg
- b.** Fluid deficits
- Estimated fluid deficit = estimated hourly maintenance (EHM) \times the number of hours NPO
 - Estimated blood volume (mL/kg)

- Premature: 90
- Full term: 85
- Infant: 80
- Child: 75
- Adult: 70 (male) and 65 (female)

- c. Glucose**
- Infants have increased metabolic demands and decreased glycogen stores; therefore, they are prone to hypoglycemia.
 - Intravenous dextrose (as D5 0.25%NS or D10 W) should be administered in all patients under 6 months of age, and in infants between 6 and 12 months if the surgery is anticipated to last longer than 1 hour.
 - Maintain serum glucose levels >40 mg/dL.
 - Other indications for intravenous dextrose administration include
 - History of liver disease
 - Total parenteral nutrition (TPN) administration
 - Severe systemic illness
 - Newborn infants of diabetic mothers
- d. NPO guidelines**
- Clear liquids: 2 hours
 - Breast milk: 4 hours
 - Formula: 6 hours
 - Nonhuman milk: 6 hours
 - Meal with fat: 8 hours
- e. Hemoglobin**
- The oxyhemoglobin dissociation curve is shifted to the left.
 - As the fetal hemoglobin decreases, there is a physiologic fall in the infant's hematocrit starting at 6–8 weeks and peaking at 12 weeks.

BIBLIOGRAPHY

- Litman RS, ed. *Pediatric Anesthesia: The Requisites in Anesthesiology*, 1st ed. Philadelphia: Mosby, 2004.
- McNiece WL, Dierdor SF. The pediatric airway. *Seminars in Pediatric Surgery* 2004;13(3):152–65.
- Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*, 7th ed. Philadelphia: Mosby Elsevier, 2006, pp 798–9.
- www.cchmc.org.
- Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1181–1202.

67. Neonatal Resuscitation^{1,2,3,4,5}

Kasia Petelenz Rubin, MD and Kanwaljit Rani Sidhu, MD

SAMPLE CASE

You are taking care of a 24-year-old female for a cesarean section. Her epidural has been dosed with 20 ml of 2% lidocaine plus 1:200,000 of epinephrine. She is a G1P0. She had adequate prenatal care and had been laboring without progression for the previous 20 hours. When the baby is delivered, the child makes no sound and no attempts at breathing. The nurses turn to you for help. What will you do? Can you leave the mother to attend to the baby? Who do you have the ultimate responsibility to care for? Assuming you get a colleague to help care for the mother, how would you evaluate and resuscitate the baby?

CLINICAL ISSUES

Ethical Issues

1. The ASA standards for basic anesthetic monitoring state that “qualified personnel, other than the anesthesiologist attending the mother, should be immediately available to assume responsibility for resuscitation of the newborn.”¹
2. While the primary responsibility of the anesthesiologist is to provide care for the mother, the guidelines for anesthesia care in obstetrics go on to state, “If the anesthesiologist is also requested to provide brief assistance in the care of the newborn, the benefit to the child must be compared to the risk of the mother.”¹

Transitional Physiology

1. Intrauterine fetal circulation
 - a. General information
 - i. Fetal shunts in utero
 - (1) Ductus venosus
 - (2) Foramen ovale
 - (3) Ductus arteriosus
 - ii. Pulmonary vascular resistance (PVR) is high.
 - iii. Systemic vascular resistance (SVR) is low.
 - b. Pathway of fetal circulation
 - i. Blood from the placenta travels to the baby through the umbilical vein.
 - ii. The umbilical vein goes to the liver and splits.
 - (1) About half of the blood bypasses the liver through the ductus venosus and goes directly to the vena cava, then to the heart.

(2) The other half of the blood goes directly to the liver.

iii. Once in the heart, the blood enters the right atrium.

(1) Most blood then flows through the foramen ovale to the left atrium.

(a) Blood then passes to the left ventricle and then to the aorta.

(b) Blood from the aorta travels to the head and upper extremities.

(2) The blood that does not enter the foramen ovale stays in the right heart and travels to the pulmonary artery.

(a) Since the placenta does the work of oxygen exchange, the lungs are not used for this purpose in the fetus.

(b) Most of the blood on its way to the lungs from the pulmonary artery is bypassed away from the lungs to the aorta through the ductus arteriosus.

iv. The blood then travels from the aorta to the umbilical arteries, and back to the placenta.

2. Birth

a. Arrest of the umbilical circulation removes the low resistance placental bed from circulation.

b. Expansion of the lungs occurs as breathing is initiated within the first 30 seconds of life.

i. By 90 seconds, sustained respirations are present.

ii. The inflation of the lungs reduces the resistance to blood flow through the lungs resulting in increased blood flow from the pulmonary arteries; decreased PVR.

(1) PVR may remain elevated, resulting in a persistent fetal circulation.

(2) Causes of elevated PVR

(a) Hypoxemia

(b) Hypercarbia

(c) Hypothermia

(d) Hypovolemia

(e) Acidosis

Risk Factors for Fetal Distress and the Associated Need for Fetal Resuscitation

1. Maternal risk factors

- a. Diabetes

67. Neonatal Resuscitation

- b. Pregnancy-induced hypertension
 - c. Previous stillbirth
 - d. Infection
 - e. Substance abuse
 - f. Cesarean section delivery
 - g. General anesthesia for delivery
 - h. Narcotic or other substance use
 - i. Chronic hypertension
 - j. Previous Rh sensitization
 - k. Bleeding in the second or third trimester
 - l. Maternal infection
2. Fetal risk factors
- a. Post-term or pre-term gestation
 - b. Multiple gestation
 - c. Poly- or oligohydramnios
 - d. Known fetal anomalies
 - e. Abnormal fetal lie
 - f. Non-reassuring fetal heart rate patterns
 - g. Meconium-stained amniotic fluid

Assessment of the Fetus during Labor

1. The fetal heart rate is the most reliable predictor of fetal well-being.
- a. It is $\geq 90\%$ accurate in predicting a 5 minute APGAR score greater than 7.
 - b. False positive rate of 35–50% in predicting fetal compromise
2. Fetal scalp pH can be used to confirm or exclude fetal acidosis.
- a. A pH of ≤ 7.20 is considered abnormal and indicates the need for immediate delivery.

Assessment of the Fetus after Delivery

1. APGAR Score
- a. APGAR scoring is a useful method to evaluate the clinical status of the patient at 1 and 5 minutes after delivery.
 - b. The scoring system is presented below.
 - c. Do not wait for the 1 minute APGAR score to begin resuscitation, if it is necessary.

- d. If the 5 minute APGAR score is less than 7, additional scores should be obtained every 5 minutes until 20 minutes have passed or 2 successive scores are more than 7.
- e. The current guidelines state that if more than 10 minutes of continuous and adequate resuscitative efforts produce no signs of life, then resuscitation should be discontinued.



KO TREATMENT PLAN

Preparation for Resuscitation

1. Equipment
- a. Suction
 - i. Bulb syringe
 - ii. Mechanical suction
 - iii. Suction catheters
 - iv. Meconium aspirator
 - b. Intubation equipment
 - i. Laryngoscope and blades (Miller #0 and #1)
 - ii. Endotracheal tubes (2.5–4.0 mm)
 - iii. Stylet
 - c. Bag and mask equipment
 - i. Neonatal resuscitation bag with a pressure valve
 - ii. Face masks in the appropriate sizes
 - iii. Oral airways
 - iv. Oxygen with a flowmeter
 - d. Extras
 - i. Radiant warmer
 - ii. Stethoscope

Initial Treatment of All Infants

1. Warm and dry the infant.
2. Aspirate the mouth, pharynx, and nose with a catheter.
3. Stimulate the infant by slapping the soles of the feet or rubbing the back.
4. Verify vital signs as per the above algorithm.

Table 67.1. APGAR Scoring

Sign	0	1	2
Appearance (skin color)	Pale, blue	Extremities blue	Completely pink
Pulse (heart rate)	Absent	<100 beats/minute	>100 beats/minute
Grimace (reflex irritability)	No response	Grimace	Cough, sneeze, cry
Activity (muscle tone)	Limp	Extremity flexion	Active motion
Respiratory effort	Absent	Slow, irregular	Good, crying

Adapted from Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1164–7, 1174–5.

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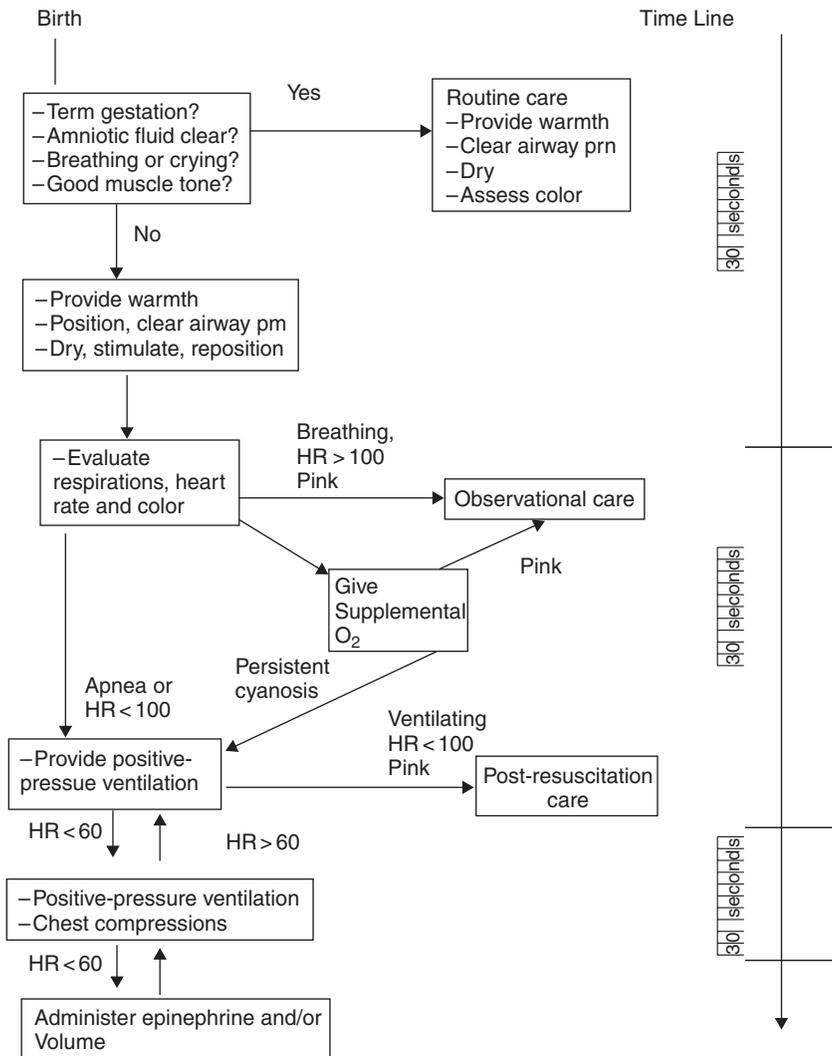


Figure 67.1. Neonatal resuscitation algorithm. Source: Reprinted with permission from Neonatal Resuscitation. (VA Arkoosh) Refresher Course Lectures. 2006. No. 137 of the American Society of Anesthesiologists. A copy of the full text can be obtained from ASA, 520 N. Northwest Highway, Park Ridge, Illinois 60068-2573.

Treatment of a Depressed Infant

1. If oropharyngeal suction reveals meconium or thick meconium-stained mucus, suction via an endotracheal tube before lungs are inflated, within 1-2 minutes of delivery.
2. If after stimulation, the infant is still apneic or has a heart rate less than 100, initiate positive pressure ventilation at rate of 30-40 breaths/minute.
3. If after 30 seconds of ventilation, the heart rate is still below 100, begin chest compressions at a rate of 100 compressions per minute.

Neonatal Resuscitation Medications

1. Sodium bicarbonate: 4.2% (0.5 mEq/mL)
 - a. Sodium bicarbonate use is not recommended during brief cardiopulmonary resuscitation.
 - b. If resuscitation is prolonged, in the face of a documented acidosis, with adequate ventilation and perfusion, 4.2% sodium bicarbonate may be infused to a total dose of 2 mEq/kg, via an umbilical catheter.

2. Naloxone: (0.4 mg/mL)
 - a. A dose of 0.01 mg/kg may be injected intravenously (IV), intramuscularly (IM), subcutaneously (SQ), or via the endotracheal tube (ETT) once adequate ventilation is achieved in the setting of respiratory depression secondary to maternal opioid administration.
 - b. Do not administer to infants of opioid addicted mothers for fear of precipitating withdrawal.
3. Epinephrine: (1:10,000 concentration)
 - a. Doses of 0.01-0.03 mg/kg may be injected IV or 0.1 mg/kg via the ETT for the treatment of asystole or persistent bradycardia despite 30 seconds of effective ventilation and chest compressions. This dose can be repeated every 3-5 minutes as needed.
4. Atropine
 - a. A dose of 0.02 mg/kg may be given IV or 0.03 mg/kg via the ETT to treat bradycardia.
5. Calcium gluconate
 - a. A dose of 100 mg/kg may be infused over 5-10 minutes to treat low cardiac output.

- b. The neonate should be on continuous EKG monitoring.
- 6. Fluids: volume expanders, normal saline, dextrose 10%, and O-negative blood
 - a. Acute volume expansion may be achieved with
 - (1) O-negative blood or maternally cross-matched blood (10 mL/kg)
 - (2) Normal saline or lactated ringers (10 mL/kg)
 - b. Albumin administration is associated with increased mortality; avoid use.
- 7. Severe acidosis (<7.0 pH) may decrease the effectiveness of the aforementioned medications.
- 8. Medications should be given with the smallest volume of fluid possible to decrease the risk of hypervolemia.

BIBLIOGRAPHY

1. Standards for Basic Anesthetic Monitoring (Approved by ASA House of Delegates 10/21/86 and last amended 10/27/04). www.asahq.org.
2. Arkoosh, VA. Neonatal Resuscitation. Annual Meeting Refresher Course Lectures, ASA Annual Meeting 2006.
3. American Heart Association AaP: 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care of Pediatric and Neonatal Patient: Neonatal Resuscitation Guidelines. *Pediatrics* 2006;117:e1029-38.
4. Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1164-7, 1174-5.
5. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2360-1.

68. Tracheoesophageal Fistula^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 27-year-old female presents with an uncomplicated delivery of a male neonate at 37 weeks estimated gestational age. In the delivery room, the nurses observe that the baby is drooling excessively and appears to be coughing and choking. Upon initiating breast-feeding, the infant becomes cyanotic. Feeding is discontinued. A nasogastric catheter is passed, and a chest X-ray reveals a distal tracheoesophageal fistula. What are your concerns? How will you induce anesthesia? How would you intubate this patient? When would you extubate?

CLINICAL ISSUES

Epidemiology

1. Incidence is approximately 1 in 4,000 live births.
2. Associated congenital anomalies are seen in approximately 50% of the cases.
 - a. Other gastrointestinal malformation
 - b. VACTERL association
 - i. Vertebral/vascular
 - ii. Anorectal
 - iii. Cardiac: ventricular septal defect (VSD), atrial septal defect (ASD), and atrio-ventricular canal defects
 - iv. Tracheo-esophageal
 - v. Radial/renal
 - vi. Limb deformities
 - c. Generalized chromosomal syndrome
3. Prematurity: 30% incidence

Diagnosis

1. Prenatal: polyhydramnios, prominent esophageal pouch, small or absent stomach “bubble” with fluid-filled loops of bowel
2. The 3 C’s associated with TEF: choking, coughing, and cyanosis
3. Neonatal
 - a. Excessive salivation
 - b. Regurgitation
 - c. Coughing
 - d. Choking with initial feeds
 - e. Respiratory distress
 - f. Cyanosis
 - g. Inability to advance a catheter into the stomach
4. Test results on this child
 - a. Chest X-ray reveals the tip of the catheter in the superior mediastinum.
 - b. Gas in the stomach and intestine indicates the presence of a distal fistula.
 - c. Water-soluble contrast or barium can illustrate the proximal pouch.



KO TREATMENT PLAN

TKO: Please note that the majority of these cases are performed at regional pediatric surgical centers. The outline

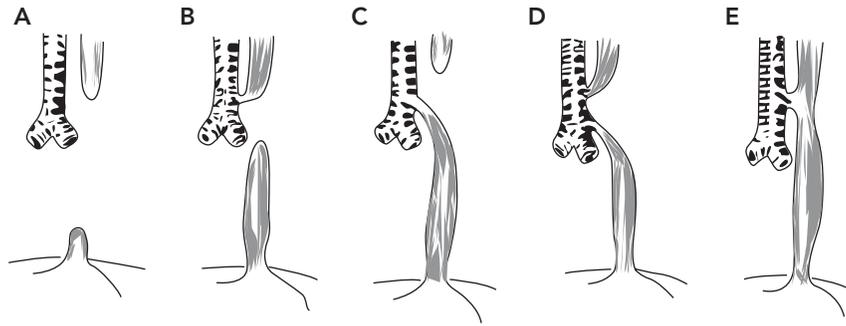


Figure 68.1. Examples of tracheoesophageal fistula.

Source: Reprinted from *Otolaryngologic Clinics of North America*, Volume 40, Issue 1, Olga Achildi and Harsh Grewal, Congenital Anomalies of the Esophagus, Pages 219–44, 2007, with permission from Elsevier.

Type A: Esophageal atresia (EA) without tracheoesophageal fistula (TEF); Type B: EA with proximal TEF; Type C: EA with distal TEF (85% of cases); Type D: EA with TEF between both esophageal segments and trachea; Type E: TEF without EA or H-type fistula.

below illustrates basic management points with which every anesthesiologist should be familiar. These topics are more likely to be addressed in the short questions rather than a long stem, due to the advanced nature of the procedure. Early or delayed surgical intervention is indicated depending on the condition of the infant and the need to prevent further aspiration of gastric secretions or contents.

Pre-operative

1. A complete pre-operative evaluation and treatment of the patient should include the following:
 - a. Evaluate respiratory complications secondary to infection or intrinsic lung disease associated with prematurity.
 - b. Verify the patient's hydration status.
2. Approximately 50% of the patients who have EA or TEF present with additional birth defects; therefore, a complete evaluation should include
 - a. Radiographs of the chest, abdomen, pelvis, and spine
 - b. Ultrasound of the spine and kidney
 - c. Echocardiography of the heart and aorta
3. Vascular access should be obtained.
4. Verify that a type and crossmatch are available.
5. The goal of pre-operative anesthetic management is prevention of aspiration of gastric contents by
 - a. Avoidance of feeding
 - b. Elevation of the head of the bed
 - c. Placement of a suction catheter in the proximal pouch

Intra-operative

1. Awake intubation versus inhalational, with or without muscle relaxation
 - a. Awake intubation with spontaneous ventilation is thought to preserve the airway protective reflexes, addressing the concerns of undiagnosed airway abnormalities,

and the risk of aspiration. This theoretically avoids the potential for arterial desaturation after induction.

- i. However, awake intubation has been found to confer no benefits in preserving arterial oxygenation or adequate heart rate and has largely been abandoned in favor of anesthetized paralyzed tracheal intubation after a rapid-sequence or modified rapid-sequence intravenous induction.

- b. In a modified rapid-sequence intravenous induction, positive pressure ventilation is avoided in an attempt to prevent gastric distension secondary to the gases passing through the fistula into the stomach.

- i. Cautious, gentle positive-pressure ventilation may be necessary to prevent desaturation.

2. Auscultate the stomach and chest to ensure that the lungs are adequately ventilated and that the stomach is not distended.

- a. Typically, the endotracheal tube is placed in the main-stem bronchus, and slowly withdrawn until breath sounds are bilateral.

- b. Remember that the infant is very small, and if breathing spontaneously, the endotracheal tube could be in the esophagus and bilateral breath sounds may still be auscultated.

3. **Other issues that may arise leading to desaturation or difficult ventilation.**

- a. Kinking or obstruction of the small endotracheal tube
- b. Migration of the oral endotracheal tube into the fistula
- c. Low lung compliance and high compliance of the fistula

Post-operative

1. Usually the esophageal anastomosis is under tension and the infant is electively paralyzed and mechanically ventilated for up to 5 post-operative days.
2. Ventilation with a bag/mask should be avoided for several post-operative days, secondary to the fragility of the anastomosis.

3. Long-term complications include
 - a. Dysphagia
 - b. Gastroesophageal reflux disease (GERD)
 - c. Respiratory infections
 - d. Choking
 - e. Esophageal strictures
 - f. Symptomatic tracheomalacia
 - g. Recurrent TEF
 - h. Wheezing or bronchial hyperreactivity
 - i. Chest wall deformities

BIBLIOGRAPHY

1. Achildi O, Grewal H. Congenital anomalies of the esophagus. *Otolaryngol Clin N Am* 2007;40:219-44.
2. Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*, 7th ed. Philadelphia: Mosby Elsevier, 2006, pp 550-2.
3. Spitz L. Oesophageal atresia. *Ophanet J Rare Dis*, 2007;2:24-37.
4. Cook-Sather SD, Tulloch H, Cnaan A, Nicolson SC, Cubina ML, Gallagher PR, Schreiner MS. A comparison of awake versus paralyzed tracheal intubation for infants with pyloric stenosis. *Anesth Analg*, 1998;86:945-51.

69. Pyloric Stenosis^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 3-week-old male infant, born 2 weeks premature, now weighing 4 kg, is scheduled for a pyloromyotomy. He has a history of nonbilious emesis for the past 5 days. He is limp and lethargic. His current vital signs are heart rate (HR) 168, respiratory rate (RR) 54, and blood pressure (BP) 72/35 mm Hg. His recent laboratory findings include an unremarkable complete blood count (CBC), Na⁺ 130, K⁺ 2.5, and a Cl⁻ 85. What are your concerns? Is this an emergency surgery? Should his electrolytes be corrected? What should they be corrected to? How would you induce this patient?

CLINICAL ISSUES

Electrolyte Abnormalities:

Chronic emesis leads to the loss of hydrochloric acid (HCl) from the stomach, which causes a hypochloremic, hypokalemic metabolic alkalosis.

1. Hydrogen and chloride ions are lost from the stomach, which result in hypochloremic metabolic alkalosis.
2. The kidneys therefore secrete potassium in exchange for the hydrogen ions in an effort to maintain a normal arterial pH.
3. As the kidneys exchange potassium for hydrogen ions, the infants become sodium-depleted from vomiting.
4. The kidney attempts to conserve sodium to maintain volume and exchanges sodium ions for potassium and hydrogen ions, causing a paradoxical aciduria.
5. The net result from the kidneys compensating for the emesis is a loss of hydrogen and potassium, which results in a hypokalemic metabolic alkalosis.

Full Stomach Precautions

1. The stomach is often filled with bile, food contents, or barium (if a barium swallow was performed as a diagnostic procedure).
2. The infant's insitu nasogastric (NG) tube should be suctioned prior to the induction of anesthesia.
 - a. If an NG is not in place, most practitioners favor the placement of an orogastric tube (OG) or NG tube prior to the induction of anesthesia in a patient with pyloric stenosis, in order to fully decompress the stomach contents.
3. Multiple laryngoscope handles and blades should be available.
4. Several sizes of endotracheal tubes with lubricated stylets in place should be ready.

Airway Management:

1. The best airway management for these full-stomach infants has been a matter of great debate.
2. Inhalational induction techniques following evacuation of the stomach contents via suctioning have generally been replaced by intravenous inductions in order to decrease the incidence of aspiration.
3. Some historical discussion remains regarding whether induction should proceed via awake versus paralyzed tracheal intubation for infants with pyloric stenosis.
 - a. Some anesthesiologists have recommended an awake tracheal intubation to maintain the protective airway reflexes due to
 - i. Concerns about undiagnosed airway abnormalities
 - ii. Increased risk of aspiration
 - iii. Potential for desaturation after the anesthetic induction

69. Pyloric Stenosis

- b.** The most recent consensus, however, advocates a rapid sequence paralyzed tracheal intubation as opposed to an awake intubation.
 - i.** Avoids multiple attempts.
 - ii.** Faster than struggling with a conscious infant
 - iii.** Awake intubations have not proven to prevent bradycardia or decreased arterial hemoglobin oxygen saturation.

Post-operative Apnea

1. There are many reports of apnea events following a pyloromyotomy.
 - a.** Patients are often less than 60 weeks post-conceptual age.
 - b.** It is thought that although the alkalemia is corrected, the cerebrospinal fluid alkalosis may take longer to correct and may play a role in post-operative ventilatory depression.
 - c.** For this reason, it is also wise to avoid administration of narcotics during the operative and post-operative periods.
 - i.** Pain control can often be adequately achieved with rectal acetaminophen and the use of local anesthetic at the surgical site.



KO TREATMENT PLAN

Pre-operative

1. Adequately volume-resuscitate the patient and replete electrolytes as needed.
2. Before proceeding with surgery, the patient's electrolytes **MUST** be normal!
 - a.** Normal electrolytes include a serum potassium level in the normal range (3.5–5.5 mEq/L) and a serum chloride level greater than 90 mEq/L or a urine chloride level greater than 20 mEq/L.
 - b.** Because the kidney will retain chloride as a result of volume contraction, a urine chloride greater than 20 mEq/L suggests that volume resuscitation has been adequate.
3. This is a **MEDICAL**, never a surgical, emergency.
4. Assuming the patient is normovolemic with normal electrolytes, then proceed to the operating room.

Intra-operative

1. Suction the stomach with a wide bore catheter in the supine, right and left lateral positions to remove as much of the stomach contents as possible.
2. Assuming the infant has a normal-appearing airway, pre-treat with 20 mcg/kg of atropine and perform a rapid sequence induction with cricoid pressure using IV propofol (or another induction agent in doses guided by the patient's hemodynamic status) and succinylcholine.
3. During the maintenance phase of anesthesia, titrate a volatile anesthetic with nitrous oxide and oxygen to allow for a rapid awakening at the conclusion of the surgery.
4. Muscle relaxants are not indicated for the maintenance of anesthesia because this is a relatively short surgical procedure in which complete skeletal muscle relaxation is generally unnecessary.

Post-operative

1. Extubate the patient when fully awake and displaying regular and adequate breathing patterns.
2. For pain control, acetaminophen can be administered rectally.
3. Post-operative monitoring is vital.
 - a.** Persistence of electrolyte and fluid imbalances is possible.
 - b.** Often patients are less than 60 weeks post-conceptual age and are therefore more likely to exhibit post-operative ventilatory depression.

BIBLIOGRAPHY

1. Litman RS, ed. *Pediatric Anesthesia: The Requisites in Anesthesiology*, 1st ed. Philadelphia: Mosby, 2004, p 233.
2. Cook-Sather SD, Tulloch V, Cnaan A, Nicolson SC, Cubina ML, Gallagher PR, Schreiner MS. A comparison of awake versus paralyzed tracheal intubation for infants with pyloric stenosis. *Anesth Analg*, 1998;86:945–51.
3. Stoelting RK, Dierdorf SF, eds. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, p 700.
4. Schwartz D, Connelly NR, Manikantan P, Nichols JH. Hyperkalemia and pyloric stenosis. *Anesth Analg*, 2003;97:355–7.

70. Epiglottitis^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 4-year-old boy presents with a history of a 24 hour fever, irritability, throat pain, and facial flushing. He speaks in a low muffled voice and is drooling. He has been unable to take food or drink by mouth secondary to pain. His temperature is 39.5 °C, heart rate (HR) is 125, and his blood pressure (BP) is 85/50 mm Hg. He is brought to the operating room for intubation. What are your concerns? How will you induce anesthesia? Which inhaled anesthetic would you use and why? When would you extubate?

A child presenting with signs suspicious for upper respiratory tract obstruction and respiratory distress will often be managed immediately in the operating room under general anesthesia before diagnostic X-rays are obtained, secondary to the extreme rapidity with which respiratory distress and complete airway compromise may occur.



KO TREATMENT PLAN

Pre-operative

1. Be certain that all necessary bronchoscopes, endotracheal tubes, and emergency tracheotomy equipment are available.

2. A skilled otolaryngologist must be present and should accompany the patient at all times once the diagnosis is suspected, should the need for a surgical airway arise.

3. Disturb the child as little as possible. If parental separation would cause undue anxiety, the parents should be allowed into the operating room suite.

4. Concerns regarding a full stomach are theoretically reasonable, as the children are not “fasted.” However, these children are often so sick, and swallowing is so painful, that food and fluid intake has likely been diminished prior to presentation. Historically, aspiration has not been a significant complication in studies of these cases.

Intra-operative

1. Even if the child presents with intravascular access, it is easiest to maintain spontaneous ventilation with an inhalational induction.

2. Maintain the child in a sitting position, and administer nitrous oxide, oxygen, and sevoflurane.

a. Sevoflurane is preferred because it causes less irritation to the airways when compared to isoflurane and desflurane.

3. The child is permitted to deepen his level of anesthesia with spontaneous ventilation.

Table 70.1. Clinical Issues

	Epiglottitis
Age (years)	Usually 3–5
Infection site	Supraglottic
Etiology	Bacterial – <i>Haemophilus influenzae B</i>
Onset and course	Rapid onset, a respiratory emergency
Fever	Usually greater than 39°C
Clinical exam	Muffled “hot-potato” voice, dysphagia, and drooling. “Tripod” position.
Pharyngeal exam	“Cherry-red” epiglottitis
X-ray	“Thumbprint” sign
Treatment	Endotracheal intubation, antibiotics

Adapted from DeSoto H. Epiglottitis and croup in airway obstruction in children. *Aes Clin N Am*, 1998;16(4):853–68; Shah S, Shariieff GQ. Pediatric respiratory infections. *Emerg Med Clin N Am*, 2007;25:961–79; Alcaide ML, Bisno AL. Pharyngitis and epiglottitis. *Infect Dis Clin N Am*, 2007;21:449–69.



Figure 70.1. X-Ray demonstrating thumbprint sign.⁴ Images used with permission from personal files of Drs. Robin Cotton and Charles Myer, Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.



Figure 70.2. Laryngoscopic view of epiglottitis. Note swelling and significant erythema.⁴ Images used with permission from personal files of Drs. Robin Cotton and Charles Myer, Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.

4. Intravenous access is then obtained and standard ASA monitors are placed.

5. With the patient deep and spontaneously ventilating, the otolaryngologist will perform a direct laryngoscopy and rigid bronchoscopy. The visualization of the larynx is often difficult, and the epiglottis needs to be very gently lifted with a blade or with a rigid bronchoscope. An endotracheal tube one or two sizes smaller than normal is placed.

6. If during the induction, any problem arises leading to a loss of spontaneous ventilation or airway obstruction, an emergent direct laryngoscopy or rigid bronchoscopy needs to be attempted.

7. The surgeon must be prepared for an emergent cricothyrotomy if airway control is not quickly achieved.

Post-operative

1. The patient typically remains sedated with an endotracheal tube in place for at least 24 to 48 hours.

2. Antibiotics and corticosteroids are the mainstays of treatment.

3. Accidental extubation must be avoided.

4. Extubation is performed after the resolution of clinical signs and the confirmation of decreased epiglottic swelling by flexible nasal fiber optic bronchoscopy or direct laryngoscopy in the operating room.

BIBLIOGRAPHY

1. DeSoto H. Epiglottitis and croup in airway obstruction in children. *Aes Clin N Am*, 1998;16(4):853-68.
2. Shah S, Shariieff GQ. Pediatric respiratory infections. *Emerg Med Clin N Am*, 2007;25:961-79.
3. Alcaide ML, Bisno AL. Pharyngitis and epiglottitis. *Infect Dis Clin N Am*, 2007;21:449-69.
4. Images from personal files of Drs. Robin Cotton and Charles Myer, Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.

71. Pheochromocytoma^{1,2,3}

Donn Marciniak, MD

A 35-year-old male is admitted for resection of a pheochromocytoma and you are consulted pre-operatively for management. His pre-operative hemodynamics are well controlled on metoprolol and phenoxybenzamine. What are your induction and maintenance plans? While the surgeon is resecting the tumor, the patient's mean arterial pressure (MAP) increases from 90 mm Hg to 160 mm Hg. How will you treat this? What can you use to prevent future episodes like this?

CLINICAL ISSUES

Definition

A pheochromocytoma is a catecholamine-secreting tumor of neural crest cell origin. Most tumors secrete norepinephrine and epinephrine, with norepinephrine being the most common. They are typically found in the adrenal medullae but may develop in any area with chromaffin tissue. This can be anywhere from the skull base to the anus.

Incidence

1. Pheochromocytomas are found in 0.005 to 0.1% of the population with a peak occurrence in the third to fifth decade.
2. 10% of pheochromocytomas are extramedullary.
3. 10–20% of patients have a familial history.
4. 90% of the tumors are solitary.
5. Tumors are bilateral in 10% of adults and 25% of children.
6. 10% of tumors are malignant.
7. Pheochromocytomas account for 0.1% of all cases of diagnosed hypertension.

Differential Diagnosis

1. Essential hypertension
2. Sympathetic stimulation: hypoxia, hypercarbia, pain, light anesthesia
3. Endocrine: thyroid storm, pheochromocytoma
4. Neurogenic: seizures, carotid sinus denervation, increased intracranial pressure
5. Renal disease: renal artery stenosis, nephritis
6. Miscellaneous: malignant hyperthermia, carcinoid syndrome, (pre-)eclampsia

Associated Syndromes

1. Von Hippel-Lindau disease: hemangioblastomas in retina, cerebellum, or other parts of the central nervous system (CNS) and pheochromocytoma
2. Von Recklinghausen's neurofibromatosis: neurofibromas, café-au-lait spots, axillary freckling, optic nerve glioma, and pheochromocytoma
3. Multiple Endocrine Neoplasia (MEN) IIa: thyroid medullary carcinoma, pheochromocytoma, and parathyroid hyperplasia/adenoma
4. MEN IIb: thyroid medullary carcinoma, pheochromocytoma, mucosal neuromas, and marfanoid appearance

Testing

1. Urine analysis for norepinephrine, epinephrine, dopamine, vanillylmandelic acid (VMA), and total metanephrines.
 - a. VMA is the metabolite of norepinephrine.
 - b. Metanephrines are the products of epinephrine.
2. Total metanephrines have the highest true-positive results at 98–99%.
3. If urine tests are negative or equivocal and a strong clinical suspicion exists, provocative testing can be performed with glucagon or suppression testing with clonidine.
 - a. A three-fold increase in norepinephrine levels within 2 minutes after the glucagon is administered indicates a pheochromocytoma with high specificity.
 - b. The clonidine suppression test is particularly useful in patients with increased plasma norepinephrine, in whom it is unclear whether the increase is due to sympathetic activation or catecholamine release from a tumor. Clonidine is administered, and a lack of a decrease in norepinephrine is highly suggestive of a pheochromocytoma.
4. Many patients are found to have died from complications of unknown pheochromocytoma at autopsy.

Clinical Features

1. Classic triad of paroxysmal sweating, hypertension, and headache is more sensitive and specific than any laboratory test evaluating for pheochromocytoma.
2. ST-T changes are often noted on EKG.
3. Catecholamine-induced cardiomyopathy occasionally manifests.

71. Pheochromocytoma

- a. Left ventricular (LV) hypertrophy can occur and progress into LV failure if the symptoms of the pheochromocytoma are not corrected.
4. Hyperglycemia reflects the beta effects of catecholamines and typically does not require insulin therapy.
5. Patients are often volume-depleted.
6. Hypomagnesemia and the associated dysrhythmias are often present.

Triggers

1. Most pheochromocytomas are not under neurogenic control.
2. Physiologic factors affecting the pheochromocytoma
 - a. Pain (this patient subset has been noted to have low pain tolerance)
 - b. Light anesthesia
 - c. Hypotension
 - d. Hypoxia
 - e. Hypercarbia
 - f. Hypoglycemia
 - g. Anger, fear, anxiety
3. Pharmacologic factors affecting the pheochromocytoma
 - a. Direct and indirect sympathomimetics
 - b. Histamine-releasing agents



KO TREATMENT PLAN

Pre-operative

1. Identify tumor location(s) with imaging.
 - a. Caution: dye used in arteriography can result in histamine release.
2. Alpha blockade is considered to be a cornerstone of therapy by most groups and has been attributed to decreasing the mortality from 45% to 0–3%.
3. Beta blockade may be considered but should not precede alpha blockade.
 - a. By blocking the beta receptors before the alpha receptors, the unopposed alpha effects may theoretically precipitate congestive heart failure.
4. No randomized, prospective trials involving alpha and beta blockade have been conducted, but they are likely to reduce the incidence of hypertensive crises and dramatic blood pressure fluctuations.
5. Most patients receive 10 to 14 days of pre-treatment and may be deemed ready for resection when there is
 - a. No documented blood pressure above 160/90 mm Hg for 24 hours
 - b. No demonstrated orthostatic hypotension
 - c. No ST-T abnormalities on the EKG
6. Consider pre-operative volume loading, as many patients with pheochromocytoma may be volume depleted.
 - a. The initiation of the alpha blockade may reveal the volume depletion, if the volume depletion has not been properly treated.

7. Co-morbid conditions related to pheochromocytoma, such as catecholamine-related cardiomyopathy, should be considered and appropriate pre-operative testing should be ordered.

Intra-operative

1. Pre-operative sedation is essential.
2. General, regional, and combined techniques have all been described for intra-operative management.
3. Histamine-releasing drugs and direct- or indirect-acting sympathomimetics should be used judiciously. Almost all drugs have been described successfully in managing intra-operative pheochromocytoma, however.
4. In addition to standard ASA monitors, a pre-induction arterial line is recommended. A pre-induction central venous catheter should be considered.
5. A pulmonary venous catheter and/or intra-operative transesophageal echocardiography can be placed as co-morbid conditions dictate.
6. Short-acting hypotensive agents such as esmolol, sodium nitroprusside, and nicardipine should be readily available.
7. Intravenous lidocaine may blunt the response to laryngoscopy and decrease the incidence of arrhythmias.
8. Magnesium should be considered as a vasodilator and to control dysrhythmias.
 - a. It may also have analgesic properties.
 - b. Patients with pheochromocytoma are often magnesium depleted.
9. Induction should be a slow and careful process regardless of the techniques or medications employed.
10. Manipulation of the tumor may cause dramatic spikes in blood pressure and should be anticipated.
 - a. A pneumoperitoneum, in the setting of a laparoscopic resection, may also cause increased catecholamine release.
11. Blood pressure may fall precipitously when the blood supply to the tumor is ligated during the surgical resection.
 - a. This can be controlled with adequate alpha blockade and volume repletion prior to the ligation.
 - b. If hypotension still occurs, it should be treated with volume and IV norepinephrine.
 - c. Rule out surgical bleeding as the cause of the hypotension.

Post-operative

1. Hypotension is often seen post-operatively. Hypovolemia and a decreased vasopressor response may be the cause, but bleeding should be considered.
2. Post-operative somnolence and decreased opioid requirements are frequently observed.
3. Hypoglycemia sometimes occurs after tumor removal when beta suppression is removed.
 - a. Peri-operative glucose testing is mandatory.
 - b. Glucose-containing intravenous fluids should be administered when indicated.

4. Persistent hypertension may signify incomplete tumor removal.
5. Careful consideration should be given to the post-operative level of care.
6. Intensive care unit monitoring is usually recommended.

BIBLIOGRAPHY

1. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams and Wilkins, 2006, pp 1142-4.
2. Fleisher LA. *Anesthesia and Uncommon Diseases*, 5th ed. Philadelphia: Saunders Elsevier, 2006, pp 440-3.
3. Yao Fsf. *Yao and Artusio's Anesthesiology*, 6th ed. Philadelphia: Lippincott Williams and Wilkins, 2008, pp 767-81.

72. Thyrotoxicosis^{1,2,3}

Donn Marciniak, MD

SAMPLE CASE

A 20-year-old female presents with untreated thyrotoxicosis for an emergency surgery. Her blood pressure is 180/100 mm Hg, pulse 125, and temperature 38 °C. What are her anesthetic risks? Would you do anything pre-operatively? Would a regional or general anesthetic be safer? How would you proceed if she refuses a regional anesthetic?

CLINICAL ISSUES

Definition

1. Thyrotoxicosis is a spectrum of disorders that involve increased circulating blood levels of thyroid hormone. The spectrum ranges from an asymptomatic elevation in laboratory values to life-threatening multisystem organ dysfunction.
2. Thyroid storm
 - a. Life-threatening exacerbation of hyperthyroidism
 - b. Usually develops in the undiagnosed or untreated hypothyroid patient because of the stress of surgery or a non-thyroid illness
 - c. Symptoms
 - i. Hyperthermia
 - ii. Tachycardia
 - iii. Dysrhythmias
 - iv. Myocardial ischemia
 - v. Congestive heart failure
 - vi. Agitation and confusion
 - d. Treatment
 - i. Large doses of propylthiouracil (PTU)
 - ii. Supportive measures to control the fever
 - (1) Cooling blankets
 - (2) Acetaminophen
 - iii. Intravenous fluids to restore intravascular volume
 - iv. Sodium iodide

v. Hydrocortisone

vi. Meperidine to reduce shivering

vii. Give digoxin for heart failure if the patient develops atrial fibrillation with a rapid ventricular response.

viii. Propranolol or esmolol

Incidence

1. Hyperthyroidism affects 0.2% of males and 2% of females.
2. Hyperthyroidism occurs in 0.2% of all pregnancies.

Etiology

1. Graves' disease
 - a. Most common etiology
 - b. Autoimmune pathogenesis with diffuse goiter and ophthalmopathy
 - c. IgG antibodies have effects similar to long-acting thyrotropin (thyroid stimulating hormone, TSH).
 - d. Placental transfer is possible.
 - e. Remits during pregnancy and is exacerbated after delivery.
2. Thyroid adenoma
3. Toxic nodular goiter
4. Thyroiditis
5. Pregnancy
6. Pituitary tumors secreting TSH
7. Exogenous iodide exposure
8. Factitious illness
9. Thyroid cancer
10. Choriocarcinoma

Signs and Symptoms

1. Anxiety or emotional instability
2. Exophthalmoses

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3. Diarrhea
4. Weight loss despite high caloric consumption
5. Increased bone resorption and hypercalcuria
6. Muscle weakness and fatigue
7. Heat intolerance
8. Mitral valve prolapse
9. Worsening angina or congestive heart failure
10. Atrial fibrillation or tachycardia
11. Hypertension
12. Increased cardiac output
13. Increased serum cortisol levels

Differential Diagnosis

1. Thyroid storm
2. Pheochromocytoma
3. Carcinoid syndrome
4. Malignant hyperthermia
5. Neuroleptic malignant syndrome
6. Anxiety
7. Infection
8. Cocaine intoxication
9. Strychnine poisoning
10. Anticholinergic exposure

Testing

1. Decreased levels of TSH: presence of a normal level almost always excludes a diagnosis of hyperthyroidism.
2. Increased plasma levels of tetraiodothyronine (thyroxine, T_4)
3. If the TSH level is low and the T_4 is normal, evaluate the triiodothyronine (T_3) level for T_3 thyrotoxicosis.
4. Diagnosis during pregnancy is difficult because T_4 -binding globulins are increased and thereby increase circulating T_4 concentrations.

Hyperthyroidism Treatment

1. The most important goal is to make the patient euthyroid before any surgical procedure.
2. Non-emergent
 - a. Antithyroid medications: can take six to eight weeks
 - i. Propylthiouracil (PTU)
 - ii. Methimazole
 - b. Radioactive iodine: not safe during pregnancy
 - c. Surgery to remove the thyroid
 - d. Glucocorticoids
 - i. Used in the management of severe thyrotoxicosis
 - ii. Reduce thyroid hormone secretion
 - iii. Reduce the peripheral conversion of T_4 to T_3
2. Emergent
 - a. β -blockers including propranolol
 - i. Used to treat symptoms including rapid heart rate, sweating, and anxiety.
 - ii. β -blockers do not prevent thyroid storm.



KO TREATMENT PLAN

Pre-operative

1. Elective surgery should be delayed in patients with poorly controlled hyperthyroidism.
2. Assess for airway compromise from an obstructing thyroid gland.
3. Consider judicious pre-medication with benzodiazepines, if indicated, to reduce the hemodynamic effects of anxiety.
4. Consider pre-medication with anti-thyroid drugs.
 - a. Methimazole, propylthiouracil (PTU), and potassium iodide will decrease the release of thyroid hormone, while PTU and glucocorticoids (dexamethasone 2 mg IV every 2 hours) will block the peripheral conversion of T_3 to T_4 .
 - b. PTU should be chosen over methimazole during pregnancy.
5. Avoid the pre-operative use of atropine, scopolamine, and ketamine secondary to the associated tachycardia.
 - a. Anticholinergics may cause tachycardia and further derangements in temperature regulation.

Intra-operative

1. Consider pre-operative placement of an arterial line.
2. Blood gas and electrolyte levels should be monitored aggressively.
3. Large-bore peripheral IVs should be placed in patients with symptomatic thyrotoxicosis or thyroid storm.
4. Thiopental may be considered for induction as it has anti-thyroid properties owing to its thiourea structure.
 - a. Avoid ketamine
5. MAC of volatile agents is not affected by hyperthyroidism but may appear so owing to the associated increase in cardiac output.
6. Exophthalmos warrants extra attention to eye protection.
7. Thyroid storm must be treated as soon as it is suspected and before lab values return.
8. Cooling may be necessary with cold lavage of body cavities, ice, and cooling blankets.
9. Consider a pre-induction central line and/or pulmonary artery catheter if the patient has a history of congestive heart failure, myocardial ischemia, pulmonary hypertension, renal failure, or significant hemodynamic instability.
10. Patients presenting for an emergent surgery may likely require a rapid-sequence endotracheal intubation. A multi-tiered approach to airway management should be in place with all equipment present before induction. A reinforced endotracheal tube should be considered.
 - a. Avoid pancuronium because of the associated tachycardia.
11. The patient's hemodynamics should be appropriately treated before induction, so as to avoid the perils of extreme hyper- or hypotension during and immediately after laryngoscopy.

- a. High output heart failure may improve with β -blockers.
 - b. Short-acting agents such as nitroglycerine, nitropruside, nicardipine, and esmolol should be used in treating the peri-operative blood pressure.
12. Sympathomimetic drugs used to treat hypotension may have an exaggerated effect and there is a theoretic concern that indirect sympathomimetics, such as ephedrine, may have lessened effect secondary to catecholamine depletion.
13. Regional anesthesia has the benefit of blocking the sympathetic nervous system. In patients without high output heart failure, a regional anesthetic is a viable option. An epidural without epinephrine may be preferable to a spinal anesthetic because of its slower onset and less severe hemodynamic perturbations.
14. Choosing a regional anesthetic in an emergent procedure is situation-dependent and the choice must take into consideration an unsecured airway, fluid shifts, and the location of the procedure.

Post-operative

1. The decision to extubate should be based on the global clinical picture and the difficulty encountered during the initial intubation.
2. If there is concern for airway collapse, extubating over a fiberoptic bronchoscope and directly observing airway patency may be considered.
3. Thyroid storm typically does not occur in the operating room but rather, 6 to 8 hours after surgery. Patient disposition should be carefully considered.
4. Aspirin should be avoided because it raises free thyroid hormone levels by displacing some of the protein-bound fraction.

BIBLIOGRAPHY

1. Fleisher, LA. *Anesthesia and Uncommon Diseases*, 5th ed. Philadelphia: Saunders Elsevier, 2006, pp 440-3.
2. Stoelting, RK. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 411-7.
3. Yao, FSF. *Yao and Artusio's Anesthesiology*, 6th ed. Philadelphia: Lippincott, Williams, and Wilkins, 2008, pp 753-66.

73. Hypothyroidism^{1,2,3}

Donn Marciniak, MD

SAMPLE CASE

A patient is scheduled for an emergency drainage of an abscess. Hypothyroidism was diagnosed 12 hours ago during her emergency room (ER) admission. Her total thyroxine (T_4) is less than 2 mcg/dL (normal values range from 4.5 to 10 mcg/dL) and her TSH level is elevated. What are your anesthetic concerns? What anesthetic would you choose? What specific post-operative complications would you expect?

CLINICAL ISSUES

Definition

Hypothyroidism is a common endocrine disorder resulting from inadequate circulating levels of triiodothyronine (T_3), T_4 , or both. It is typically a primary process in which the thyroid gland produces insufficient amounts of thyroid hormone despite adequate TSH production. It can also be secondary or tertiary. Secondary hypothyroidism occurs when the anterior pituitary gland does not produce enough thyroid-stimulating

hormone (TSH), and tertiary hypothyroidism occurs when the hypothalamus fails to produce enough thyrotropin-releasing hormone (TRH). **Patient presentation ranges from asymptomatic to comatose with multisystem organ failure.**

Incidence

1. 4.6% of the population is hypothyroid as defined by TSH levels (0.32% overt and 4.3% subclinical).
2. 1:4000 newborns are affected with congenital hypothyroidism (cretinism).
3. Hashimoto's thyroiditis is the most common presentation in the United States.

Etiology

1. Chronic thyroiditis (Hashimoto's disease)
 - a. Chronic autoimmune disease that results in a progressive destruction of the thyroid.
 - b. The thyroid becomes enlarged and potentially distorts or obstructs the airway.

73. Hypothyroidism

- c. Most common form of primary hypothyroidism in the United States
2. Iatrogenic
 - a. Neck or brain irradiation
 - b. Subtotal/total thyroidectomy
 - c. Radioiodine therapy or severe iodine depletion
 - d. Medications: amiodarone, methimazole, iodines, propylthiouracil, dopamine, and lithium
3. Genetic defects in hormone synthesis
4. Congenital defects in thyroid development
5. Anterior pituitary damage or destruction: Sheehan's syndrome
6. Hypothalamic dysfunction

Differential Diagnosis

1. Addison's disease
2. Sleep apnea
3. Chronic fatigue syndrome
4. Depression
5. Fibromyalgia
6. Infectious mononucleosis
7. Iodine deficiency
8. Lymphoma
9. Syndrome of inappropriate antidiuretic hormone (SIADH)

Signs and Symptoms

1. Fatigue and memory impairment
2. Hypothermia and cold intolerance
3. Depression and emotional lability
4. Weight gain and decreased appetite
5. Slowed speech and movements
6. Dry skin, coarse hair, or hair loss
7. Macroglossia
8. Goiter and hoarseness
9. May see systemic hypertension with a narrowed pulse pressure.
10. Bradycardia
11. Pericardial effusion
12. Non-pitting edema (myxedema)
13. Hyporeflexia with delayed relaxation
14. Blurred vision
15. Nerve entrapment

Testing

1. The measured TSH concentration is high when compared to the relatively low blood levels of T_3 or T_4 with primary hypothyroidism.
2. Resin triiodothyronine uptake (RT_3U) can be used to calculate the thyroid binding ratio. When multiplied by total T_4 , the free T_4 estimate is obtained. This correlates closely with the metabolic status of the patient.
3. Serum thyroxine by radioimmunoassay (T_4 -RIA) can yield the serum level of T_4 . Normal values are 4.5 to 10 mcg/dL. Since

most T_4 is bound to thyroid binding-globulin (TBG), processes that affect TBG levels can affect total T_4 levels. Androgens, hypoproteinemia, and nephrosis can lower the TBG and thus T_4 since free T_4 is less stable.

4. Free serum triiodothyronine by radioimmunoassay (T_3 -RIA) can detect serum levels of T_3 , normally 75 to 200 ng/dL. The upper limit of normal declines with age.

Clinical Features

1. Because lethargy and fatigue are common in hypothyroid patients, hypothyroidism is often not diagnosed until pre-operative testing since there may be a lack of motivation to see a primary care physician.
2. Cardiovascular changes are often the earliest clinical manifestation.
 - a. Systolic and diastolic myocardial functions are impaired and patients can occasionally experience congestive heart failure.
 - b. The EKG may show prolonged PR, QRS, and QT intervals due to pericardial effusions.
 - c. Torsades de pointes is possible.
3. Cortisol deficiency is possible secondary to the associated adrenal cortex atrophy.
4. The patient may be unable to excrete free water leading to SIADH. Fluids should be replaced judiciously, as these patients are prone to hyponatremia.
5. Myxedema coma
 - a. Rare
 - b. Symptoms
 - i. Loss of deep tendon reflexes
 - ii. Hypothermia
 - iii. Hypoventilation
 - iv. Hyponatremia
 - v. Hemodynamic instability
 - vi. Coma
 - vii. Death
 - c. Medical emergency
 - d. High mortality of 25–50%.
6. Hypothyroid patients with coronary artery disease are difficult to manage because establishing a euthyroid state may exacerbate angina.
 - a. Coronary revascularization may be considered emergent in some circumstances.
 - b. Thyroid replacement therapy must be cautiously undertaken.

Myxedema Coma Treatment

1. Admit the patient to the intensive care unit.
2. Correct hypovolemia and electrolyte abnormalities.
3. Intravenous (IV) levothyroxine (T_4) and liothyronine (T_3)
4. Rule out an infectious cause of the myxedema coma and treat with the appropriate antibiotics.
5. Give IV hydrocortisone 100 mg IV then repeat 25 mg IV every 6 hours.

73. Hypothyroidism

6. Tracheal intubation and controlled ventilation as needed.
7. Conserve body heat.

Hypothyroidism Treatment

1. Non-emergent
 - a. Levothyroxine (Synthroid) tablet
 - b. IV or intramuscular dosing can be given if the oral route is precluded for long periods of time.
2. Emergent (myxedema coma)
 - a. IV levothyroxine (Synthroid)
 - b. Symptomatic treatment



KO TREATMENT PLAN

Pre-operative

1. Elective surgery should probably be delayed for severe, symptomatic hypothyroidism to avoid the numerous associated peri-operative problems.
 - a. Intra-operative hypotension
 - b. Heart failure during cardiac surgery
 - c. Gastrointestinal complications
 - d. Neuropsychiatric complications
2. There is little reason to postpone an elective procedure in a patient with mild to moderate hypothyroidism.
 - a. There has been no associated increase in blood loss, arrhythmias, hypothermia, hyponatremia, delayed recovery, poor wound healing, pulmonary complications, and/or hospital duration when studies have compared hypothyroid and euthyroid peri-operative complications.
3. For elective procedures, patients should take their thyroid supplementation despite the long half-life of thyroxine.
4. Pre-operative cortisol supplementation should be considered because of the co-existing adrenal insufficiency.
5. Cautious pre-medication with opioids and benzodiazepines is advised due to a theoretical concern of increased sedation and respiratory depression.

Intra-operative

1. The induction of anesthesia should be accomplished with a drug that has minimal hemodynamic effects.
 - a. Ketamine has been considered the ideal induction drug in these patients because it increases myocardial

contractility, heart rate, and systemic vascular resistance, which may be detrimentally lowered in the hypothyroid patient.

- b. However, any induction drug can be used if given in a cautious and judicious manner.

2. Despite an elevated level of serum catecholamines, there is no evidence of a decreased response to exogenous catecholamines.
3. Volatile anesthetics should be titrated carefully due to the risk of cardiac depression.
4. The MAC is unaffected by hypothyroidism. It may appear decreased owing to a decrease in cardiac output (which may speed the rate of induction with an inhaled anesthetic) or due to the associated hypothermia.
5. Decreased production of CO₂ can make these patients prone to hypoventilation.
6. Use of an arterial line should be guided by the patient's clinical status and the procedure being performed.
7. Other intra-operative complications may include
 - a. Difficult intubation secondary to an enlarged tongue
 - b. Hypoglycemia
 - c. Anemia
 - d. Hyponatremia
 - e. Hypothermia

Post-operative

1. Recovery from sedation may be prolonged and an extended post-anesthesia care unit (PACU) stay may be required.
2. The inability to wean from mechanical ventilation may be due to over-sedation and hypothermia.
3. Despite the many concerns intra- and post-operatively, there is no evidence to suggest that patients with mild to moderate hypothyroidism experience more profound hypothermia or hypotension, require more cardiovascular support with vasopressors or ionotropes, or need more post-operative ventilator support than a euthyroid patient.

BIBLIOGRAPHY

1. Barash, PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams and Wilkins, 2006, pp 1142-4.
2. Fleisher, LA. *Anesthesia and Uncommon Diseases*, 5th ed. Philadelphia: Saunders Elsevier, 2006, pp 440-3.
3. Seeley RR, Stephens TD, Tate P. *Anatomy and Physiology*. 8th ed. New York: McGraw-Hill, 2008, p 621.

74. Peri-Operative Diabetes Management^{1,2,3,4,5,6,7}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 19-year-old female is scheduled for a septoplasty. She is a type I diabetic. She says that her blood glucose is controlled by an insulin pump. Her endocrinologist is from an outside hospital. What are your pre-operative concerns? What labs would you like? What is your anesthetic plan?

CLINICAL ISSUES

Incidence of Diabetes Mellitus

1. Affects about 7–10% of the population of the United States of America.
2. About 15–25% of hospitalized patients are diabetic, according to the American Diabetes Association.
3. Diabetics undergo surgery at a higher rate than non-diabetics.
4. Peri-operative morbidity and mortality are higher in diabetics.
 - a. A tighter inpatient glycemic control tends to decrease the incidence of
 - i. Morbidity and mortality
 - ii. Bloodstream infections
 - iii. Acute renal failure
 - iv. Transfusion requirements
 - v. Critical illness polyneuropathy
5. More than 90% of diabetics are considered to be type II diabetics.
 - a. Elderly
 - b. Overweight
 - c. Minority ethnicity
 - d. Lower socioeconomic background
6. About 3–5% of pregnant women develop gestational diabetes.

Etiology

1. Genetic susceptibility
2. Environmental triggers
3. Autoimmune

Diagnostic Criteria According to the American Diabetes Association:⁴

1. Symptoms of diabetes plus a random glucose level >200 mg/dL.

2. Fasting plasma glucose level >126 mg/dL
3. Two-hour plasma glucose level >200 mg/dL during an oral glucose tolerance test

Classification

1. Primary
 - a. Insulin-dependent
 - i. Type I
 - (1) Absolute deficiency in insulin production
 - (a) Pancreatic β -cell failure
 - (2) These patients will die without insulin secondary to the development of ketoacidosis.
 - (3) Onset usually before age 30
 - (4) Autoimmune destruction of the islet cells in the pancreas
 - b. Non-insulin-dependent
 - i. Type II
 - (1) Combination of
 - (a) Relative deficiency in insulin
 - (b) Insulin resistance
 - (c) Increased glucose production
 - (2) Milder form
 - (3) Affects all ages.
 - (4) Usually can be treated with diet, exercise, and oral hypoglycemic agents.
 - (5) These individuals are not at risk for ketoacidosis.
2. Secondary
 - a. Pancreatic disease
 - b. Hormonal abnormalities
 - c. Drug or chemically induced
 - d. Insulin receptor abnormalities
 - e. Genetic syndrome
3. Gestational diabetes
 - a. Glucose intolerance first recognized in pregnancy
 - b. Complicates about 4% of all pregnancies in the United States.
4. Syndrome X
 - a. Insulin resistance with hyperinsulinemia
 - b. Rarely have hyperglycemia.

Complications

1. End-organ pathology secondary to chronic hyperglycemia
 - a. Diabetic nephropathy leading to end-stage renal disease

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- b. Cardiac**
 - i. Coronary artery disease**
 - (1) Myocardial infarction
 - (2) Diabetes is an independent risk factor in post-operative myocardial ischemia among patients undergoing cardiac and non-cardiac surgery.
 - ii. Hypertension**
 - iii. Cardiac autonomic neuropathy**
 - iv. Microangiopathic cardiomyopathy**
- c. Stroke**
- d. Polyneuropathy**
- e. Stiff joints**
- f. Retinopathy**
- g. Increased risk of infections**
- h. Hypo-/hyperglycemia**
- i. Diabetic ketoacidosis**
- j. Nonketotic hyperosmolar coma**

Treatment

- 1. Type I**
 - a. Exogenous insulin**
 - i. Rapid-acting**
 - (1) Insulin lispro injection (Humalog): not recommended to be given intravenously/IV.
 - (a) Onset: 15–30 minutes (subcutaneous/SC)
 - (b) Peak: 30–90 minutes (SC)
 - (c) Duration 3–5 hours (SC)
 - (2) Regular (Humulin, Novolin)
 - (a) Onset: 15 minutes (IV) and 30–60 minutes (SC)
 - (b) Peak: 15–30 minutes (IV) and 2–5 hours (SC)
 - (c) Duration: 30–60 minutes (IV) and 8–12 hours (SC)
 - ii. Intermediate-acting**
 - (1) Isophane (NPH)
 - (a) Onset: 1–4 hours (SC)
 - (b) Peak: 4–14 hours (SC)
 - (c) Duration: 10–24 hours (SC)
 - iii. Insulin analog**
 - (1) Glargine (Lantus)
 - (a) Onset: 1–2 hours (SC)
 - (b) Peak: None (constant concentration over 24 hours.)
 - (c) Duration: 24 hours (SC)
 - (2) Aspart (Novolog)
 - (a) Onset: 5–15 minutes (SC)
 - (b) Peak: 1 hour (SC)
 - (c) Duration: 2–4 hours (SC)
 - 2. Type II**
 - a. Diet**
 - i. American Diabetes Association calorie-restricted diet**
 - b. Exercise**
 - c. Weight loss**
 - d. Oral hypoglycemic drugs**
 - (i) Insulin secretagogues: stimulate insulin secretion.
 - (1) Sulfonylureas: (short acting: glyburide, glipizide)

(ii) Biguanides: reduce hepatic glucose production and improve glucose utilization.

(1) Metformin

(a) Contraindication: renal failure secondary to the increased risk of lactic acidosis

(iii) α -glucosidase inhibitors: reduce postprandial hyperglycemia by delaying glucose absorption.

(1) Miglitol

(2) Acarbose

(iv) Thiazolidinediones: reduce insulin resistance by binding to receptors in the nucleus of adipocytes.

(1) Pioglitazone

e. Exogenous insulin

3. New therapies

a. Transplantation of pancreatic tissue

b. Islet cell transplant

c. Immunosuppression

d. Inhaled insulin

Factors That Increase Endogenous Insulin Requirements

- 1. High-carbohydrate diet**
 - a. Total parenteral nutrition**
 - b. Tube feeds**
 - c. Dextrose-containing intravenous fluids**
- 2. Infection**
- 3. Sepsis**
- 4. Stress**
- 5. Medications**
 - a. Corticosteroids**
 - b. Thyroid preparations**
 - c. Oral contraceptives**
 - d. Thiazide diuretics**
 - e. Atypical antipsychotics**
 - f. Lithium**
 - g. Protease inhibitors**
 - h. Rifampin**
 - i. Phenytoin**
 - j. Medications mixed with dextrose-containing solutions**

Factors That Decrease Endogenous Insulin Requirements

- 1. Exercise**
- 2. Coumadin**
- 3. Decreased carbohydrate intake**
 - a. Fasting**
 - b. Nausea and vomiting**

Diagnosis and Treatment of Diabetic Ketoacidosis (DKA)

- 1. Acute medical emergency**
- 2. Symptoms**
 - a. Acute abdominal pain**
 - b. Nausea and vomiting**

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- c. Lethargy
- d. Signs of hypovolemia
- 3. Absolute or relative deficiency of insulin that results in ketone acids in the blood
 - a. Hyperglycemia
 - b. Glucosuria
 - c. Intracellular dehydration
 - d. Acidosis
 - e. Electrolyte imbalance
- 4. Diagnosis
 - a. Serum ketone acids >7 mmol/L
 - b. Decrease in serum bicarbonate to < 10 mEq/L
 - c. Decrease in pH level to <7.25
- 5. Labs that should be ordered
 - a. Urinalysis
 - b. Glucose level
 - c. Serum electrolytes to determine the anion gap
 - d. Serum ketone estimation
 - e. Urea nitrogen level
 - f. Complete blood cell count
 - g. Arterial blood gas: acid-base balance
- 5. Treatment
 - a. Fluids
 - i. Restore intravascular volume
 - (1) Start with 1 L of normal saline and continue an IV infusion of normal saline.
 - (a) 0.45% normal saline should be used if the patient's osmolality is elevated.
 - (2) 5% dextrose IV should be started once the serum glucose falls below 250–300 mg/dL to prevent secondary hypoglycemia.
 - ii. Fluid therapy should be guided by urine output and central venous pressure monitoring.
 - b. Insulin
 - i. Regular insulin 10–20 units (or 0.2 units/kg) IV should be given initially.
 - ii. Infusion of 1–2 units of regular insulin per hour (or 0.1 unit/kg/hour) should be started depending on the serum glucose level.
 - iii. Goal rate of glucose level reduction should not exceed 50 mg/dL/hour.
 - (1) A rapid rate of reduction can lead to cerebral edema.
 - c. Sodium bicarbonate
 - i. Used to correct severe metabolic acidosis (pH <7.20).
 - d. Potassium
 - i. Potassium stores are often depleted.
 - ii. Serum potassium levels will decrease as a result of
 - (1) Hemodilution
 - (2) The correction of acidosis results in the movement of potassium from the extracellular space to the intracellular space.
 - iii. Potassium should be added to the IV fluids 3–4 hours after the initiation of therapy.
 - e. Treat the underlying condition.
 - i. Antibiotics for sepsis

Diagnosis and Treatment of Nonketotic Hyperosmolar Coma

1. Syndrome of profound dehydration
2. Usually seen in patients with type II diabetes when they are unable to drink enough fluids to keep up with urinary losses secondary to glycosuria.
3. No ketoacidosis
4. Presents with
 - a. Extreme hyperglycemia: 1,000 mg/dL
 - b. Hyperosmolality
 - c. Volume depletion
 - d. Mental status changes
5. Mortality rate is around 50%.
6. Treatment
 - a. Rapid administration of large amounts of IV fluid
 - b. Insulin
 - c. Dextrose
 - d. Potassium

Hypoglycemia

1. Blood glucose <50 mg/dL
2. Complications of hypoglycemia
 - a. Arrhythmias: bradycardia
 - b. Hypotension
 - c. Seizures
 - d. Irritability
 - e. Cognitive defects
 - f. Respiratory failure
 - g. Death
3. Risk factors for hypoglycemia
 - a. Decreased oral intake
 - b. Renal insufficiency
 - c. Liver disease
 - d. Infection
 - e. Pregnancy
 - f. Cancer
 - g. Burns
 - h. Adrenal insufficiency
4. Risk factors for hypoglycemic unawareness
 - a. β -blockade
 - b. Sedation
 - c. Advanced age
 - d. Long history of diabetes
 - e. Diabetic neuropathy
5. Treatment
 - a. 15 g of fast-acting carbohydrate
 - i. 4 oz of fruit juice or soda
 - ii. 25 ml (one ampule) of 50% dextrose IV push
 - b. Recheck the blood glucose after 15 minutes.
 - c. Repeat the glucose administration if the glucose level is <80 mg/dL.
 - d. Repeat the blood glucose check 60 minutes after the last glucose administration.

Acute Hyperglycemia

1. Consequences
 - a. Impaired wound healing
 - b. Dehydration
 - i. Osmotic diuretic effect of high-serum glucose levels
 - c. Impaired immune system response
 - i. Increased risk of post-operative infection
 - d. Proteolysis

TKO: Anesthetic Considerations:

1. Diabetes affects oxygen transport and can present with decreased oxygen saturation, especially in pregnant patients.
2. Commonly present with autonomic dysfunction
 - a. Intra-operative hypothermia
 - b. Inability to regulate blood pressure: orthostatic hypotension
3. Increased risk of coronary artery disease
 - a. Patients more likely to have “silent cardiac ischemia.”
 - b. Consider peri-operative β -blocker therapy.
4. Delayed gastric emptying
 - a. All diabetics should be considered to have a “full stomach.”
5. More likely to have cerebrovascular accidents and peripheral vascular disease
6. The altered consciousness of anesthesia may mask the symptoms of hypoglycemia.

Importance of Strict Glucose Control

1. Prevents diabetic ketoacidosis
2. Prevents hyper-/hypoglycemia
3. Prevents dehydration
4. Improves wound healing

Serum Glucose Goals

1. Serum blood glucose <110 mg/dL in the intensive care unit (ICU) setting
2. Serum blood glucose less than 180 mg/dL in the non-ICU setting
 - a. Pre-prandial blood glucose should be <110 mg/dL.



KO TREATMENT PLAN

Pre-operative

1. Diabetes can affect every organ system.
2. Perform a complete history and physical examination.
 - a. Evaluate the patient for symptoms of polyuria/polydipsia, and blurred vision.
 - b. Cardiac history: asymptomatic cardiac ischemia is common.
 - i. EKG

- ii. Stress test (as needed)
 - iii. Echocardiography (as needed)
- c. Autonomic neuropathy
 - i. Orthostatic hypotension
 - ii. Lack of sweating
 - iii. Early satiety
 - iv. Gastroparesis
 - (1) Gastric reflux
 - (2) Nausea/vomiting
 - (3) Regurgitation
 - (4) Aspiration
 - v. Lack of change in pulse with deep inspiration
 - vi. Resting tachycardia
 - vii. Painless myocardial ischemia
- d. Peripheral neuropathy
- e. Syncope
- f. Erectile or bladder dysfunction
- g. Cerebrovascular disease
- h. Renal dysfunction
- i. Airway exam
 - i. Stiff joint syndrome
 - (1) Limited atlanto-occipital joint mobility
 - (2) Limited temporomandibular joint mobility
 - (3) Limited cervical spine mobility
 - (4) Positive prayer sign
 - ii. Associated obesity
3. Check a hemoglobin A_{1c} to determine the patient’s chronic level of control.
 - a. Used to assess the glucose level over the past 2–3 months
 - b. A value >9% is an indicator of very poor control.
4. Check basic electrolytes and renal function tests.
5. Urinalysis for detecting glucose and ketones
6. Complete blood count
7. Arterial blood gas to determine acid-base status
8. Last intake of a meal?
9. Last dose of insulin?
10. Type I diabetics should be the first case of the day in order to disrupt their treatment regimen as little as possible.
11. Check the serum glucose and repeat again immediately pre-operatively.
12. If time allows, talk with the patient’s endocrinologist about the preferred peri-operative diabetes management for the patient.

Pre-operative Preparation

1. Diet-controlled diabetic
 - a. Pre-operative fasting blood glucose on the morning of surgery
 - b. Repeat check every 3 hours until oral intake is resumed.
 - c. If the surgery is major or the patient has very poorly controlled blood glucose (>200 mg/dL), an intravenous infusion of insulin and dextrose should be considered.
 - i. Hourly glucose monitoring

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2. Diabetics requiring medical treatment

a. Type II diabetic not taking insulin

i. Discontinue oral hypoglycemic agents 1–2 days pre-operatively.

ii. Omit the morning dose of the oral hypoglycemic drugs.

iii. Discontinue Metformin at least 24 hours before surgery.

(1) Metformin possesses a risk for the development of lactic acidosis.

(2) It should be stopped 24 hours before and for at least 48 hours after any procedure using intravenous contrast dye.

(3) Avoid completely in patients with renal dysfunction, congestive heart failure, recent myocardial infarction, any hypoxic state, current alcohol abuse, or impaired hepatic function.

iv. Serum glucose should be checked immediately before and after the surgery.

(1) For minor surgery, a blood glucose >200 mg/dL can be treated with 4–10 units of regular insulin subcutaneously.

(2) For major surgery (lasting greater than 1 hour), intravenous insulin infusions should be used with more frequent blood sugar checks.

v. Most oral agents should be restarted once the patient resumes eating.

(1) Metformin should be held for 48–72 hours following surgery or the infusion of any iodinated radiocontrast dye.

(a) Renal function should be evaluated and determined to be normal prior to the reinstatement of Metformin.

b. Diabetic compliant with insulin therapy

i. There are many protocols, with no established consensus.

(1) Long-acting insulin, such as Lantus, can be stopped 2 days prior to surgery.

(a) The patient should then begin a regimen of intermediate- and short-acting insulin.

(b) Recently, it has been determined that if the patient has been under good control, the long-acting Lantus can be continued throughout the day of surgery.

i. It maintains a stable glucose level for 24 hours.

ii. It acts like a basal infusion.

(2) Often, patients are told to reduce their bedtime insulin dose once they are NPO and take a half dose of their usual long- or intermediate-acting insulin on the morning of surgery.

(a) These patients require frequent glucose checks. (Hourly checks are preferred.)

(b) They are to use short-acting insulin as needed.

(c) They may require a 5% dextrose IV infusion on the morning of surgery (2 mg/kg/min).

(i) This infusion is usually started at the time the patient would have had the next meal.

(3) Continuation of SC insulin infusion pump

(a) The pump is often stopped just prior to surgery, and management is based on frequent blood glucose measurements.

(b) Glucose management often requires a continuous IV insulin infusion.

(4) SC regular insulin sliding scale with frequent blood sugar checks

(5) IV infusion plus frequent blood sugar checks

(a) The infusion rate can be determined by calculating insulin (units/hr) = serum glucose (mg/dL)/150.

ii. A blood glucose level should be measured the morning of surgery.

(1) The blood glucose level should be <250 mg/dL before proceeding to surgery.

(2) The glucose level should be treated accordingly with regular insulin boluses or infusion.

iii. Intravenous insulin infusions should be stopped and the usual insulin treatment resumed once oral intake is established.

3. Glucose administration

a. Glucose should be provided to prevent catabolism, starvation ketosis, and insulin-induced hypoglycemia.

4. Potassium infusion

a. The infusion of insulin and glucose induces an intracellular translocation of potassium, resulting in a potential hypokalemia.

5. Hydration

a. Avoid Ringer's lactate.

b. Normal saline is the recommended fluid.

6. Antibiotics

7. Pre-medication

a. Metoclopramide: improves gastric emptying.

Emergency Surgery

1. Rule out DKA and diabetic autonomic neuropathy of the gastrointestinal tract.

a. These can often present with abdominal pain.

b. Surgery can be prevented completely if this is the cause of the abdominal pain.

2. Obtain good intravenous access.

3. Send labs.

a. Glucose

b. Electrolytes

c. Acid-base assessment

4. Correct lab abnormalities as quickly as possible.

5. Life-saving surgery should not be delayed.

a. In a patient with DKA, the surgery should be delayed as long as safely possible in an attempt to correct the associated electrolyte abnormalities.

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Intra-operative

1. Standard ASA monitors
2. Surgery results in a stress response with a resultant increase in blood glucose levels.
 - a. Plasma insulin levels remain constant.
 - b. There is a phase of relative insulin resistance after surgery.
3. This patient should have a rapid-sequence induction and intubation secondary to her high risk of gastroparesis and aspiration.
4. Etomidate is recommended in patients with autonomic dysfunction.
 - a. Less risk of induction-induced hypotension than with propofol and thiopental
5. I would consider the placement of an arterial line for frequent blood glucose and electrolyte measurements.
 - a. Intra-operative hyperglycemia should be treated with IV regular insulin.
 - i. Small doses up to 10 units of regular insulin can be bolused at a time.
 - ii. Each unit of regular insulin lowers the serum glucose level by about 30 mg/dL.
 - iii. A continuous infusion of regular insulin starting at 1–2 units per hour can be started.
 - iv. The goal glucose level should be around 80–150 mg/dL.
6. Maintenance
 - a. Sevoflurane and isoflurane impair glucose tolerance to the same degree.
7. Check the serum glucose every 1–2 hours.
8. Maintain normothermia.

Post-operative

1. Type I diabetics
 - a. Continue an infusion of 5–10% dextrose-insulin-potassium, as determined by the blood glucose and potassium levels and the projected length of the NPO status or a “sliding-scale” of subcutaneous regular insulin.
 - b. Check electrolytes and serum glucose every 1–2 hours.
 - c. Resume the patient’s usual protocol once she resumes a regular diet.
 - d. Once this sample patient is awake and alert, you can resume her SC insulin pump.
2. Type II diabetics
 - a. Serum glucose levels every 4 hours are usually sufficient.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 714–8.
2. Goldman L, Ausiello D. *Cecil*, 23rd ed. Philadelphia: Saunders Elsevier, 2008, Ch. 457.
3. Piccini JP, Nilsson KR. *The Osler Medical Handbook*, 2nd ed. Philadelphia: Saunders Elsevier, 2006, Ch. 25.
4. Yao FSF, Fontes ML, Malhotra V. *Yao & Artusio's Anesthesiology: Problem-Oriented Patient Management*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp 782–95.
5. Lor-Trivedi M. Perioperative management of the diabetic patient. *eMedicine from Web MD*. September 4, 2008.
6. Marks JB. Perioperative management of diabetes. *American Family Physician*, 2003;67:93–100.
7. Dagogo-Jack S, George K. Management of diabetes mellitus in surgical patients. *Diabetes Spectrum*, 2002;15:44–8.

75. Rapid Sequence Intubation (RSI)^{1,2}

Ryan J. Gunselman, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

Following a motor vehicle collision, a 24-year-old male is brought to the operating room for an emergent exploratory laparotomy due to a rigid abdomen and positive focused abdominal sonography for trauma (FAST) exam in the emergency department. The patient is awake and alert. However, he is in pain and is somewhat uncooperative. His blood alcohol content (BAC) is 0.24%. The patient denies any significant medical history. He admits to binge drinking and smoking. The patient said he last ate around 2 hours ago while at a bar. His airway exam reveals a mallampati II with a thyromental distance of 6 cm. How do you plan to secure this patient's airway? Does he need a RSI? If so, how would you do it? What are the situations in which a person would require a RSI? What are the contraindications to a RSI?

CLINICAL ISSUES

Definition of RSI

1. Technique used for rapid endotracheal tube placement
2. Performed in those patients who are at an increased risk for aspiration of gastric contents

Patient Identification

Identifying which patients are at risk for aspiration is the first and most important step in RSI.

1. Full stomach: <8 hrs fasting
2. Last oral intake is unknown.
3. Morbid obesity
 - a. These patients typically have gastroparesis in addition to increased intra-abdominal pressures, both of which increase risk for aspiration.
4. Severe gastroesophageal reflux disease (GERD)
5. Decreased gastrointestinal motility
6. Intra-abdominal processes: abscess, obstruction, bleeding, etc.
7. Trauma
 - a. NPO status is often unknown.
 - b. The patient may be unable to protect his airway due to depressed mental status following head trauma.
 - c. The patient has delayed gastric emptying immediately once the accident occurs.

8. Pregnancy
 - a. Delayed gastric emptying
 - b. More acidic gastric contents
 - c. Increased intra-abdominal pressures
9. Decreased gag reflex or depressed mental status: unable to protect own airway

Preparation for a RSI

1. History and physical exam, including complete airway exam and review of any drug-related allergies
2. Make sure that all emergency airway equipment is available.
 - a. Laryngoscope blades
 - b. Suction
 - c. Oxygen
 - d. Mask
 - e. Oral and nasal airways
 - f. Gum Elastic Bougie
 - g. Glidescope/Wu-scope/fiber optic scope

Contraindications to a RSI

1. Difficult airway
 - a. History per the patient or medical record.
 - b. Short neck, limited range of motion, small chin
 - c. Cervical collar in place
 - d. Facial injuries
 - e. Facial burn
2. Contraindication to succinylcholine
 - a. Elevated potassium levels
 - b. History of malignant hyperthermia
 - c. Pseudocholinesterase deficiency
3. Cervical spine injury
 - a. Cricoid pressure could cause increased damage to the cervical spine.
 - b. An awake fiber optic intubation is probably the best choice in this patient to avoid any additional cervical damage during the intubation.

TKO: If a RSI is contraindicated, then the risk of a failed intubation takes priority over the aspiration risk.

- a. Awake fiber optic intubation
- b. Awake direct laryngoscopy
- c. Blind nasal intubation



Figure 75.1. Rapid sequence intubation procedure. Photo credit: J. Lovich-Sapola, MD.



KO TREATMENT PLAN

Pre-operative

1. Proper emergency airway equipment available
 - a. Laryngoscope blades
 - b. Suction
 - c. Oxygen
 - d. Mask
 - e. Oral and nasal airways
 - f. Bougie
 - g. Glidescope/Wu-scope/fiber optic scope
 - h. Pillows/blankets to optimize sniffing position
 - i. Intubating laryngeal mask airway
 - j. Cricothyroidotomy kit
 - k. Jet ventilation tubing
2. Adequate intravenous (IV) access
3. Medications
 - a. Induction agent, narcotic, succinylcholine, non-depolarizing muscle relaxant
4. Placement of a nasogastric/orogastric (NG/OG) tube to suction the contents of the patient's stomach is not recommended.
 - a. Will only be able to suction out some fluids, and not solid food.
- b. It is believed that the NG/OG passing through the lower esophageal sphincter makes it incompetent and can actually "wick" up the gastric contents.
- c. If an NG/OG is currently in place, it should be suctioned prior to the RSI and should not be removed for the intubation.

Intra-operative

1. Apply the standard ASA monitors.
2. Preoxygenate with 100% oxygen via a face mask for 3–5 minutes, refraining from any positive pressure that may introduce intragastric distension.
3. Cricoid pressure: Sellick's maneuver
 - a. In most situations, this is begun at the time of the induction agent administration, or immediately after due to the fact that it can be uncomfortable for an awake, conscious patient.
 - b. Palpation of the cricoid cartilage is performed and approximately 8–10 pounds of posterior-cephalad pressure is applied by an assistant.
 - c. This technique should occlude the esophagus thereby helping prevent aspiration of gastric contents.
4. Medication administration
 - a. Induction with propofol, etomidate, thiopental, or ketamine
 - i. Each agent can be used, but you must choose one and defend it.
 - ii. Assuming our sample case patient is hemodynamically stable, I would perform this RSI with propofol. He is an otherwise healthy, young male without any cardiac history.
 - iii. If the sample patient was hemodynamically unstable, I would choose ketamine or etomidate.
 - iv. Just pick one induction agent. The examiners want to know how you will do it, not all the different ways it could be done.
 - b. Paralytic is given immediately after the induction agent.
 - a. Succinylcholine is most commonly used for RSI.
 - b. A fast-acting non-depolarizing muscle relaxant can also be used effectively (i.e., 1.2 mg/kg of rocuronium). This is often called a "modified rapid sequence intubation."
 - c. Depending on the agent used, one can monitor for fasciculations to cease or check train of four to assess adequacy of paralysis to facilitate endotracheal intubation.
5. Do not ventilate the patient during the induction.
 - a. This increases the risk of aspiration by potentially filling the stomach with air.
 - b. If the patient absolutely requires ventilation, keep the peak airway pressures <20 cm H₂O.
6. Direct laryngoscopy
 - a. Once the patient is adequately paralyzed, visualization of the larynx is performed using a standard laryngoscope blade.

- i. Direct laryngoscopy with other devices has been documented to have success. (i.e., glidescope, Wu-scope, etc.).
- b. Once the vocal cords are visualized, an endotracheal tube is passed through and the balloon is inflated.
- c. Cricoid pressure is maintained until end-tidal CO₂ is confirmed and breath sounds are auscultated bilaterally in the chest.
- d. The endotracheal tube is then secured in the normal fashion.

Post-operative

1. Extubation criteria should be met as in any other case.

2. Patients meeting the criteria should be completely awake and able to protect their airway prior to endotracheal tube removal as they are still at higher risk for aspiration.
3. Many practitioners will choose to suction the stomach contents with an NG/OG tube prior to extubation or waking the patient.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 481–547 and 1617–51.
2. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006, pp 287–8.

76. Cervical Spine Precautions^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 27-year-old male presents to the operating room after falling off scaffolding at a concert 3 hours ago. He is having his left tibia repaired. He denies any other injury. His toxicology screen is positive for alcohol and cocaine. What would be an acceptable evaluation of his cervical spine?

CLINICAL ISSUES

Incidence

1. Roughly 2% of patients with a closed-head injury who survive to reach the hospital will have a fracture of the cervical spine.
2. The incidence of cervical spine injury is about 2–6% for all blunt trauma victims.
3. Ligamentous injuries are more common at C5 and C6, whereas fractures are often seen at C1 and C2.

Movement with Intubation

1. The primary force applied by direct laryngoscopy is extension of the occiput on C1 combined with flexion at the lower vertebrae.
2. The more difficult the glottic exposure, the greater the force applied.

3. Thus, it is possible to aggravate or cause a serious spinal cord injury with the “hypnotic-relaxant-direct laryngoscopy” approach.

How Is the Cervical Spine Cleared?

1. Alert, non-intoxicated patients without neck pain, depressed level of consciousness, intoxication, distracting injury, or neurologic abnormality or symptoms can be clinically cleared.
2. A patient’s cervical spine can not be cleared, even with appropriate radiographs, if he has a distracting injury. As in this sample case, cervical injury must be assumed and cervical spine precautions must be taken.
3. A normal cervical spine X-ray does not rule out cervical injury. A ligamentous injury will not always be identifiable on an X-ray.
4. A CT/MRI scan may be required to rule out ligamentous injury. There is rarely time for this testing in an emergency/trauma case.

Indications for Cervical Spine Precautions

1. All acute trauma patients with depressed level of consciousness
2. Patients reporting neck pain



Figure 76.1. Trauma response helicopter. Photo credit: Metro Life Flight, Cleveland, Ohio.

3. Posterior midline cervical spine tenderness
4. Upper extremity paresthesias
5. Focal motor deficits
6. Whenever the pain of other injuries is likely to mask the neck pain
7. Falls, diving accidents, and high-speed motor vehicle accidents



KO TREATMENT PLAN

Intra-operative

1. When there is any uncertainty regarding the airway or the cervical spine, direct laryngoscopy with atlanto-occipital extension should be avoided.
2. If a rapid-sequence intubation is used, the standard approach should include in-line stabilization. Be careful when applying cricoid pressure. Overly aggressive pressure might lead to cervical damage.

In-line Stabilization

1. Hold the patient's occiput firmly on the backboard or operating table. This limits the amount of "sniff."
2. Immobilization of the cervical spine in a neutral position can be accomplished by using an assistant to provide manual in-line stabilization. A semi-rigid collar, sandbags placed on both sides of the head and neck, and the backboard can

also help in maintaining the proper neck position. Note that well-fitted hard collars make it almost impossible to do direct laryngoscopy.

3. Hold the patient's head in line with the cervical spine to prevent any cervical twisting.
4. Pre-oxygenate the patient, ideally for 2 to 3 minutes, but this time may have to be limited depending on the patient's condition.
5. Direct laryngoscopy will be more difficult with correct in-line stabilization.
6. Some physicians recommend leaving the posterior half of the cervical collar in place during laryngoscopy to act as a strut between the shoulders and the occiput and therefore serve to further limit the atlanto-occipital extension.
7. While the cervical neck is immobilized, you can use different techniques to improve your success of intubation including McCoy blade, Wu/Bullard scope, Glidescope, flexible fiber optic, light wand, and retrograde intubation.
8. If the cervical collar was removed during the intubation, it should be reapplied immediately after the intubation is confirmed.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2152-3.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 1265.
3. Smith CE. *Trauma Anesthesia*. New York: Cambridge University Press, 2008, pp 346-7.

77. Burn Anesthesia¹

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 47-year-old male was upset with his girlfriend and poured gasoline on her house and set it on fire. While admiring his work he sat back and lit a cigarette. At this point he went up in flames. He presents to the emergency room with a 75% total body surface area (TBSA) burn. He is awake and talking. He has facial burns, singed nasal hair, and increased secretions. He is telling you that he will be fine, and doesn't need a breathing tube. Pulse ox reads 99%. What are your specific concerns? What is your plan for airway management? What is your plan for fluid therapy?

CLINICAL ISSUES

Airway

1. Upper airway inhalation injury is usually due to a heat injury, which leads to swelling and upper airway obstruction secondary to edema.
2. Patients with significant burns to the face and neck are at increased risk of airway injury.
3. Signs that are highly indicative of inhalation injury include
 - a. Facial burns
 - b. Airway soot
 - c. Singed nasal hair
4. A patient with inhalation injury can progress from a stable to a catastrophic airway closure within seconds.
5. Some symptoms of impending obstruction include
 - a. Carbonaceous sputum
 - b. Increasing respiratory rate
 - c. Increased secretions
 - d. Stridor
 - e. Dyspnea
 - f. Use of accessory muscles
 - g. Dysphagia
 - h. Progressive hoarseness
6. It is important to note that a patient with minimal airway distress should be questioned as to the events associated with the burn (the patient's name and information), medical history, allergies, surgical history, medications, and other pertinent information prior to manipulation of the airway.
7. When a patient has upper airway injury with impending signs of obstruction, the patient will require rapid tracheal intubation.
8. Other indications for immediate intubation include
 - a. Cardiovascular instability
 - b. Central nervous system (CNS) depression
 - c. Massive burns greater than 60% total body surface area
9. In general, it is always best to intubate early rather than late. With time and treatment of the patient with massive fluid resuscitation, the likelihood of airway edema increases significantly.
10. It is important to evaluate the patient's airway for potential difficulty of intubation prior to any airway manipulation. The following conditions should raise one's suspicions of a potentially difficult airway independent of the burn injury. These include
 - a. Full stomach
 - b. Emergency situation
 - c. Cervical collar in place
 - d. Abnormal anatomy
11. Patients who are morbidly obese can be very difficult to ventilate. The large head and face make it difficult to mask ventilate, and the patient does not tolerate lying supine due to decreased functional residual capacity and decreased respiratory compliance.
12. Also, a pregnant patient is at risk for a difficult intubation secondary to the anatomic changes that occur with pregnancy including
 - a. Pharyngolaryngeal edema
 - b. Weight gain
 - c. Enlarged breasts
 - d. Full dentition
 - e. Propensity for rapid arterial oxygen desaturation

Tracheal Intubation with a Normal-Appearing Airway

1. Tracheal intubation of a patient with a normal-appearing airway is usually accomplished with a rapid-sequence intubation using an intravenous induction drug and a rapid-acting muscle relaxant.
2. Succinylcholine is generally considered safe in the first 24 hours after a burn.
3. Rocuronium is a good alternative to succinylcholine during the initial 24 hours and can also be given safely after this initial period.
4. Always pre-oxygenate the patient with 100% oxygen prior to airway manipulation to ensure a good oxygen reserve during the patient's period of apnea.

5. It is important to remember that if the patient has a cervical collar in place, the patient will require manual in-line stabilization during the airway manipulation.
6. Once the endotracheal tube is in place, it is important that it is adequately secured. Unfortunately, the burned face often makes this difficult secondary to topical wound agents, continual swelling and edema of the face and neck, and fluid extruding through the facial burn.

Tracheal Intubation with an Abnormal-Appearing Airway

1. Tracheal intubation of a patient with an abnormal airway is most often secured safely with the patient awake.
2. The most important things to remember during an awake intubation are topical anesthesia, proper patient positioning, and supplemental oxygen.
3. Minimal intravenous opioids are recommended because excessive opioid sedation may worsen the airway obstruction.
4. The safest technique for endotracheal tube placement depends on the operator's expertise. Alternatives include
 - a. Flexible fiber optic scope
 - b. Bullard scope
 - c. Wu scope
 - d. Laryngeal mask airway (LMA)
 - e. Glide Scope
5. If the patient will not cooperate with an awake intubation, then consider taking the patient to the operating room for an inhalational induction with continued spontaneous ventilation, or a surgical airway including retrograde techniques, transtracheal jet ventilation, cricothyroidotomy, or tracheostomy.
6. Inhalational induction with oxygen and sevoflurane is often necessary for children.

Respiratory Physiology

1. Predictive indicators of an inhalation injury include
 - a. History of a closed-space fire
 - b. Impaired mental status
 - c. Loss of consciousness
 - d. Associated drug or alcohol use
 - e. Facial burns
 - f. Airway soot
 - g. Singed nasal hair
 - h. Abnormal finding on the nasopharyngoscopy and bronchoscopy
 - i. Airway edema
 - j. Carbonaceous material in the airway
 - k. Abnormal flow-volume loops showing extra thoracic obstruction and an elevated carbon monoxide level greater than 15%.
2. The first phase of inhalation injury may include asphyxia and acute toxicity.

- a. Asphyxia occurs secondary to the lack of oxygen during combustion.
 - b. Acute toxicity occurs with the inhalation of carbon monoxide and cyanide.
 - c. In any patient with a burn injury it should be presumed that they will have some degree of carbon monoxide and cyanide poisoning.
3. Carbon monoxide (CO) toxicity
 - a. Carbon monoxide is a byproduct of combustion.
 - b. It is responsible for 80% of deaths associated with smoke inhalation and accounts for the majority of deaths that occur at the scene of the fire.
 - c. Carbon monoxide has an affinity for hemoglobin that is 250 times greater than oxygen.
 - d. Carbon monoxide preferentially binds with the hemoglobin molecule and prevents oxygen from loading onto the molecule. This causes a leftward shift on the oxygen dissociation curve, impairing oxygen delivery and unloading at the cellular level. This results in tissue hypoxia and metabolic acidosis.
 - e. Carbon monoxide poisoning can be diagnosed with an arterial blood gas and measurement of carboxyhemoglobin levels with co-oximeter blood analysis. The arterial oxygen pressure, pulse oximetry saturation, and arterial oxygen saturation may be normal and misleading. Pulse oximetry interprets the carboxyhemoglobin as oxyhemoglobin, therefore giving a falsely elevated SpO₂.
 - f. Mixed venous oxygen saturation monitoring does not detect the presence of carboxyhemoglobin and progressively overestimates fractional oxyhemoglobin and carboxyhemoglobin increases.
 - g. Patients with carbon monoxide poisoning may manifest no signs of peripheral cyanosis secondary to their characteristic "cherry red" appearance.
 - h. At a carbon monoxide level of less than 15% the patient rarely has any signs or symptoms, but at 15–20% the patient will likely have a headache, tinnitus, and confusion. At a carbon monoxide level of 20–40% the patient will have nausea, fatigue, and disorientation, and at 40–60% the patient will have hallucinations, display combativeness, and show cardiovascular instability, followed by death when the levels are greater than 60%.
 4. The second phase of inhalation injury begins at 24 to 96 hours after the injury and is the result of pulmonary parenchymal damage that is caused by the chemical irritation of smoke inhalation. Airway edema, tracheobronchitis, pulmonary edema, atelectasis, increased airway resistance, and decreased static lung compliance are likely to occur. The patient often has symptoms of dyspnea, rales, rhonchi, wheezing, and copious tracheal secretions and exudates. The secretions are often very viscous and may contain carbonaceous particles and pieces of mucous membrane. The clinical picture is almost identical to adult respiratory distress syndrome (ARDS). The initial inhalation injury and resultant pneumonias often influence the mortality, which increases from 20% with inhalation injury alone to 60% when combined with pneumonia.



KO TREATMENT PLAN

Pre-operative

1. Intravenous access
 - a. Establishing vascular access is extremely important prior to burn excision and grafting procedures.
 - b. In most cases, a minimum of two large-bore peripheral intravenous lines or one peripheral and one central line are necessary.
 - c. It is often difficult to place intravenous lines in these patients due to the extent of the burn.
 - d. Central venous trauma lines provide excellent routes for rapid fluid administration and the delivery of vasoactive drugs, with the additional advantage of the placement of central venous or pulmonary artery catheters.
 - e. Pulmonary artery catheters are not routinely placed because of the risk of infection but can be used for a brief period of time in patients with burn injury and ischemia or valvular heart disease to guide fluid administration.
 - f. The internal jugular and subclavian veins are most commonly used for catheterization, but femoral vessels can be used if the burn involves the neck.
 - g. Traditionally, central venous access was guided only by the anatomic landmarks and arterial pulsations. This blind approach assumes that all patients have the same anatomy and that their veins have no thrombosis. An ultrasound probe can be used to aid in the placement of the central venous line. The use of an ultrasound probe greatly decreases the risk of complications including pneumothorax, arterial puncture, hemothorax, hematoma, and nerve injury.
 - h. These complications are the most significant in patients with coagulopathies, mechanical ventilation, poor pulmonary function, chronic intravenous use, and soft tissue edema, all of which are usually present in a burn patient.
 - i. If a central venous line is not possible, then a small-bore peripheral vein can be dilated to a larger gauge using specially designed kits.
 - j. In children, if intravenous access can not be achieved in a timely manner, an intraosseous infusion can be used to deliver drugs, fluids, and blood with a low incidence of complications until an intravenous line can be placed.
 - k. Intravenous lines can be safely placed through a burn site if the usual sterile technique is used.
 - l. It is current practice at most institutions to change the central venous line about every three days to a new location, not over a guide wire.
 - m. Intravenous lines should always be connected to high-efficiency rapid infusion fluid warmers in the operating room to help in preventing significant heat loss.
2. Treatment of CO poisoning
 - a. The treatment consists of the administration of 100% oxygen using a face mask or endotracheal tube.
 - b. The high concentrations of oxygen accelerate the dissociation of carboxyhemoglobin by 50% every 30

minutes. This decreases the half-life of carboxyhemoglobin by nearly a factor of four compared to the half-life when breathing room air.

c. Although rarely clinically practical, some hospitals have access to hyperbaric oxygen therapy, which is thought to accelerate the dissociation more quickly. Hyperbaric oxygen appears to be the most useful in patients who are comatose with carboxyhemoglobin levels greater than 30%. Hyperbaric oxygen treatment is not recommended in patients with greater than 40% total body surface area (TBSA) burns if it will delay fluid resuscitation.

3. Intubation

Our sample patient requires immediate intubation. He has signs of inhalation injury and a greater than 60% TBSA burn. He may be awake and talking now, but it is very likely that his airway edema will rapidly progress and he will become a near impossible intubation, especially once his fluid resuscitation begins. Also, despite his pulse ox reading of 99%, he has a high likelihood of CO poisoning and therefore a significantly worse oxygenation status than the current picture presents. Get a rapid history and secure his airway. Send labs, including an arterial blood gas (ABG). Also, obtain a chest X-ray (CXR). Establish good IV access and start the fluid resuscitation.

Intra-operative

1. Monitors and Foley catheter
 - a. Monitoring for a major burn excision and grafting should be based on the knowledge of the patient's medical condition and the extent of the surgery.
 - b. **Vital signs and urine output measurement are considered the standard of care in the resuscitation assessment of the burn patient.**
 - c. Invasive monitoring is reserved for the selected high-risk patient and the patient whose resuscitation is failing.
 - d. Electrocardiogram monitoring may be done from burned surfaces using needle electrodes or surgical staples to which an alligator clamp is attached.
 - e. Standard electrocardiogram pads may be placed under a dependent part of the body to provide a satisfactory electrocardiographic signal in most patients.
 - f. Pulse oximetry has been the standard of care in all patients undergoing anesthesia since the early 1990s. They usually give reliable readings of the blood oxygen saturation, but there are times when significant limitations on the accuracy and availability of pulse oximetry occur. When peripheral perfusion is poor (as in states of hypovolemia, hypothermia, vasoconstriction, low cardiac output, and low mean arterial pressure), oxygenation readings become extremely unreliable or cease. Sites for pulse oximeter readings are frequently difficult to find in the burn patient. Standard sites such as fingers and toes may be affected by the burn or unusable due to tourniquet placement. Alternative sites (ear, nose, tongue) can be used with a standard pulse oximeter probe if the fingers are too severely burned to be used. Esophageal reflectance pulse

77. Burn Anesthesia

oximetry may offer advantages over the standard transmission oximetry if the skin sites for monitoring are limited. Ultimately, the physician may have to rely on intermittent arterial blood samples and blood gas analysis.

g. Arterial blood pressure should be monitored invasively during any large surgical debridement. Indications for an arterial line include

- i.** Frequent arterial blood sampling
- ii.** Continuous real-time monitoring of blood pressures
- iii.** Failure to take indirect blood pressure measurements
- iv.** Intentional pharmacologic cardiovascular manipulation
- v.** Assessment of supplementary diagnostic clues

h. Invasive hemodynamic monitoring is recommended in patients with serious burns who do not respond as expected to fluid resuscitation. This suggests that even if patients have normal vital signs, they can actually be hypovolemic.

i. Urine output and arterial blood pressure cannot be considered sufficient as criteria for fluid resuscitation in patients with pre-existing cardiopulmonary or renal disorders and may even provide inaccurate information.

j. Pulmonary artery catheterization may be indicated in patients who do not respond to fluid resuscitation or who have pre-existing cardiac disease. The pulmonary artery catheter is used to assess the cardiac output, stroke volume, systemic vascular resistance, and calculation of oxygen transport parameters.

k. Cardiac output is regarded as one of the most important hemodynamic variables for the assessment of intravascular volume status and the guidance of fluid resuscitation. Central venous pressure and pulmonary capillary wedge pressure have been used as pre-load indicators to guide volume therapy. It is often required that the central venous catheter be placed through burned tissue. The catheters should be removed as soon as possible after the excision and grafting procedure to minimize the risk of local and systemic infection.

l. The indication for extended hemodynamic monitoring is frequently present with a total body surface area burn greater than 50%.

m. Precordial and transesophageal echocardiography can be used to evaluate ventricular function and reliably estimate pulmonary artery capacitance, since pulmonary artery catheters are not routinely placed secondary to the increased risk of infection

n. A Foley catheter should be placed in the patient's bladder to evaluate hourly urine output. Even with a severe burn to the genitalia, the catheterization can almost always be performed. The use of silver-impregnated Foley catheters can significantly decrease the rate of urinary tract infection in the burn patient.

2. Thermal regulation

a. Temperature monitoring is essential for any burn patient undergoing anesthesia because hypothermia is common and often difficult to prevent.

b. The burn patient develops a hypermetabolic state in proportion to the severity of the burn injury.

c. In the burn unit and the operating room, the patient's temperature should remain thermo-neutral to avoid further increases in the metabolic rate.

d. Hypothermia can also exacerbate coagulopathy and can be potentially devastating during operative debridement of burns.

e. The burn patient is particularly susceptible to hypothermia due to the evaporative heat loss through the wounds.

f. Temperature regulation can be achieved by having the ambient temperature of the operating room greater than 28°C, and having all topical and intravenous fluids warmed to 38°C.

g. When possible, the non-operative sites should be covered, and a forced air warming device used. If available, over-the-bed warming lamps and hot water pads should be used.

3. Induction and maintenance of anesthesia

a. General anesthesia with the combination of an opioid, volatile anesthetics, and muscle relaxants is the most widely used technique for burn excision and grafting.

b. The induction agents are chosen based on the hemodynamic stability of the patient.

c. Supplemental opioids are important in burn patients secondary to the intense pain they experience. Burn patients usually require large doses of opioids to remain comfortable even in the absence of movement or surgical procedures. Also, because they regularly receive opioids as a part of their daily care, they become tolerant to these drugs.

d. The choice of volatile anesthetic does not appear to influence the outcome of the anesthesia for burn surgery. Sevoflurane has the advantage of being an ideal agent for inhaled induction of anesthesia in burn patients with abnormal airways and it is less irritating to the already damaged airways. Isoflurane decreases cardiac output and oxygen consumption; however, the reductions parallel one another so that the oxygen supply to the tissue remains sufficient to meet metabolic demands.

e. Various intravenous agents have been used successfully in burn patients. Ketamine offers the advantage of stable hemodynamics and analgesia. It is beneficial for dressing changes and bedside procedures. Its drawbacks include the tendency for dysphoric reactions, which can be decreased with co-administration of benzodiazepines, and the tolerance that develops with repeated use. In a hemodynamically unstable patient, etomidate is a reasonable alternative to ketamine for the induction of anesthesia. Etomidate should not be used for frequent dressing changes due to the possible adrenocortical suppressive effects of the drug. In patients who are adequately resuscitated and not septic, thiopental or propofol may be used.

f. Depolarizing muscle relaxants, including succinylcholine, have led to considerable debate concerning the

timing and use in burn patients due to the potential for hyperkalemia and cardiac arrest. Burn injury results in denervation of the tissue, which causes the entire skeletal muscle membrane, as opposed to the motor endplate only, to develop acetylcholine receptors. Upon administration of succinylcholine, an exaggerated number of acetylcholine receptors are depolarized, resulting in a massive efflux of potassium from the cell into the extracellular fluid. The hyperkalemia can not be prevented by giving a defasciculating dose of nondepolarizer prior to the administration of the succinylcholine. The larger the total body surface area (TBSA) of the burn, the higher is the likelihood of a hyperkalemic response. The potassium concentration increases within the first minute after succinylcholine administration, peaks within 5 minutes, and starts to decline by 10–15 minutes. The hypersensitivity to the succinylcholine begins 48 hours after the burn, and peaks at one to three weeks. The hyperkalemic response may persist for up to two years. Therefore, succinylcholine is safe for the first 48 hours, and is best avoided after that.

g. Nondepolarizing muscle relaxants (NDMR) are often used during excision and grafting of burn patients. Patients with thermal injury are usually hyposensitive or resistant to the action of NDMRs. This effect may take up to a week to develop and may be observed for as long as 18 months after the burn has healed. A marked resistance to NDMRs occurs when the burn is greater than 30% to 40% TBSA. Therefore, the burned patient will require larger than normal doses of NDMR to achieve a desired effect and the duration of action will be shorter than normal. Dose requirements can be increased by up to 250–500%. If muscle relaxants are being used, then neuromuscular function should be regularly monitored in the patient.

4. Estimated blood loss and fluid resuscitation during excision and grafting

- a.** Excisional treatment of burn wounds is usually associated with a large operative blood loss.
- b.** Aggressive fluid resuscitation is imperative to improving mortality, especially in the initial phase of treatment and during operative excision and grafting.
- c.** The volume of the fluid given during resuscitation may not be as important as the timeliness in which it is given.
- d.** Blood loss is usually replaced with crystalloids, colloids, packed red blood cells, and fresh frozen plasma.

e. Hemodynamic changes after a burn are significant and must be managed carefully to optimize intravascular volume, maintain end-organ perfusion, and maximize the oxygen delivery to the tissues.

f. The initial goal of the cardiovascular resuscitation during excision and grafting is to correct or prevent hypovolemia.

g. Intravenous fluid is usually given in proportion to the percentage of the TBSA burned and is guided by the clinical assessment, vital signs, and urine output. In a patient with normal renal function, the urine output should be at least 0.5 mL/kg/hour in adults and 1 mL/kg/hour in children.

h. Lactated Ringer's solution is usually the fluid of choice, except in patients younger than two years old; they should receive 5% dextrose Ringer's lactate.

i. Changes in the intravenous fluid should be made on a regular basis, at least hourly, based on the patient's hemodynamic response.

j. Failure to achieve this goal leads to burn shock with progressive oxygen debt, anaerobic metabolism, and lactic acidosis.

k. During the active fluid resuscitation in the operating room, frequent arterial blood samples should be sent to follow the changing levels of base excess and lactate levels.

Post-operative

1. It is important to remember that the burn patient does not only have initial airway concerns but may have a compromised airway for life.
2. These patients often require frequent reconstructive surgeries long after the initial insult. Patients with healed burns of the neck, face, and chest may develop scar contractures that make direct laryngoscopy difficult. These patients also have a high incidence of laryngeal and tracheal strictures, and bronchial stenosis due to the inhalation injury and prolonged intubation.

BIBLIOGRAPHY

1. Smith, C. *Trauma Anesthesia*. New York: Cambridge University Press, 2008, pp 322–42 (Chapter 21, Burn Anesthesia; Lovich-Sapola JA).

78. Liver^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 42-year-old male with post-necrotic cirrhosis requires a liver transplant. What physiologic derangements do you expect? What tests would you require pre-operatively? What is your anesthetic of choice? What problems are you specifically concerned about in the anhepatic and reperfusion phases?

CLINICAL ISSUES

Organ Matching

1. The ABO blood group is the primary immunologic barrier to transplantation.
2. Different classifications include
 - a. Identical (ex.: A to A)
 - b. Compatible (ex.: O to A)
 - c. Incompatible (ex.: A to O)
3. Identical matching is preferred because patient and graft survival is the best.
4. In some situations, compatible and even incompatible organs may be transplanted.

Indications for Liver Transplant

1. Chronic hepatitis C: 40% of all liver transplants
2. Acute viral hepatitis
3. Noncholestatic
 - a. Alcohol: Laennec's cirrhosis
 - b. Hepatitis A, B, and C
4. Cholestatic
 - a. Primary biliary cirrhosis
 - b. Primary sclerosing cholangitis
5. Metabolic disease
 - a. Wilson's disease
6. Selected cases of hepatocellular carcinoma often associated with hepatitis B or C
7. Drug-induced fulminant hepatic failure
 - a. Acetaminophen overdose
 - b. Idiosyncratic
 - c. Poisonous mushrooms or herbs
8. Other rare liver diseases
 - a. α_1 -antitrypsin deficiency
 - b. Cystic fibrosis
 - c. Budd-Chiari syndrome
 - d. Amyloidosis

Classification System for Liver Transplant

1. Model for End-Stage Liver Disease (MELD)
 - a. A mathematical formula based on the patient's serum creatinine, bilirubin, and INR
 - b. Score ranges from 6 to 40
 - c. The higher the score, the higher patients are ranked on the transplant waiting list.

Contraindications to a Liver Transplant

1. Significant coronary artery disease (CAD)
2. Cardiac dysfunction
3. Moderate to severe pulmonary hypertension: mean pulmonary artery pressure >50 mm Hg
4. Uncontrolled infection or sepsis
5. Advanced malignant hepatic disease
6. Metastatic disease
7. Uncontrolled and markedly elevated intracerebral pressure in the setting of fulminant hepatic failure
8. Active alcohol abuse or illicit drug abuse in the previous 6 months
9. Lack of good social support
10. AIDS (not HIV)

Pathophysiology of End-Stage Liver Disease

1. Virtually all organ systems can be affected.
2. Portal hypertension results secondary to increased hepatic resistance to flow as a result of cirrhotic changes in the liver and a hyperdynamic state secondary to systemic vasodilation and volume expansion
3. Ascites, pleural effusion, and edema
4. Abnormalities in electrolyte levels
 - a. Hyponatremia
 - i. Treatment: stop diuretics and free water restriction.
 - ii. Liver transplant should be postponed for a sodium level less than 120 mEq/L due to the risk of central pontine myelinolysis.
 - b. Hypokalemia
 - c. Hyperkalemia
 - i. It is important to correct prior to the liver transplant.
 - d. Hypomagnesemia
 - i. Common with cirrhosis
5. Increased intracranial pressure (ICP) secondary to cerebral edema

6. Hepatic encephalopathy with symptoms ranging from confusion to coma
7. Significant cardiovascular changes occur, including some or all of the following:
 - a. A hyperdynamic state
 - b. Low systemic vascular resistance
 - c. Elevated heart rate
 - d. Normal or slightly decreased blood pressure
 - e. Supernormal cardiac output
 - f. Some patients develop an associated cirrhotic, alcoholic, or infiltrative cardiomyopathy that is associated with depressed cardiac function.
8. Hepatopulmonary syndrome
 - a. Triad of liver disease, arterial hypoxemia, and intrapulmonary vascular dilatations
 - b. Decreased oxygenation ($\text{PaO}_2 < 70$ mm Hg or $\text{PAO}_2 - \text{PaO}_2$ gradient > 20 mm Hg on room air) associated with intrapulmonary vascular dilatation
 - c. A hallmark is intrapulmonary shunting.
 - d. Patients may have changes in oxygen saturation with a change in position from supine to standing.
 - e. This often resolves spontaneously over the months after the liver transplant.
9. Portopulmonary syndrome
 - a. An uncommon complication of end-stage liver failure that may worsen after the liver transplant.
 - b. This syndrome may progress rapidly and has a very high morbidity and mortality rate.
 - c. Patients are considered to have portopulmonary hypertension if they meet 3 criteria.
 - i. Mean pulmonary artery pressure > 25 mm Hg
 - ii. Pulmonary vascular resistance (PVR) > 120 dyn·sec·cm⁻⁵
 - iii. Pulmonary capillary wedge pressure (PCWP) higher than 15 mm Hg in the setting of portal hypertension
10. Renal dysfunction occurs in a significant number of patients and is usually associated with primary renal disease, acute tubular necrosis, or hepatorenal syndrome.
11. Hepatorenal syndrome
 - a. Develops in the setting of severe liver disease and in the absence of other causes such as drug toxicity, hypovolemia, and underlying intrinsic disease.
 - b. There are two types of hepatorenal syndrome.
 - i. Type I will develop rapidly over a few weeks and is progressive with a high mortality rate. This type can resolve spontaneously with a liver transplant.
 - ii. Type II follows a less acute course and is mainly seen in patients that are resistant to diuretic therapy.
12. Gastrointestinal bleeding from varices and delayed gastric emptying
13. Coagulation defects
 - a. The liver is the primary site of synthesis of enzymes involved in the coagulation cascade, except factor VIII and von Willebrand's factor.
 - b. These often include a decrease in the levels of factors II, V, VII, IX, X, protein C, protein S, and antithrombin III.
 - c. This usually manifests with a prolonged prothrombin time (PT) and INR.
 - d. The patients also often have thrombocytopenia, qualitative platelet dysfunction, and disorders of the fibrinolytic system.
 - e. While the patients with end-stage liver disease are at an increased risk of bleeding, they are also at an increased risk for thrombotic complications such as portal vein thrombosis.
 - f. Treatment
 - i. Pre-operative vitamin K
 - ii. Patient with sites that are actively bleeding should receive fresh frozen plasma (FFP), packed red blood cells (PRBC), cryoprecipitate, and platelets as dictated by the patient's clinical assessment and labs.
 - iii. Desmopressin (DDAVP) may be considered for patients with renal disease and uremic bleeding.
 - iv. A curative transplant should not be postponed to correct coagulation abnormalities.
 - v. FFP and platelets are often transfused prior to central and arterial line placement if the patient has significant coagulation defects.
14. Hypoalbuminemia
 - a. Low levels of serum albumin contribute to ascites formation.
 - b. Opioids and barbiturates bind to albumin, and therefore a low albumin level can lead to an increase in the free fraction of the drug and a theoretically exaggerated clinical effect.

TIPS Procedure

1. Transjugular intrahepatic portosystemic shunt
2. The shunt provides a portosystemic communication for the treatment of portal hypertension.
3. Decreases the risk of recurrent variceal bleeding.
4. Helps with the treatment of refractory ascites.



KO TREATMENT PLAN

Pre-operative

1. Candidates for a liver transplant have undergone a rigorous pre-operative evaluation from multiple medical specialists over weeks to months.
2. All cases are considered an EMERGENCY with the exception of living donors.
3. Your history and physical should concentrate on any changes that have occurred since the most recent evaluation.
4. Review any available echocardiogram, dobutamine stress echocardiography, cardiac positron emission tomography (PET) scan, cardiac catheterization, pulmonary function tests, and renal function tests.
5. Diagnosed pulmonary hypertension should be treated prior to the transplantation.

78. Liver

- a. This usually requires the placement of a pulmonary artery catheter (PAC) for measurements before surgical incision.
 - b. If the patient has a recent cardiac catheterization that shows no pulmonary hypertension, a PAC may not be necessary for the surgery.
6. Aortic stenosis should be treated with a valvuloplasty before the liver transplant. After the graft is stable, an aortic valve replacement can be considered.
7. A head CT should be ordered for altered mental status to evaluate for intracranial bleed, herniation, and/or the extent of cerebral edema.
8. Order pre-operative labs including hematocrit, electrolytes, bilirubin, albumin, a coagulation panel (PT, partial thromboplastin time (PTT), INR, and fibrinogen), and a blood type and cross.
9. Pre-medications
 - i. Avoid central nervous system depressants in a patient with obvious encephalopathy.
 - ii. Midazolam and fentanyl can be safely titrated to effect in patients without encephalopathy.
 - iii. These patients are considered as “full stomach”; therefore, ranitidine can be considered.

Intra-operative

1. The blood bank should be notified and blood products should be immediately available.
 - a. Red blood cells (10–20 units)
 - b. FFP (5–10 units)
 - c. Single-donor platelets (4–10 units)
 - d. Cryoprecipitate (10 units)
2. Induction can be done with a single IV and all additional lines placed post-induction.
3. Epidural catheters are discouraged secondary to peri-operative coagulopathy.
4. Induction can be accomplished with propofol, thiopental, or etomidate, plus opioids and short- or intermediate-acting neuromuscular blockers.
 - a. Cisatracurium or atracurium are reasonable choices of muscle relaxants for transplantation secondary to their extra-hepatic metabolism. But all forms of muscle relaxants have been used successfully, in part due to the fact that a new, functioning liver will be transplanted.
 - b. A rapid-sequence or modified-rapid-sequence induction is often warranted secondary to significant ascites and/or the associated delayed gastric emptying.
5. An orogastric/nasogastric tube should be placed to decompress the stomach and improve surgical exposure.
 - a. This is not without risk.
 - b. Significant bleeding may occur secondary to the associated coagulopathy or esophageal varices.
 - c. A Minnesota or Sengstaken-Blakemore tube should be available for placement if significant esophageal bleeding occurs.
6. Post-induction hypotension may occur secondary to the patient’s very low systemic vascular resistance and relative hypovolemia. This can be treated with a vasoconstrictor, such as phenylephrine.
7. Maintenance anesthesia can be safely implemented with a balanced technique using a mixture of a volatile anesthetic, oxygen, air, and opioids.
 - a. All volatile anesthetics are appropriate except for halothane, secondary to its associated hepatitis risk.
 - b. Total intravenous anesthesia with propofol, opioids, and benzodiazepines can also be used.
 - c. Nitrous oxide should be avoided due to the risk of bowel distension and intravenous air embolism.
8. Hemodynamic monitoring should include a radial arterial line, 2–3 large-bore IV catheters, a rapid transfusion catheter, and central venous pressure monitor (CVP).
 - a. In a patient with known pulmonary hypertension, a pre-induction or pre-incision PAC should be placed and measured.
 - b. Newly diagnosed moderate to severe pulmonary hypertension should be considered as a reason to abort the procedure.
 - c. A PAC may also be useful in patients with renal insufficiency, respiratory compromise, or unstable hemodynamics prior to surgery.
 - d. Continuous transesophageal echocardiography can be used in place of a PAC. Again, be prepared for possible esophageal bleeding due to varices.
9. Pre-incision antibiotics and immunosuppressive agents should be verified with the surgical team.
10. Maintain normothermia using fluid warmers, heated gel pads, and/or a forced-air heating blanket. Normothermia is essential for optimal homeostasis.
11. Frequent measurements of arterial blood gases, blood glucose, electrolytes, and hematocrit are routine for most anesthesiologists.
 - a. Also, consider monitoring the coagulation panels (PT, PTT, INR, and fibrinogen).
 - b. A baseline set of labs should be sent post-induction.
12. Blood loss and coagulopathy are treated with the transfusion of red blood cells, FFP, platelets, and cryoprecipitate.
13. Every patient must have a Foley bladder catheter to follow the patient’s volume status.
14. Ascites will be drained. Prepare for the hemodynamic effects related to drainage of large volumes of ascites, including hypotension.

Dissection Phase

1. The surgical goal of this phase is to mobilize the vascular structures around the liver and isolate the common bile duct. Manipulation of the liver can impede venous return and result in hypotension. Also, acute decompression of ascites during this phase can also result in hypotension.
2. Blood loss may be very high during this phase.
3. Adequate fluid replacement is important. Crystalloids and colloids can be used.

4. Diuresis should be established early using loop diuretics, dopamine, mannitol, and fenoldopam.
5. It is important to correct any abnormalities in coagulopathy.

Anhepatic Phase

1. The anhepatic stage begins with the excision of the native liver and control of bleeding by clamping the liver off from circulation. The ice-cold donor liver is placed on the surgical field.
2. The anhepatic phase is associated with many physiologic disturbances.
 - a. Profound acidosis
 - b. Hypocalcemia
 - i. Citrate accumulates in patients that receive citrated blood products.
 - ii. The liver normally metabolizes citrate.
 - c. Hypomagnesemia
 - d. Hyperkalemia
 - i. Treatment
 - (1) Force diuresis
 - (2) β -adrenergic agonists
 - (3) Insulin plus glucose
 - (4) Alkalinization
 - (a) Hyperventilation
 - (b) Sodium bicarbonate
 - (5) Continuous venovenous hemofiltration (CVVH)
 - e. Hyper- or hypoglycemia
 - i. The liver is a major regulator of blood glucose.
 - ii. No gluconeogenesis occurs during the anhepatic phase.
3. Fluid management can be challenging. Avoid overly aggressive fluid management while treating the blood pressure.
 - a. Risk of fluid overload and resulting cardiopulmonary compromise
 - b. Liver/intestinal swelling
4. The liver is implanted using one of multiple techniques.
 - a. Traditional technique: complete vascular occlusion is established by clamping the hepatic artery, portal vein, infrahepatic vena cava, and suprahepatic vena cava. Venous return will fall by 50–60% with this technique. Cardiac pre-load becomes dependent on collateral flow and severe hypotension can occur. This is associated with a decrease in cardiac output and increased heart rate. Venovenous bypass (VVBP) may be used to increase the venous return, improve the systemic hemodynamics, increase renal perfusion pressure, and decompress portal pressures for a better surgical field. If VVBP is not used, volume loading with a target CVP between 10 and 20 mm Hg plus small doses of vasopressors will be needed before the infrahepatic and suprahepatic caval clamps are placed.
 - i. Venovenous bypass complications include arm lymphedema, air embolism, and vascular injury.
 - ii. VVBP has limited benefits when the bypass time is short.

- b. Piggyback technique: the vena cava is partially or entirely occluded during the anhepatic phase. Hemodynamics are improved with the partial occlusion, but this is surgically more difficult. Venovenous bypass is not needed with this technique.

Reperfusion Phase

1. Vascular clamps are removed from the liver graft.
2. This phase is associated with significant hemodynamic instability and cardiac arrest.
3. Prior to the clamp removal the patient should be on 100% oxygen, have an adequate CVP, and have a hematocrit in the mid-30s.
 - a. All emergency drugs and blood products should be available.
 - b. Consider giving
 - i. 50 mEq of IV HCO_3^- to treat the acid load from the grafts
 - ii. 500 mg IV infusion of CaCl_2 to counteract the effects of elevated potassium on the heart
 - iii. Glucose
4. Reduced cardiac contractility, arrhythmias, severe bradycardia, profound hypotension, and hyperkalemic arrest have been reported.
5. Post-reperfusion syndrome: hemodynamic instability of an unknown mechanism that is associated with high potassium concentrations in the preservative solution and decreased systemic vascular resistance
 - a. Acute systemic hypotension
 - b. Bradycardia
 - c. Elevated pulmonary artery pressures
 - d. Supraventricular or ventricular dysrhythmias
6. The hepatic artery anastomosis is completed.
7. Watch for coagulopathies, including dilution/consumption of clotting factors, platelet entrapment, endogenous heparinoid-like substances, and primary fibrinolysis.
8. An early indication of graft function is the lack of calcium requirement even when FFP is being infused.
9. Within the first hour, the cardiac output decreases and the systemic vascular resistance increases. These are early signs of good graft function.

Post-operative

1. Early post-operative extubation can be done if the patient meets the usual extubation criteria.
2. Nearly all patients will require an intensive care unit (ICU) admission regardless of whether they remain intubated or not.
3. Post-operative pain control is usually not a problem. Analgesic requirements are usually decreased when compared to other abdominal surgeries.
4. The recovery period requires frequent evaluations of the cardiac and pulmonary function, electrolytes, renal and liver function, and coagulation.

5. FFP is occasionally needed post-operatively to offset an initially “sluggish” liver.
6. An insulin infusion may be required secondary to peri-operative steroids and post-operative liver function.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2245–53.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1364–7.
3. Yao FSE, Fontes ML, Malhotra V. *Anesthesiology: Problem Oriented Patient Management*, 6th ed. Philadelphia: Lippincott Williams and Wilkins, 2008, pp 486–513.

79. Renal^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 47-year-old male is scheduled for a cadaveric kidney transplant. He is due for hemodialysis today. His last hemodialysis was 2 days ago. An EKG, chest X-ray, and labs are sent. He is complaining of chest pressure and tightness. He last ate 8 hours ago. What are your primary anesthetic concerns? Does it matter when his last dialysis was? His potassium (K^+) is 6.1 mEq/L. Does this change your anesthetic? The EKG and chest X-ray show left ventricular hypertrophy. Does that affect your management? Do you consider the patient to be NPO since he last ate 8 hours ago? Assuming that you proceed with the surgery, what monitors will you use? Would you use general or regional anesthetic? What is your plan for the maintenance of anesthesia?

CLINICAL ISSUES

Increased Anesthetic Risks

1. End-stage renal disease (ESRD) can cause multiple organ system dysfunctions.
2. Patients have a less predictable response to the anesthetic drugs and techniques.
3. They are at higher risk for cardiac and other peri-operative complications secondary to their underlying disease.
4. Cardiovascular complications are the leading cause of death after renal transplantation.

Organ Matching and Allocation

1. Cadaveric and living donors
2. Initial testing is for ABO compatibility.
3. Second line of testing determines the HLA profile of the recipient.

4. Final test is a cross match that is performed by mixing the recipient blood with the donor blood cells to determine the presence of pre-formed reactive antibodies against the donor antigens.

Indication

1. ESRD caused by glomerular disease
2. ESRD caused by diabetes mellitus
3. ESRD caused by hypertensive kidney disease
4. Polycystic kidney disease
5. Familial or congenital diseases of the kidney
6. Tubulointerstitial disease

Pathophysiology of ESRD

1. Once urine output falls below 400 mL/day, the patient becomes oliguric. This results in abnormalities in Na^+ , K^+ , Ca^{2+} , Mg^{2+} , and phosphate levels.
2. Cardiovascular diseases including acute myocardial infarct, cardiac arrest, cardiac arrhythmias, pericardial disease, and cardiomyopathy are associated pathologies. They account for over 50% of the deaths of patients on dialysis.
3. Dilated cardiomyopathy and concentric hypertrophy develop as a response to increases in intravascular volume and afterload. This resulting fluid overload and congestive heart failure is a result of the patient’s inability to excrete the daily fluid intake.
4. The accumulation of uremic toxins and metabolic acids contributes to the poor myocardial performance.
5. The resulting hypervolemia due to oliguria leads to hypertension that damages the kidneys and thereby creates a condition for escalating hypertension.

6. Renal failure accelerates the progression of atherosclerosis.
7. Diabetes mellitus (DM) is the cause of ESRD in nearly 30–40% of cases. Patients with DM and ESRD have a higher cardiovascular risk than patients with uremia alone.
8. Chronic uremia causes delayed gastric emptying. All patients presenting for a kidney transplant should be considered to have a full stomach. Rapid sequence induction should be considered.
9. Normochromic, normocytic anemia can occur secondary to the decreased erythropoiesis and retained toxins.
10. Qualitative platelet defect
11. Central nervous system disturbances may occur, including myoclonus, seizure, stupor, and coma.

Exclusion Criteria for Renal Transplants

1. Severe heart, lung, or liver disease
2. Most malignancies
3. Active or untreatable infections such as tuberculosis



KO TREATMENT PLAN

Pre-operative

1. In regard to the sample case, chest pain has a 65% sensitivity/specificity for coronary artery disease.
2. This patient requires a complete cardiac workup.
3. Kidney transplants are not emergent.
4. Well-tolerated, prolonged cold preservation of the cadaveric kidney provides enough time (up to 36 hours) for an adequate pre-operative exam.
5. The patient also should be dialyzed to allow normalization of electrolyte imbalances and volume status before surgery.

General Considerations for Evaluation during the Waiting List Period

1. Order pre-operative pulmonary function tests.
2. Screen the patient for any existing tumors or infection including dental evaluation, colonoscopy, mammography, Pap test, and viral serologies.
3. The patient should have good control of his diabetes.
4. Psychiatric evaluation

Considerations in the Immediate Pre-operative Period

1. Estimate volume status by comparing the patient's current weight and his "dry weight."
2. Order a basic metabolic panel. Potassium levels greater than 6.0 mEq/L require a delay in surgery and correction of the level with hemodialysis.
3. Pre-operative cardiac evaluation is extremely important.
 - a. Pre-operative EKG/echocardiogram may be enough for a young patient with newly diagnosed ESRD unrelated to DM.

b. Stress echocardiogram or cardiac catheterization may be indicated for a patient with long-standing ESRD associated with DM.

4. Peri-operative β -blockade should be started.
5. Evaluate the coagulation status with a prothrombin time (PT), INR, partial thromboplastin time (PTT), fibrinogen, and platelet count.
6. Order a hematocrit level.
7. Type and screen the patient for blood products although patients rarely require a blood transfusion due to the associated minimal blood loss.
8. Verify immunosuppressant pre-induction orders with the nephrologist/surgeon prior to induction.

Intra-operative

1. Apply standard ASA monitors.
2. Renal transplants are usually done under general anesthesia but have been successfully done using epidural anesthesia. Regional anesthesia is usually avoided secondary to the concerns over uremic platelet dysfunction and residual heparin from the pre-operative dialysis.
3. Goals for induction and intubation should include careful control of heart rate and blood pressure to minimize the possibility of myocardial ischemia. This can be achieved by blunting the response to direct laryngoscopy with opioids such as fentanyl or remifentanyl, or using a short-acting β -blocker such as esmolol.
4. Pre-treat with a non-particulate antacid.
5. Rapid sequence induction should be performed secondary to a presumed full stomach unless the patient has a known difficult airway. Succinylcholine is not contraindicated in patients with ESRD, assuming that they have been recently dialyzed and their potassium level has been corrected. The increase in serum potassium with a bolus of succinylcholine is about 0.5 mEq/L for patients with or without ESRD.
6. General endotracheal anesthesia provides stable hemodynamics, muscle relaxation, and a predictable depth of anesthesia. Volatile anesthetics plus opioids or total intravenous anesthesia with opioids and propofol are both appropriate. Desflurane, isoflurane, and sevoflurane are all acceptable choices of volatile anesthetics. Secondary to the theoretical risk of renal toxicity with sevoflurane, it is often not the first choice of anesthetic and requires at least 4 L/min of fresh gas flow rates.
7. Atracurium and cisatracurium are the preferred muscle relaxants secondary to their spontaneous Hoffman degradation and plasma cholinesterase metabolism. Pancuronium, gallamine, and curare are not recommended because they depend on the kidney for elimination and therefore have a prolonged duration of action in ESRD patients. Patients with ESRD also have been shown to have an increased sensitivity and prolonged duration of action with vecuronium; therefore, it is often not recommended.
8. Arterial line and central venous pressure (CVP) monitoring are only necessary for patients with more advanced

co-morbid conditions. The goal CVP value is in the range of 10 to 15 mm Hg to optimize cardiac output and renal blood flow.

9. Severe co-morbid conditions such as symptomatic coronary artery disease (CAD) or congestive heart failure should be monitored for the development of ischemia with a pulmonary artery catheter or transesophageal echocardiography.

10. Always monitor the hemodialysis shunt or fistula during positioning and intra-operatively for the presence of a thrill, and document the patency. Avoid blood pressure cuffs and tourniquets over the shunt or fistula.

11. Antibiotics should be given prior to surgical incision.

12. Hypotension may occur with unclamping of the iliac vessels and reperfusion of the graft. Every effort should be made to avoid hypotension secondary to the renal graft function being so dependent on adequate perfusion. Vasoconstrictors with strong α -adrenergic effects, such as phenylephrine, should be drugs of last resort. The typical goal is to maintain the systolic blood pressure greater than 90 mm Hg and the mean systemic pressures greater than 60 mm Hg.

13. Once the anastomosis of the kidney is begun by the surgeon, a diuresis should be initiated with mannitol and furosemide. Heparin and verapamil should be available in the operating room. The anesthesiologist may also be asked to give the first dose of the immunosuppressant. The kidney graft is not good at concentrating the urine and reabsorbing sodium initially; therefore, you should pay special attention to the patient's electrolytes.

14. Monitoring the urine output is very important. Immediate urine output is seen in over 90% of living donor kidney transplants and between 40% and 70% of cadaveric transplants. Near the end of the procedure, a decrease in urine output strongly suggests mechanical impingement of the graft, vessel, or ureter. Intra-operative ultrasound can be used to examine the flow in the arterial and venous anastomoses. Intra-operative urine output is frequently enhanced intra-operatively by infusions of mannitol, loop diuretics, and occasionally dopamine.

15. Diabetic patients should have frequent blood glucose checks in the effort to maintain their blood glucose in a range from 80 to 110 mg/dL.

16. Transfusion is rarely required, but if the patient is cytomegalovirus (CMV) negative and is receiving a CMV negative kidney, then he or she should receive CMV negative blood.

Post-operative

1. Pain control

a. Post-operative pain is usually mild to moderate.

b. Post-operative pain control can be managed with fentanyl, sufentanil, alfentanil, and remifentanyl.

c. Caution should be used when administering morphine, meperidine, or oxycodone because they or one of their active metabolites is dependent on renal excretion and therefore may accumulate.

d. Patient-controlled analgesia (PCA) is usually a good choice for post-operative pain management.

e. Non-steroidal anti-inflammatory agents (NSAIDs) are contraindicated because of the resulting vasoconstriction and decreased blood flow to the kidneys.

2. The plan should be for full reversal of the anesthetic and extubation if possible. Renal transplant patients rarely require an intensive care unit (ICU) stay.

3. Monitor urine output closely. Re-exploration should not be delayed if kinking of the vascular attachments or obstruction of the ureter is suspected.

4. Common post-operative complications

a. Ureteral obstruction

b. Ureteral fistula

c. Vascular thrombosis

d. Lymphocele

e. Wound complications

f. Bowel perforation

g. Femoral neuropathy

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2237-42.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1362-4.

80. Delayed Emergence, Change in Mental Status, Delirium, and Agitation^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 27-year-old patient is in the post-anesthesia care unit (PACU) following a surgical repair of his left femur. He was found unconscious in the snow at a ski resort. He was awake prior to the induction of anesthesia. He denied any medical or surgical history. His toxicology screen was still pending. You are called to the PACU to see the patient because he is confused and combative. What is your differential diagnosis? How will you treat this?

CLINICAL ISSUES

Incidence

1. More common in patients 65 years or older
2. Increased incidence in hip fractures, total knee replacements, cataracts, and cardiac surgery, especially after cardiopulmonary bypass.
3. 90% of patients regain consciousness within 15 minutes of admission to the PACU.³

Differential Diagnosis

1. Hypoxia and hypercarbia
 - a. Check an arterial blood gas (ABG), end tidal CO₂, and pulse oximetry.
 - b. Rule out CO₂ narcosis.
2. Hypotension
3. Hypothermia
 - a. Less than 33 degrees Celsius can produce profound unconsciousness.^{1,2,3}
 - b. Core temperature below 30°C can cause fixed pupillary dilation, areflexia, and coma.³
4. Residual medications and polypharmacy
 - a. Muscle relaxants (depolarizing and non-depolarizing)
 - b. Volatile anesthetics
 - c. Opioids
 - d. Induction drugs
 - e. Pre-medications including benzodiazepines
 - f. Anticholinergics resulting in central anticholinergic syndrome
 - i. Scopolamine
 - g. Ketamine
 - h. Droperidol
 - i. Steroid psychosis

- j. Cardiac anti-dysrhythmics
- k. Tricyclic anti-depressants
- l. Antihistamines
5. Electrolyte abnormalities
 - a. Diabetic ketoacidosis (DKA)
 - b. Hyper- and hypo-glycemia
 - c. Hyponatremia
 - d. Hypermagnesemia
 - e. Hypercalcemia
 - f. Acidosis/alkalosis
 - g. Hypercalcemia
6. Adverse neurologic outcome
 - a. Cerebral vascular accident (CVA)/cerebral edema/transient ischemic attack (TIA)
 - b. Hepatic/renal encephalopathy
 - c. Postictal
 - d. Anoxia
 - e. Infection
 - f. Increased intracranial pressure (ICP)
 - g. Ruptured cerebral aneurysm
7. Infections
 - a. Meningitis
 - b. Sepsis
 - c. Pneumonia
 - d. Bladder infection
8. Substance abuse
 - a. Alcohol intoxication or withdrawal
 - b. Cocaine
 - c. Heroin
 - d. Over-the-counter sleep aids
 - e. Drug withdrawal
9. Endocrine abnormalities
 - a. Addison's disease
 - b. Cushing's disease
 - c. Hyper-/hypothyroid (thyroid storm)
10. Hepatic abnormalities
 - a. Encephalopathy
 - b. Delayed drug clearance
11. Renal abnormalities
 - a. Delayed drug clearance
 - b. Full bladder
 - c. Uremia
12. Pain and anxiety



KO TREATMENT PLAN

Diagnose and treat the most likely cause. The differential diagnosis and treatment plan are essentially the same for delayed emergence, change in mental status, delirium, and agitation. If during the exam you are presented with any of these conditions, follow the step-wise approach listed below. Always start with the conditions that are most likely for your sample case.

Primary Concerns in this Sample Case

1. Hypothermia secondary to being found unconscious in the snow
2. Head injury from the trauma
3. Intoxication

Post-operative

1. Check the patient's oxygenation and ventilation.
2. Check vital signs.
 - a. Oxygen saturation
 - b. Pulse
 - c. Temperature
 - d. Blood pressure
 - e. Respiratory rate
 - f. Character of spontaneous ventilations
3. Review all medications given.
4. Investigate the patient's pre-operative level of responsiveness.
5. Check a toxicology screen.
6. Check the patient's response to a stimulus.
7. Pharmacologic reversal agents
 - a. Naloxone
 - i. Pure opioid receptor antagonist
 - ii. Give 0.04 mg IV in increments every 2 minutes up to 0.2 mg.
 - iii. Has been found, in high doses, to reverse the effects of alcohol, barbiturates, and benzodiazepines.³
 - iv. No effect on volatile anesthetics

- v. May partially antagonize ketamine and nitrous oxide.³
- vi. May be useful in the treatment of post-anesthetic apnea in infants, even when no opioids have been given.³
- b. Flumazenil
 - i. Benzodiazepine antagonist
 - ii. Give 0.2 mg IV per minute to a total of 1 mg.
- c. Physostigmine
 - i. 1.25 mg IV can reverse the effects of some sedatives and inhaled anesthetics.
 - ii. Treats central anticholinergic syndrome.
- d. Reversal of non-depolarizing muscle relaxants with neostigmine and glycopyrrolate
- e. Consider an unrecognized phase II block from succinylcholine use in a patient with pseudocholinesterase deficiency.
- f. Consider IV local anesthetic toxicity.
8. Check ABG, electrolytes, and blood glucose.
 - a. Rule out CO₂ narcosis.
 - b. Rule out hyponatremia.
 - c. If hypoglycemia is presumed, treat immediately with 50% dextrose IV, even before verifying the glucose value.
9. Perform a neurological check: pupils and CVA history.
10. Head CT scan and neurology consult if the condition is persistent
 - a. CT scan can determine elevated ICP and acute intracranial hemorrhage.
 - b. Consider a grand mal seizure, delirium tremens, and cerebral anoxia.

BIBLIOGRAPHY

1. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 744-5.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 343-5, 420-1, 2483, and 2717-8.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1400-2.
4. Ford MD, Delaney KA, Ling LJ, et al. *Clinical Toxicology*, 1st ed. Philadelphia: WB Saunders Company, 2001, ch. 56.

81. The Bleeding Tonsil^{1,2,3,4}

Kasia Petelenz Rubin, MD

SAMPLE CASE

A 9-year-old boy presents two days after an adenoidectomy and tonsillectomy for recurrent adenotonsillitis. He appears pale and lethargic. He is dizzy when upright, and lies on the bed throughout the entire exam. His mother states that two days ago, the child appeared to have had some bleeding from the mouth, but that it had stopped until 1 hour before presentation. Vital signs are heart rate (HR) 120, respiratory rate (RR) 26, and blood pressure (BP) 75/46 mm Hg. What are your concerns? How will you evaluate the patient's volume status? How will you induce general anesthesia in this patient?

CLINICAL ISSUES

Risk and Incidence of a Tonsillar Re-bleed

1. Post-operative hemorrhage rates vary depending on the surgical method and study cohort but are typically quoted to range between 1% and 6%.

Patient Volume Status

1. The extent of blood loss and dehydration may be difficult to determine.

Intravenous (IV) Access

1. Good IV access must be established.
2. If it is lost, it must be reestablished without delay.
3. In an extremely agitated and hypovolemic child with acute vasoconstriction, it may be difficult to obtain an intravenous line. In this case, intraosseous infusion may be necessary.

Full Stomach Precautions

1. A patient who presents with a bleeding tonsil has a full stomach, filled with swallowed blood.
2. Precautions in this case include
a. Two well-functioning suction devices with large-bore suction tubes
b. Extra laryngoscope handles and blades
c. Several cuffed endotracheal tubes with lubricated stylets in place
d. Availability of an assistant to provide cricoid pressure during the rapid sequence induction

Table 81.1. General Principles for Pediatric Volume Status

The patient is sitting up and talking without dizziness.	1. Mild to moderate hypovolemia 2. There is time to evaluate the volume status with an H/H, urine specific gravity, and physical status exam.
The patient is lying down, pale, and hypotensive.	1. May be on the verge of hypovolemic shock, with the development of lactic acidosis. 2. Begin immediate volume resuscitation without delay for lab values. 3. Check H/H after the resuscitation has begun.
The patient is tachycardic and hypertensive.	1. Outpouring of catecholamines in the pediatric patient results from hemorrhage, hypovolemia, fear, or excitement. 2. Peripheral vasoconstriction delays the clinical onset of severe hypotension in the awake patient.

Abbreviations: H/H = hemoglobin and hematocrit
Adapted from Vergheze ST, Hannallah RS. Pediatric otolaryngologic emergencies. *Anesthesiol Clin North Am*, 2001;19(2):237–56.



KO TREATMENT PLAN

Pre-operative

1. Review the prior anesthetic record of the initial surgery to rule out any anesthetic problems.
2. Note any co-existing medical conditions including loose teeth and the use of medications such as aspirin.
3. Determine the child's volume status by performing a history and physical exam.
4. Anesthesia-induced vasodilation may lead to severe hypotension.
5. Prior to proceeding with the surgery, you must have an IV!!!!
6. Initiate vigorous volume resuscitation with normal saline or lactated Ringer's solution immediately.
7. Blood is rarely used as the primary solution for volume replacement in these children.

Intra-operative

1. Pre-medication is controversial. It is generally advised that no pre-medication be given, but if the child's condition

permits, a small dose of intravenous midazolam may be administered.

2. After application of the standard ASA monitors, pre-oxygenate the child in a lateral, head-down position to allow the blood to drain out of the mouth.
3. After pre-oxygenating the patient, turn the child supine, and administer atropine, 0.02 mg/kg, followed by a rapid sequence induction with cricoid pressure using propofol, etomidate, ketamine, or sodium thiopental (with the doses guided by the patient's hemodynamic status), and succinylcholine with cricoid pressure. Any of these induction drugs are safe as long as you use a properly titrated dose and can justify why you chose that drug.
4. Intubate using a cuffed endotracheal tube to help prevent the blood from entering the trachea around the tube.
5. During the maintenance phase of anesthesia, titrate a volatile anesthetic with nitrous oxide and oxygen, supplemented with an opioid, such as fentanyl, to enable rapid awakening at the conclusion of surgery.
6. Decompress the stomach using a large-bore orogastric tube.
7. Do not extubate the child until he is fully awake and a normal gag and cough reflex has returned, in order to avoid aspiration.

Post-operative

1. After extubation, patient management is guided by the child's clinical condition before and immediately after surgery.
2. A child with a minor bleed may even be sent home immediately following surgery.
3. Other bleeds are typically monitored overnight.
4. Patients who have come to the operating room hypotensive after a severe bleed may need to stay in the intensive care unit in order to adequately manage their hemodynamics.

BIBLIOGRAPHY

1. Verghese ST, Hannallah RS. Pediatric otolaryngologic emergencies. *Anesthesiol Clin North Am*, 2001;19(2):237-56.
2. Motoyama EK, Davis PJ, eds. *Smith's Anesthesia for Infants and Children*, 7th ed. Philadelphia: Mosby Elsevier, 2006, pp 798-9.
3. Miller RD, ed. *Miller's Anesthesia*, 6th ed. Philadelphia: Churchill Livingstone, 2005, p 2544.
4. National Prospective Tonsillectomy Audit: Tonsillectomy technique as a risk factor for postoperative hemorrhage. *Lancet*, 2004;364:697-702.

82. Post-Carotid Bleed^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

You are called to the post-anesthesia care unit (PACU) to evaluate an 88-year-old patient who has just had a right carotid endarterectomy. She is having significant neck swelling over the incision site. On arrival, you note that she is anxious and short of breath. You apply a face mask/ambu bag and have difficulty ventilating. What do you do next? Do you attempt to intubate, or do you open the wound?

CLINICAL ISSUES

Post-carotid Bleed

1. Incidence of 2-5%.
2. Usually results from venous oozing.
3. Rarely, they are rapidly expanding.

Causes of Post-carotid Respiratory Insufficiency

1. Recurrent laryngeal nerve injury
2. Hypoglossal nerve injury

3. Massive hematoma

4. Deficient carotid body function leading to a loss in the ventilatory drive to increase respiration when the patient encounters a drop in PaO₂.



KO TREATMENT PLAN

Post-operative

1. Venous oozing is treated with external digital compression for 5-10 minutes and reversal of any residual heparin with protamine.
2. Rapidly expanding hematomas require prompt bedside evaluation secondary to the associated tracheal compression and loss of a secure airway.
 - a. Aggressive post-operative blood pressure control
 - b. Immediate evacuation at the bedside if the airway is compromised. You may have to do it yourself. Use a #11 scalpel or suture removal scissors to cut the skin and

subcutaneous sutures. Evacuate the hematoma and apply pressure to any visible areas of bleeding.

- c. Call the surgical team.
 - d. Secure the airway.
 - e. Return to the operating room for surgical closure.
 - f. Correct any associated coagulopathies.
3. Closely monitor the patient in an intensive care unit (ICU) setting for 8–12 hours post-operatively.

TKO: Remember that during the oral board exam you could have a very similar scenario with a post-thyroidectomy patient.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, p 2105.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 953–4.

83. Post-Operative Nausea and Vomiting^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 50-year-old female complains of persistent nausea and vomiting 5 hours after the drainage of a subhepatic abscess under general anesthesia. How will you proceed? What is your workup plan? What is your treatment plan?

CLINICAL ISSUES

Differential Diagnosis of the Causes of Nausea

1. Hypoxia
2. Hypotension
3. Pain
4. Anxiety
5. Infection
6. Chemotherapy
7. Gastrointestinal obstruction
8. Narcotics/volatile anesthetics/etomidate
9. Movement
10. Vagal response
11. Pregnancy
12. Increased intracranial pressure (ICP)

Increased Risk of Post-operative Nausea and Vomiting

1. Patient-related factors
 - a. Diabetes
 - b. History of motion sickness or post-operative nausea and vomiting
 - c. Increased level of anxiety
 - d. Current viral illness
 - e. Pancreatic disease
 - f. Non-smokers
 - g. Patients who are menstruating

h. Female gender

i. Pregnancy

j. Dehydration/hypovolemia

k. Hypotension

2. Anesthesia-related factors

a. Gastric distension

b. Opioids

c. Muscle relaxant reversal agents

d. Volatile anesthetics and nitrous oxide

e. Inadequate hydration

3. Surgery-related factors

a. Operative procedure

i. Laparoscopic surgery

ii. Strabismus surgery

iii. Major breast surgery

iv. Ear, nose, and throat surgery

v. Lithotripsy

b. Increased length of surgery time

c. Blood in the gastrointestinal tract

d. Premature ambulation

e. Pain



KO TREATMENT PLAN

Pre-operative

1. The best way to treat post-operative nausea and vomiting is to prevent it in the first place.
2. Metoclopramide (10–20 mg IV or 0.2 mg/kg IV)
 - a. Gastrokinetic drug
 - b. Dopamine antagonist
 - c. Reduces gastric volume.
 - d. Facilitates gastric and small bowel motility, therefore increasing gastric emptying.
 - e. Increases lower esophageal sphincter tone.

83. Post-Operative Nausea and Vomiting

- f. No effect on acidity
- g. Onset 3–5 minutes
- 3. H₂ receptor antagonist and proton pump inhibitors
 - a. Increase gastric pH.
 - b. Decrease gastric volume by reducing gastric acid secretions.
- 4. Non-particulate antacid (sodium citrate or sodium bicarbonate)
 - a. Rapidly increases the gastric fluid pH.
 - b. It does increase the gastric volume.
 - c. Decreases the severity of the aspiration.
 - d. The unpleasant taste may promote vomiting in some patients.
 - e. Effectiveness is lost by 30–60 minutes after the ingestion.
- 5. Transdermal scopolamine patch
 - a. Best for motion sickness
 - b. Must be placed several hours prior to the surgery.
 - c. Side effects include dry mouth, somnolence, mydriasis, and dizziness.

Intra-operative

1. Adequate intravenous (IV) fluids
2. Propofol-based anesthesia
3. Dexamethasone (4–8 mg IV)
4. Limit volatile anesthetics and nitrous oxide.

Post-operative

1. Look at the patient's vital signs. Is the patient hypotensive or hypoxic? Treat accordingly.

2. IV fluids to treat any dehydration
3. Medications
 - a. Ondansetron (4–8 mg IV), granisetron, and dolasetron (most cost-effective)
 - i. Highly selective serotonin (5-HT₃) receptor antagonists
 - ii. Lack sedative, dysphoric, and extrapyramidal side effects.
 - b. Phenothiazines (chlorpromazine and promethazine)
 - i. Block dopamine receptors in the chemoreceptor trigger zone of the brain.
 - ii. Risk of extrapyramidal (indirect motor pathway) side effects and significant sedation
4. Limit patient movement.
5. Order labs to rule out electrolyte abnormalities.
6. Send an arterial blood gas to rule out hypoxia.
7. Obtain an EKG because myocardial ischemia can present with nausea.
8. Call the surgeon; the persistent nausea and vomiting may be related to the patient's subhepatic abscess.

BIBLIOGRAPHY

1. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 208 and 580–1.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2595–601, 2720–1.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1238–40.

84. Malignant Hyperthermia (MH) and Masseter Muscle Rigidity^{1,2,3,4}

Jessica A. Lovich-Sapola, MD

MALIGNANT HYPERTHERMIA

SAMPLE CASE

A 7-year-old male is scheduled for a herniorrhaphy. The child has no prior medical history. Per his parents, he had a previous tonsillectomy with no complications at age 2. He is adopted, so no family history is available. He has a mask induction with sevoflurane. After induction, an intravenous line is placed. Twenty minutes into the case the resident notes some mild tachycardia and a slowly increasing end tidal CO₂. What are you concerned about? What tests would you order? Assuming this is MH, how would you treat it?

CLINICAL ISSUES

Definition

1. MH is a hypermetabolic disorder of skeletal muscle with varied presentations.⁴
2. MH is a pharmaco-genetic disease.
3. The patients have a genetic predisposition for the development of the disease once exposed to the triggering agents or stressful environmental factors.
4. There are multiple associated modes of inheritance, but all have a presumed defect in calcium release channels.
5. Intracellular hypercalcemia in the skeletal muscle activates metabolic pathways that result in adenosine triphosphate (ATP) depletion, acidosis, membrane destruction, and cell death.⁴

Incidence

1. 1 in 40,000 adult anesthesia cases
2. 1 in 12,000 pediatric anesthesia cases

Risk Factors: (Use MH precautions and non-triggering agents)

1. Muscle biopsy diagnosis of MH
2. Family history of MH
3. Central core disease
4. King-Denborough syndrome

5. Duchenne and Becker muscular dystrophy and related muscular dystrophies and myopathies

Possible Associations: (Use MH precautions and non-triggering agents)

1. Sudden infant death syndrome
2. Smith-Lemi-Opitz syndrome
3. Charcot-Marie-Tooth syndrome
4. Heatstroke
5. Mitochondrial cytopathies
6. Burkitt's lymphoma
7. Osteogenesis imperfecta
8. Myotonia congenita
9. Neuroleptic malignant syndrome
10. Myelomeningocele
11. History of heat-, stress-, or exercise-induced rhabdomyolysis

Testing

1. About 70% of susceptible patients have an increased resting plasma creatinine kinase (CK) concentration. Other patients may have a normal CK value; therefore, the test is not definitive.
2. Skeletal muscle biopsies with in vitro contracture testing using halothane and caffeine separately provide definitive confirmation. The combined caffeine-halothane test is associated with a high incidence of false-positive results. The ryanodine contracture test using a skeletal muscle biopsy may be the most specific diagnostic test for MH.

Triggers

1. Succinylcholine and potent inhalational agents
2. Local anesthetics used for spinals, epidurals, and nerve blocks are considered safe.
3. Intravenous propofol, barbiturates, benzodiazepines, and etomidate are safe.
4. Nitrous oxide is safe.

Clinical Features

1. Symptoms may develop within 10 minutes to hours after the beginning of the anesthetic.
2. Some cases of MH can even develop post-operatively.

84. Malignant Hyperthermia (MH)

Early

1. Tachycardia not otherwise explainable is usually the first sign.
2. Irregular heart rate (cardiac dysrhythmias) including ventricular bigeminy, multifocal ventricular premature beats, and ventricular tachycardia
3. Masseter spasm
4. Tachypnea (an early sign in spontaneously breathing patients)
5. Rapid exhaustion of the soda lime
6. Warm soda lime canister
7. Increased minute ventilation
8. End-tidal CO₂ concentrations increase despite proper ventilation and adequate fresh gas flows. This is usually one of the earliest, most sensitive, and specific signs.
9. PaCO₂ increases to 100 to 200 mm Hg.
10. Metabolic and respiratory acidosis (pH 7.14 to 6.80)
11. Hyperkalemia and peaked T waves on the EKG
12. Unstable blood pressure

Intermediate

1. Core body temperature increases at a rate of 1–2 degrees Celsius every five minutes.
2. Sweating and flushing
3. Cyanosis and a decrease in arterial and central venous oxygen saturation
4. Skin mottling
5. Dark blood is found in the surgical site secondary to increased plasma myoglobin.

Late

1. Generalized muscular rigidity (despite the use of a muscle relaxant)
2. Prolonged bleeding
3. Oliguria
4. Myoglobinuria (dark urine)
5. CK concentrations are elevated >1000 IU. The laboratory value often exceeds 20,000 in the first 12–24 hours.
6. Hypercalcemia
7. Hyperphosphatemia
8. Disseminated intra-vascular coagulation (DIC)
9. Pulmonary edema
10. Acute renal failure
11. Central nervous system damage including blindness, seizures, coma, or paralysis
12. The blood gas analysis will show hypercarbia with a respiratory and metabolic acidosis without marked oxygen desaturation.
13. Lactacidemia
(MH is characterized by signs and symptoms of hypermetabolism. If the patient develops any of the above symptoms, send an arterial blood gas, potassium level, and CK level.)

Preparation for a MH Susceptible Patient

1. Ask about personal and family history of MH and any adverse anesthetic reactions, including unexplained fever or death.
 - a. It is important to note, however, that up to 50% of patients who experience an MH episode have had an uneventful anesthetic in the past.
2. Pre-treatment with dantrolene is not recommended secondary to the side effects and its inability to completely prevent MH.
3. Provide a non-triggering anesthetic to any patient with a history of MH susceptibility, including a family history.
4. A fully stocked MH cart should always be immediately available when using any potent volatile anesthetics or succinylcholine. This should include 36 vials of dantrolene, sterile water (without a bacteriostatic agent) to reconstitute the dantrolene, sodium bicarbonate, furosemide, dextrose, calcium chloride, regular insulin (refrigerated), and anti-arrhythmics.
5. Prepare the anesthesia machine.
 - a. Ensure that the vaporizers are disabled by removing or taping them in the off position.
 - b. Change the CO₂ absorbent.
 - c. Set the O₂ flow to 10 L/minute for 20 minutes.
 - d. Attach a clean disposable breathing bag to the Y-piece of the circle system with the ventilator set to inflate the bag periodically.
 - e. Use a new disposable breathing circuit, and wait until the expired gas analyzer indicates the absence of any volatile agent within the circuit.
6. The patient should be sedated during the pre-induction period to reduce the chance of stress-induced MH.

Post-operative Plan for an Uneventful Anesthetic in a MH Susceptible Patient

Assuming an uneventful anesthetic, the patient may be discharged from the post-anesthesia care unit (PACU) if he or she presents with no signs of MH after a minimum of one hour of every 15 minute vital sign monitoring and 1.5 hours of phase 2 PACU monitoring.



KO TREATMENT PLAN

Intra-operative

1. Call for help. Call for the MH cart and dantrolene. Also, have someone call the MH hotline at #1-800-MH-HYPER (#1-800-644-9737) for added assistance.
2. Discontinue all triggering agents.
3. Change to a clean circuit not exposed to volatile anesthetic agents, using a new clean anesthesia machine or an oxygen tank with an Ambu bag.
4. Hyperventilate with 100% oxygen.
5. Start intravenous dantrolene 2–3 mg/kg IV bolus every 5 minutes up to 10 mg/kg, then start an infusion of 1–2 mg/kg/hr.

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6. Monitor and treat acidosis. Give intravenous sodium bicarbonate 1–2 mEq/kg guided by the pH and base deficit.
7. Cool the patient aggressively with cooling blankets, cold intravenous normal saline at a rate of 15 mL/kg, cold body cavity lavage (including the surgical wound, bladder, and down the nasogastric tube), and apply ice bags to the body.
 - a. The goal of the cooling is to get the patient's temperature to 38 to 39°C.
8. Closely monitor urine output with a Foley catheter. Maintain a goal urine output of >1–2 mL/kg/hr using fluids, furosemide (1 mg/kg IV), and mannitol (0.25 gm/kg IV). Also watch for signs of myoglobinuria.
9. Send off frequent labs, including an arterial/venous blood gas, electrolytes, hepatic function, coagulation panel, CBC, CK, serum glucose, and urine myoglobin. These labs should be closely followed for 24–48 hours.
10. Treat hyperkalemia with a combination of 0.1–0.2 U/kg of regular insulin and 500 mg/kg of dextrose IV, calcium, or bicarbonate.
11. Treat any arrhythmias that develop using amiodarone, lidocaine, adenosine, procainamide, or any other drugs indicated according to the ACLS protocol. Dysrhythmias usually subside with the resolution of the hypermetabolic phase.
 - a. Do not use calcium channel blockers to treat any of the associated arrhythmias as this may worsen the hyperkalemia.
12. Correct metabolic acidosis with 1–2 mEq/kg IV of sodium bicarbonate based on the arterial pH.
13. Have the surgeon stop the surgery as soon as possible. Either expedite or abort the surgery.
14. Consider the placement of an arterial line and central venous catheter.

Post-operative

1. Alkalinize the urine and diurese. Monitor for acute renal failure.
2. Follow CK levels to track the severity of the rhabdomyolysis.
3. Follow all labs and vital signs closely. Beware of hypothermia, hyperkalemia, hypokalemia, and hypervolemic overshoot.
4. Watch for signs of disseminated intravascular coagulopathy (DIC), including thrombocytopenia, hemolysis, and abnormal bleeding.
5. Elevated liver functions are often observed 12–36 hours post-MH crisis.
6. Follow central nervous system (CNS) function serially after an MH crisis.
7. Continue dantrolene 1 mg/kg IV q 4–6 hours for up to 72 hours after an episode of MH.
8. Submit forms to the MH registry.
9. Continue to monitor the patient for up to 72 hours in the surgical intensive care unit. Measurements should include urine output, arterial blood gases, pH, and serum electrolyte concentrations.

MASSETER MUSCLE RIGIDITY (MMR)^{1,2,3,4}

SAMPLE CASE

A 27-year-old male is scheduled for an elective cosmetic procedure. He has no significant medical or surgical history. After induction with intravenous propofol and succinylcholine, the resident notices that she is unable to open the patient's mouth. What are your concerns? Should you cancel the case?

CLINICAL ISSUES

Definition of MMR

1. Rigidity of the jaw muscles after the administration of succinylcholine.⁴
2. MMR has a spectrum of severity from mildly increased jaw tension to “jaws of steel.”
3. Patients who develop MMR after the administration of succinylcholine may have up to a 50% chance of developing MH.
4. If the MMR occurs with volatile anesthetics only, the correlation with MH is almost absolute.
5. The muscle rigidity does not decrease with a repeat dose of succinylcholine or the administration of a non-depolarizing muscle relaxant.⁴

Presentation

1. Jaw rigidity with flaccid limbs after the administration of succinylcholine.
2. Isolated masseter spasm is usually a normal variant but can be a sign of MH.

Differential Diagnosis⁴

1. Myotonic syndrome/dystonia
2. Temporomandibular joint dysfunction
3. Underdosing of succinylcholine
4. Not allowing sufficient time for the succinylcholine to work prior to attempting intubation
5. Normal succinylcholine-induced increase in muscle tone
6. Inadequate analgesia
7. Opioid rigidity
8. Malignant hyperthermia



KO TREATMENT PLAN

Mild Jaw Tension

1. The case may continue, but switch to a non-triggering anesthetic.
2. Make sure you have end-tidal CO₂ and core temperature monitoring.
3. Observe for 12–24 hours for any signs of MH.
4. Must alert the patient of the potential MH risk and need for further testing.

“Jaws of Steel”

1. Delay the procedure unless it is an emergency.
2. This patient requires overnight observation.
3. Observe the patient for any signs of MH, including an increase in temperature, increased pulse rate, cola-colored urine, myoglobinuria, and increased CK levels.
4. Must alert the patient of the potential MH risk and need for further testing.

BIBLIOGRAPHY

1. <http://www.mhaus.org>.
2. Duke J. *Anesthesia Pearls*. Philadelphia: Hanley & Belfus, 2003, pp 151–4.
3. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 716–21.
4. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 530–1.

85. Anaphylaxis^{1,2,3,4,5,6,7}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

You are called to the MRI scanner to see a 20-year-old female who is undergoing a scan of her cervical spine with and without contrast dye. Anesthesia was called secondary to the patient's becoming unresponsive and difficult to ventilate. The nurse reports a rash on the patient's arm. Vital signs: blood pressure 60/40 mm Hg and heart rate 110. Auscultation of lungs reveals bilateral wheezing. What is the most likely cause? How would you treat this?

CLINICAL ISSUES**Definitions**

1. Anaphylaxis is a severe life-threatening allergic reaction secondary to an antigen-antibody interaction.
2. It is an immediate hypersensitivity reaction (type I).
3. Such reactions are usually produced by an immunoglobulin E-mediated (IgE) release of pharmacologically active substances.
4. Anaphylaxis requires prior exposure to the antigen or a substance of similar structure.
 - a. Muscle relaxants
 - b. Thiopental
 - c. Some non-barbiturate hypnotics (propofol)
 - d. Some opioids (morphine, fentanyl)
 - e. Protamine
 - f. Latex
5. Anaphylactoid reactions have an identical or a very similar clinical response but are not IgE-mediated.
 - a. Most narcotics
 - b. Radiographic contrast media
 - c. d-tubocurarine

- d. Plasma expanders
- e. Thiopental

Incidence

1. 1 in 1,000 to 1 in 25,000 anesthetics.
2. The mortality rate can be as high as 6%.

Triggers of Anaphylaxis in Order of Approximate Frequency of Occurrence in the Peri-operative Period

1. Muscle relaxants, especially succinylcholine, rocuronium, vecuronium, and atracurium, can cause up to 60–70% of anesthesia-related anaphylaxis.
 - a. Cross-reactivity can occur with acetylcholine, choline, morphine, neostigmine, pentolinium, procaine, and promethazine.
 - b. The pre-exposure to these drugs may sensitize a patient to a resulting reaction to muscle relaxants without prior anesthesia.
2. Latex is the second-most common intra-operative anaphylactic agent. Patients at the highest risk include health care workers, children with spina bifida and genitourinary abnormalities, multiple prior surgeries, history of fruit allergies (including banana and avocado), and workers with occupational exposure to latex. These patients should receive medical treatment in a “latex-free” environment.
3. Antibiotics, particularly β -lactam antibiotics and vancomycin, can cause an anaphylactic reaction. Penicillin accounts for the greatest number of fatal anaphylactic drug reactions in the general population.
4. Induction agents or hypnotics, including thiopental and propofol, may cause a rare but often life-threatening reaction. An allergic reaction to propofol often presents with bronchospasm.

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5. Opioid anaphylaxis is rare. Morphine more commonly evokes an anaphylactoid reaction secondary to the histamine release from mast cells.

6. Volume expanders: including synthetic plasma protein solutions

7. Blood products: allergic reactions occur in about 3% of properly cross-matched blood.

8. Protamine: increased risk in patients with a history of allergy to seafood (protamine is derived from salmon sperm) and patients with diabetes mellitus treated with protamine-containing insulin preparations. Men with vasectomies may develop circulating antibodies to spermatozoa, but any increased risk is unlikely. Protamine can also cause a direct histamine response, especially if given too rapidly.

9. Methylmethacrylate (bone cement)

10. Radiographic contrast media evoke an allergic reaction in about 5% of patients. This reaction tends to be an anaphylactoid reaction and can be modified by pre-treatment with corticosteroids and diphenhydramine.

11. **Local anesthetics: local anesthetic-induced allergic reactions** are rare. Ester- based local anesthetics are metabolized to a highly antigenic compound called para-aminobenzoic acid (PABA) and are more likely than the amide- based local anesthetics to evoke an allergic reaction. Esters and amides have very rare cross-reactivity and it is considered safe to choose a local anesthetic from the other group. Just make sure that you are using a preservative-free solution.

Clinical Signs and Symptoms

It may take 2–15 minutes for symptoms to develop in a sensitized patient, with rare instances taking as long as 2.5 hours. An allergic reaction to latex has been shown to often have a delayed onset of symptoms of up to 30 minutes. Late-phase or biphasic reactions can even occur 8–12 hours after the initial reaction. Protracted anaphylaxis may last up to 32 hours, despite aggressive treatment.

First Wave

1. Vasodilation
2. Feeling of impending doom

Second Wave

1. Cutaneous manifestations
 - a. Urticaria
 - b. Rashes
 - c. Edema of the skin and face/angioedema
 - d. Erythema/flushing
 - e. Pruritus
2. Pulmonary
 - a. Upper airway edema and obstruction
 - b. Lower airway obstruction
 - c. Bronchospasm
 - d. Pulmonary and laryngeal edema

- e. Dyspnea
 - f. Wheezing
 - g. Chest discomfort
 - h. Coughing
 - i. Sneezing
 - j. Decreased pulmonary compliance
 - k. Acute respiratory failure
3. Cardiovascular
 - a. Hypotension secondary to hypovolemia
 - b. Tachycardia
 - c. Vasodilation at the levels of the capillary and postcapillary venule
 - d. Increased capillary permeability with up to 50% extravasation of intravascular fluid volume into extracellular fluid spaces
 - e. Cardiovascular collapse with associated myocardial ischemia and cardiac dysrhythmias
 - f. Syncope
 - g. Dizziness
 4. Gastrointestinal
 - a. Nausea
 - b. Vomiting
 - c. Diarrhea
 5. Miscellaneous
 - a. Headache
 - b. Substernal chest pain
 - c. Seizure
 - d. Disorientation

Treatment Goals

1. Correct the arterial hypoxemia.
2. Inhibit the further release of chemical vasoactive mediators.
3. Restore the intravascular volume.

Differential Diagnosis

1. Vasovagal reaction
 - a. Urticaria, flushing, angioedema, and pruritus are usually absent. Skin is usually cool and pale.
 - b. Bradycardia
 - c. Bronchospasm is absent.
 - d. Blood pressure is usually normal or increased.
 - e. Hypotension
 - f. Weakness
 - g. Nausea and vomiting
 - h. Diaphoresis
2. Acute anxiety
 - a. Panic attack
 - b. Hyperventilation syndrome
3. Myocardial dysfunction
4. Pulmonary embolism
5. Systemic mast cell disorder
6. Foreign-body aspiration
7. Acute poisoning

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8. Hypoglycemia/hyperglycemia
9. Seizure disorder
10. Hereditary angioedema
11. Asthma
12. Urticaria pigmentosa
13. Basophilic leukemia
14. Shock (hemorrhagic, hypoglycemic, cardiogenic, or endotoxic)
15. Flushing syndromes
 - a. Niacin, nicotine, angiotensin-converting enzyme inhibitors (ACEIs), and alcohol.
 - b. Postmenopausal
16. Carcinoid
17. "Red man" syndrome with vancomycin
18. Monosodium glutamate (MSG)
19. Sulfites
20. Pheochromocytomas
21. Capillary leak syndrome



KO TREATMENT PLAN

Pre-operative

Intravenous contrast material is probably the most frequently used agent that causes anaphylactoid reactions outside of the operating room. Pre-treatment with diphenhydramine, ranitidine, and corticosteroids (up to 1 gram of methylprednisolone) has been reported to be useful in preventing or ameliorating anaphylactoid reactions to IV contrast material and perhaps narcotics.

Avoid all medications that may have a potential cross-reactivity. If the patient has a latex allergy, remove all latex from the operating room and suggest that the patient be scheduled as the first case of the day, when latex particles are the lowest in the air.

Intra-operative

1. Assess her airway. Look for signs of dysphonia, stridor, cough, wheezing, or shortness of breath.
2. Evaluate her vital signs. Place standard ASA monitors. Look for hypotension or cardiac arrhythmias.
3. Assess the patient's state of consciousness.
4. Look at the patient's skin for diffuse or localized edema, pruritus, urticaria, and/or angioedema.
5. Stop the offending agent. Remove all latex from the environment. Discontinue all anesthetic drugs as soon as possible.
6. Call for help.
7. Secure the airway.
8. Ensure adequate oxygenation with 100% oxygen.
9. Consider early tracheal intubation if any angioedema is present.
10. Subcutaneous or intramuscular epinephrine (1:1000 = 1 mg/mL) 0.2–0.5 mL SC or IM every 5 minutes can be used

for minor non-life-threatening symptoms such as a rash. Pediatric dosing is 0.01 mg/kg to a maximum dose of 0.3 mg. The patient's thigh is the preferred location for injection. This technique should not be used for patients under general anesthesia. The sample patient requires more aggressive treatment because she already has hemodynamic compromise.

11. IV epinephrine is to be administered when the patient develops life-threatening symptoms such as cardiac arrest or profound hypotension that fails to respond to intravenous volume replacement and several SC/IM injections of epinephrine (1:1000 = 1 mg/mL). Give 5–10 mcg IV boluses for hypotension, 100–500 mcg IV for cardiovascular collapse, and 1–3 mg IV for cardiac arrest. Then infuse 1–4 mcg/min up to a maximum dose of 10 mcg/min. If the patient is in cardiac arrest, follow ACLS protocol and consider atropine and transcutaneous pacing. Prolonged resuscitation is encouraged because efforts are more likely to be successful in a patient with anaphylaxis.

12. Volume expansion with IV fluids: 3–4 L of crystalloid normal saline are recommended.

13. If the patient's hypotension is refractory to volume replacement and epinephrine, consider a vasopressor infusion. Consider dopamine, norepinephrine, phenylephrine, or vasopressin to maintain perfusion pressure until the intravascular volume can be restored.

14. Treat any dysrhythmias.

15. Antihistamines are a second-line therapy to epinephrine and should never be used alone in the treatment of anaphylaxis. Use an H1 and H2 antagonist combined.

a. H1 antagonist: diphenhydramine 25–100 mg IV for adults and 1mg/kg for children (up to 50 mg)

b. H2 antagonist: ranitidine 12.5 to 50 mg IV in adults and up to 1 mg/kg in children.

16. Intravenous corticosteroids should be given every 6 hours to a dose equivalent to 1–2 mg/kg/day. No effect may be seen for up to 4–6 hours.

17. Bronchospasm resistant to epinephrine should be treated with inhaled β -agonist bronchodilators (nebulized albuterol).

18. Aminophylline should be considered in patients with persistent bronchospasm and hemodynamic instability as a second-line therapy after β -agonist bronchodilators.

19. Consider a glucagon infusion of 1–5 mg IV given over 5 minutes followed by an infusion of 5–15 mcg/min titrated to a clinical response when a concomitant β -adrenergic blocking agent complicates the treatment. Observe aspiration precautions secondary to the glucagon's increased side effects of causing nausea and emesis.

20. Sodium bicarbonate 0.5–1 mEq/kg IV can be given for persistent hypotension or acidosis.

21. Send labs

a. Serum tryptase: peaks 1–2 hours after the onset of symptoms and can persist for up to 6 hours. Ideal measurement is obtained between 1 and 2 hours after the initiation of symptoms. Can be used to differentiate between anaphylactic and anaphylactoid reactions.

85. Anaphylaxis

b. Plasma histamine metabolites: levels begin to increase within 5–10 minutes and remain elevated for only 30–60 minutes.

c. Urine histamine metabolites: levels may remain elevated for up to 24 hours.

d. Plasma-free metanephrine and urinary vanillylmandelic acid can be drawn to rule out a pheochromocytoma.

e. Serum serotonin and urinary 5-hydroxyindoleacetic acid can be drawn to rule out carcinoid syndrome.

Post-operative

All patients who have experienced an anaphylactic reaction require 24 hours of continued ICU-like observation due to the fact that another wave of pro-inflammatory cytokines can promote the recurrence of symptoms. Elective surgeries should be postponed up to 24–36 hours after the symptoms resolve. A truly emergent surgery should proceed with caution. Delay the surgery until the patient is stable and adequately resuscitated. The patient can be transferred to the ICU intubated or remain in the OR for resuscitation prior to resuming the emergent surgery. Be prepared to treat any recurring anaphylaxis.

Counseling and allergy skin testing can be done to determine the true cause of the allergic reaction. However, if the

clinical history to a specific agent is strong, allergy testing may be unnecessary or even dangerous. An allergist may be helpful if the diagnosis is doubtful, symptoms recur or are difficult to control, the patient is a candidate for sensitization, the patient requires daily medications for prevention, or the patient requires intense education. The patient should also be encouraged to wear a medical alert bracelet and carry self-administrable epinephrine.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 514, 1092–3.
2. Rakel RE. *Textbook of Family Medicine*, 7th ed. Philadelphia: Saunders, 2007, figure 26–1.
3. Goldman L, Ausiello D. *Cecil Textbook of Medicine*, 23rd ed. Philadelphia: Saunders, 2007, Chapter 458.
4. Lieberman P, Kemo S, Oppenheimer J, et al. The diagnosis and management of anaphylaxis: An updated practice parameter. *J Allergy Clin Immunol*, 2005;115(3) Suppl 2:s483–523.
5. Chacko T, Ledford D. Peri-anesthetic anaphylaxis. *Immunol Allergy Clin North Am*, 2007;27(2):213–30.
6. Stoelting RK, Dierdorf SF. *Anesthesia and Co-Existing Disease*, 4th ed. Philadelphia: Churchill Livingstone, 2002, pp 612–20.
7. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1302–7.

86. Peripheral Nerve Blocks^{1,2,3,4,5}

Joni Maga, MD

SAMPLE CASE

A 63-year-old obese male has come for a pre-surgical evaluation prior to his total knee arthroplasty. What anesthetic techniques can be offered to him? What other information is needed?

TKO: Peripheral nerve blocks (PNB) can be used as the sole anesthetic, as an adjuvant to general anesthesia or sedation, and for post-operative pain relief. When implemented properly, they are a valuable tool in any anesthesiologist's armamentarium.

CLINICAL ISSUES

Benefits of Peripheral Nerve Blocks

1. Some studies have concluded the following:
 - a. A PNB can provide superior post-operative analgesia when compared with parenteral opioids (usually intravenous (IV) morphine) and intra-articular opioids in joint operations.
 - b. A PNB can contribute to faster post-operative recovery for inpatients and outpatients.
 - c. A PNB can facilitate rehabilitation by providing pain control and increased passive joint mobility.
 - i. There is also some evidence that this can decrease days of hospitalization and therefore reduce medical costs.
 - d. The side effects of opioids may be avoided: nausea/vomiting, itching, respiratory depression, ileus, and constipation.
 - e. The need for airway manipulation may be avoided (i.e., asthma).
 - i. Note: In the case of a difficult airway there is concern that although a PNB can obviate the need for a secure airway. Failure or complications of a PNB can result in having to obtain an airway in an emergency situation with a difficult airway candidate.
 - f. Other benefits suggested
 - i. Increased patient satisfaction
 - ii. Reduced post-anesthesia care unit (PACU) care
 - iii. Increased mental alertness and responsiveness
 - iv. Faster ambulation
 - v. Improved pain control especially for patients with pre-existing chronic pain conditions and opioid tolerance

General Contraindications for a Peripheral Nerve Block

1. Patient refusal
2. Proven allergy to local anesthetics
 - a. Ester allergies are usually due to para-aminobenzoic acid.
 - b. Amide allergies are usually due to preservatives or antibacterial additives.
3. Local infection
4. Active bleeding in an anticoagulated patient
 - a. Non-compressible vasculature may be a concern near nerve sites in the anticoagulated patient.
 - b. Note: There are guidelines for neuraxial regional techniques in the anticoagulated patient that can be extrapolated but generally are too strict to be applied to most PNB techniques.
5. Preexisting peripheral/central nerve injury
 - a. There is no evidence that a PNB will worsen a pre-existing neuropathy; however, the post-operative neurological assessment and follow-up may be complicated by a PNB and in some cases may outweigh the benefits of the block.
6. Need for functional nerve assessment
 - a. This may be the difference between doing the PNB pre-operatively versus post-operatively.

Complications of a Peripheral Nerve Block

1. Local anesthetic toxicity
 - a. Intravascular injection or slow systemic absorption (delayed 5–30 minutes) in highly vascular sites
 - b. Always use standard monitors (EKG, blood pressure, and pulse oximetry) and have the appropriate resuscitation equipment nearby (oxygen, suction, bag-mask, and intubation equipment) when placing a PNB.
 - c. Inject the local anesthetic slowly with frequent aspiration.
 - d. The patient receiving a PNB should always have functioning intravenous access.
 - e. The patient should remain monitored for at least 30 minutes post-block to monitor for complications.
 - f. The symptoms of local anesthetic toxicity are usually gradual and on a continuum.
 - i. Signs of tinnitus and circumoral paresthesia appear first followed by dizziness, lightheadedness, and anxiety followed by a seizure, which will precede cardiac arrest.

86. Peripheral Nerve Blocks

2. Peripheral nerve injury

- a. Most common mechanisms: needle trauma or intraneural injection
 - i. Do not inject under pressure or if the patient experiences increased pain upon injection.
 - ii. The etiology (surgical versus PNB) of the nerve injuries is usually clinically indistinguishable.
 - (1) Document any previous numbness or nerve injury prior to the PNB.
 - iii. Most peripheral nerve injuries will resolve without further incident, usually within 3 months.
 - (1) However, some will require longer than 3 months to resolve while nerve regeneration takes place.
 - iv. Consider a neurology consult with motor injuries and persistent sensory injuries that are unexplainable in relation to the operative technique or a pre-existing condition.
 - v. Patient reassurance should also be part of the management.

3. Vascular puncture: hematoma

Local Anesthetics and Dose

1. The most commonly used local anesthetics in PNB are
 - a. Lidocaine
 - i. Concentration: 1.5–2%
 - ii. Fast onset
 - iii. Short duration
 - b. Mepivacaine
 - i. Concentration: 1.5–2%
 - ii. Fast onset
 - iii. Short duration
 - c. Bupivacaine
 - i. Concentration: 0.5%
 - ii. Slow onset
 - iii. Long duration
 - d. Ropivacaine
 - i. Concentration: 0.5–1%
 - ii. Slow onset
 - iii. Long duration
 - iv. Note: ropivacaine has been shown to be less cardiotoxic and to induce less motor block than bupivacaine.
 - e. The dose and combination are variable with the addition of epinephrine as well as the volume necessary for that particular site.
 - i. Interscalene and supraclavicular block: 30 mL
 - ii. Infraclavicular and axillary block: 30–40 mL
 - iii. Lumbar plexus, femoral, and popliteal block: 35–40 mL
 - iv. Posterior sciatic: 20–25 mL

TKO: Note: The volume suggested is the volume necessary to block the nerves or plexus in that area. The concentration can be adjusted so total dose calculated is less than the toxic dose.

2. Additives

- a. Epinephrine, 1:200,000 or 1:400,000
 - i. Increases duration and intensity of the block by slowing the systemic absorption of the local anesthetic.
 - (1) Mepivacaine and lidocaine but not bupivacaine or ropivacaine
 - ii. Can also be valuable when detecting intravascular injection or absorption.
- b. Sodium bicarbonate (NaHCO_3), add 1 mL per 10 mL of local anesthetic
 - i. Reduces the onset time of lidocaine and mepivacaine but not bupivacaine and ropivacaine.
 - (1) Increases the pH of the local anesthetic solution making the solution closer to the pKa and thereby making the lipid soluble form more prevalent. The non-ionized lipid soluble form crosses the membrane.
- c. Clonidine, 1–2 mcg/kg has been shown to increase the duration of some blocks.

Techniques

1. Interscalene brachial plexus block
 - a. Indications: operations involving the shoulder, upper, and lower arm
 - b. Shortcomings
 - i. Can spare C8 and T1 nerve distribution (ulnar nerve) 15–30% of the time due to lack of local anesthetic spread to the lower nerve roots.
 - ii. Can be detrimental in patients with chronic obstructive pulmonary disease (COPD) (see below).
 - c. Complications specific to the interscalene block
 - i. Pneumothorax
 - ii. Spinal or epidural block: injection into the intervertebral foramina or dural sleeve
 - (1) When using a nerve stimulator, avoid injecting when you still have a muscle twitch at a voltage less than 0.2 mA.
 - iii. Vertebral artery injection/injury
 - iv. Hoarseness: the most common side effect of this block
 - (1) Occurs mostly with a right-sided injection.
 - (2) Incidence is 10–20%.
 - v. Phrenic nerve paralysis and therefore diaphragmatic paralysis
 - (1) The incidence is 100% due to the position of the nerve running anterior to the anterior scalene muscle.
 - (2) Consider another technique on patients with severe pulmonary COPD and those dependent on the use of their accessory respiratory muscles.
 - (3) Avoid bilateral interscalene blockade in patients with respiratory compromise.
 - vi. Cervical plexus blockade can occur with the injection of large volumes of local anesthetic.
 - vii. Horner's syndrome: local anesthetic spread to the sympathetic chain

86. Peripheral Nerve Blocks

- i. Will likely require blockade of the lumbar plexus, femoral, or saphenous nerves for supplementation.
- ii. Proximal approach will cover tourniquet pain but contribute to hamstring weakness and can potentially block the parasympathetic nerves that control bladder emptying.
- c. Complications specific to the sciatic block: none; see general complication list.



KO TREATMENT PLAN

1. Identify the proper patient and the correct operation. Choose the technique that exemplifies the most benefits over the risks.
2. Consider surgical time, positioning, and the addition of sedation or general anesthesia when choosing a pre-operative technique.
3. Consider the need for post-operative nerve assessments, ambulation, and anticoagulation.

Case Discussion

In this patient, a complete history and physical including an airway exam are needed before deciding if a PNB is right for

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him. If the patient proves to be a difficult airway, do not use the PNB as a way to avoid it. Remember, it is better to handle a difficult airway electively rather than emergently. Also, consider the patient's body habitus and the skill level of the regionalist. Consider the strength of the non-operative leg since the operative leg will be further weakened by the block and the patient may be more prone to falling. Assuming no other medical history and a normal airway, this patient would benefit from a continuous femoral blockade performed pre- or post-operatively.

BIBLIOGRAPHY

1. Barash, M.D., Paul G., Bruce F. Cullen, M.D., and Robert K. Stoelting, M.D., eds. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 718–45, 1118–26.
2. Hadzic, Admir. *Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill Professional, 2006, pp 144–65, 403–543.
3. Finucane, Brendan T., ed. *Complications of Regional Anesthesia*. New York: Springer, 2007, pp 39–52, 74–86.
4. Second Consensus Conference on Neuraxial Anesthesia and Anticoagulation, April 25–28, 2002, Regional Anesthesia in the Anticoagulated Patient: Defining the Risks.
5. Mulroy, Michael F. Regional Anesthesia – When to Say Yes, When to Say No, ASA Annual Meeting Refresher Course Lectures, 2007, p 507.

87. Complex Regional Pain Syndrome (CRPS) Types I and II^{1,2,3}

Brendan J. Astley, MD

SAMPLE CASE

A 21-year-old female softball pitcher had a surgical ulnar nerve transposition/release for pain in her pitching arm three months ago. Now she still complains of severe pain daily. She can barely bend her elbow and has decreased function of her hand. She notes increased sensitivity to cold temperatures and generalized swelling, which is more pronounced on the ulnar side of her hand. She is in your office for pain management. What are your concerns? What is her diagnosis? How will you confirm the diagnosis? How will you treat the patient's pain?

CLINICAL ISSUES

Definition

CRPS is a chronic progressive disease characterized by severe pain, swelling, and changes in the skin as a result of dysfunction of the central and peripheral nervous system.

Historically, many terms that reference these symptoms have been used to depict this condition. Previously, the two subtypes of CRPS have been referred to as reflex sympathetic dystrophy and causalgia. Because these terms have lost their clinical utility, in 1994 the ISAP developed the term *complex*

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regional pain syndrome to emphasize the following clinical characteristics:

1. Complex: multiple and varied clinical features
2. Regional: majority of cases involve a region of the body, usually an extremity
3. Pain: cardinal feature, often out of proportion to original insult and essential to the diagnosis
 - a. Type I: also known as reflex sympathetic dystrophy (RSD), Sudeck's atrophy, shoulder and hand syndrome; does not have demonstrable nerve lesions.
 - b. Type II: also known as causalgia; has evidence of obvious nerve damage.

Typical Features of CRPS

CRPS Types I and II

Features of both types that can be shared include

1. Decreased function of the extremity
2. Pain out of proportion to known injury
3. Pain that is now chronic in nature
4. Skin changes such as mottling and cyanosis
5. Increased sweating of the affected limb during the acute phase
6. Allodynia: a painful sensation caused by stimulation that is non-noxious
7. Hyperalgesia: an exaggerated painful response that is caused by a sensation that is usually noxious
8. Edema
9. Shiny skin at the affected site
10. Hair loss at the affected site
11. Temperature sensitivity: cold intolerance

Type I

1. Caused by an unknown or minor limb injury
2. The pain is present in a non-dermatomal distribution pattern.

Type II

1. The pain presents after a known or major peripheral nerve injury.
2. The pain may present with a dermatomal distribution or not.

In either type, the pain can spread into new dermatomes, especially if caused by sympathetic nervous system activation. Also, rarely (2% of cases) the pain and physical exam findings may include other extremities, usually in a mirror image fashion, which would suggest a more central origin for the pathology.

There are three stages of the disease:

1. Stage one (0–3 months):
 - a. Severe, burning pain at the site of the injury
 - b. Muscle spasm
 - c. Joint stiffness, restricted mobility, and decreased range of motion
 - d. Nail growth
 - e. Puffy swelling
 - f. Redness and warmth
 - g. Hyperhidrosis
 - h. Radiographs are usually normal.
 - i. Positive bone scan
 - j. Aggressive treatment at stage one yields the best outcome.
2. Stage two (3–6 months from symptom onset):
 - a. Intense pain
 - b. Skin atrophy with hard edema
 - c. Diminished hair growth
 - d. Nails become cracked, brittle, grooved, and spotty.
 - e. Osteoporosis develops quickly.
 - f. Joints thicken.
 - g. Muscles atrophy.
3. Stage three (6–12 months from symptom onset):
 - a. Irreversible changes occur in the skin, muscles, and bones.
 - b. The pain becomes unyielding and may involve the entire limb.
 - c. There is marked muscle atrophy with advanced contractions.

Incidence

1. CRPS type I has an incidence of 1–2% after a fracture.
2. 2:1 to 4:1 female to male ratio
3. The mean age of presentation is 37 to 50 years old.

Mechanism for CRPS Pain

1. The pathophysiology of this disease is still unclear.
2. The pain usually starts acutely within hours to days.
3. Most pain researchers believe CRPS is developed and maintained by abnormalities in the peripheral and central nervous systems.
4. The peripheral component is evidenced by
 - a. Peripheral sensitization of primary nociceptive afferent neurons associated with sympathetic efferent coupling
 - b. Up-regulation of ion channels and adrenergic receptors
 - c. Increased concentrations of neuropeptides
5. The central component is explained by spinal cord changes in the dorsal horn containing wide dynamic range (WDR) neurons.
 - a. Mechanisms of central sensitization resulting in increased perception of pain could include spontaneous firing of the WDR neuron as well as amplification of ascending signals or insufficient descending inhibitory signal.
 - b. Multiple neurotransmitters, receptors, and cytokines have been implicated.
 - i. Glutamate
 - ii. Magnesium
 - iii. Glycine

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- iv. Substance P
 - v. GABA
 - vi. NK (neurokinin)
 - vii. 5HT (serotonin)
 - viii. α_2 receptors
 - ix. μ -receptors
 - x. Prostaglandin
6. Newer imaging techniques – positron emission tomography (PET) and functional magnetic resonance imaging (MRI) – have confirmed a central reorganization of somatosensory sensations that improve with the treatment of the pain.
- a. This means that actual areas of the brain have been “rewired” due the chronic nature of this pain.
 - b. Therefore, there is an actual pathologic anatomy that may be the cause of, or result of, the chronic pain.
7. Vascular blood flow changes can occur after a major nerve injury.
- a. This impaired blood flow can lead to dystrophic changes.
 - i. Skin and nail changes
 - ii. Impaired blood flow causing mottling
 - b. May be associated with hyperactive sympathetic discharge and impaired sympathetic function.
8. Inflammatory cells such as macrophages and mast cells are implicated in inflammatory changes associated with CRPS, but whether their role is primary or secondary remains controversial.
- a. Localized neurogenic inflammation may cause sweating, edema, and vasodilatation.
 - b. This inflammatory response is usually only seen in the acute phase, less than 6 months post-injury.
9. In CRPS, after the inflammation subsides from the initial event, it is thought that the nociceptors in the affected limb are still sending signals centrally. This causes the central nervous system to become hyperexcitable.
10. A genetic predisposition exists.
- a. Certain women have a specific human leukocyte antigen (HLA) profile found on chromosome 6 and/or angiotensin-converting enzyme (ACE) gene deletion on chromosome 17.

Tests to Help Confirm the Diagnosis

1. There is no single test used to diagnose CRPS.
2. Most physicians agree that the patient’s clinical history is the most important factor in making the correct diagnosis.
3. Response to sympathetic blockade
 - a. Previously, a reduced pain response to a sympathetic blockade was considered mandatory for the diagnosis of CRPS.
 - b. This is no longer the case because a sympathetic pain block may or not may alleviate the pain, even if the pain is sympathetically maintained.
 - c. The sympathetic block can help differentiate between sympathetically maintained pain (SMP) and sympathetically independent pain (SIP), but it does not definitively diagnose CRPS.

4. Sudomotor testing: sweat testing
 - a. During any stage of CRPS, the patient may have abnormalities in sweat gland function.
 - b. Sweating may be excessive or reduced.
 - c. Special tests can be used to measure resting sweat output, thermoregulatory sweating, and quantitative sudomotor axon reflex testing.
 - d. These tests are helpful if positive, but not if they are negative.
 - e. These tests are difficult to do and are currently available in only a few parts of the United States.
5. Three-phase bone scan
 - a. Osteopenia is common within one year from the beginning of the syndrome.
 - b. A three-phase bone scan can help confirm the diagnosis of CRPS associated osteopenia.
 - c. It is not particularly useful because it is usually positive in only 50% of CRPS cases.
6. Skin temperature measurements at rest may be only slightly different in the affected limbs.
 - a. With whole body cooling or warming, the differences dramatically increase the sensitivity and specificity for CRPS.
 - b. The variation in temperature between the extremities is due to vascular constriction and dilation caused by sympathetic nervous system dysfunction.
 - c. Infrared thermography is a diagnostic imaging procedure that records the body surface temperature by detecting heat emitted from the surface of the skin. It can detect very small differences in temperature.
7. Nerve conduction testing and electromyography (EMG)
 - a. Half of all CRPS patients will show an abnormality.
 - b. Not very useful, and rarely used for diagnosis of CRPS.
8. Plain X-rays and MRI
 - a. More useful in later stages, and not as a screening tool
 - b. X-ray may show demineralization of bone.
 - c. MRI can show soft tissue changes and swelling.
9. Post-traumatic neuralgia (PTN) may mimic CRPS I and II.
 - a. In PTN, the pain is felt only in the area innervated by the injured nerve.
 - b. It usually presents with a less complex picture overall.
 - c. The patient may complain of burning pain.
 - d. There may be some cold sensitivity.
 - e. Sympathetic nerve blocks may relieve this pain but less often than in CRPS.



KO TREATMENT PLAN

The patient in the sample case likely has CRPS. Her diagnosis can be made by a thorough clinical examination. Treatment and physical therapy should begin immediately.

Treatment

1. Physical therapy and occupational therapy are vitally important to maintain function in a given extremity. Physical

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therapy should be instituted as early as possible. Most patients require some medications/treatments to tolerate the physical therapy. Take advantage of the window of opportunity that exists after a block is performed. During this relatively pain-free period, intensive physical therapy should occur to improve function of the affected limb.

2. Non-steroidal anti-inflammatory medications (NSAIDs)
 - a. These can be used to control low levels of pain and as an adjuvant therapy.
3. Opioids
 - a. Most experts would agree that these should be prescribed in the early phase of the disease.
 - b. Studies are lacking as to long-term benefits.
4. Steroids: high dose
 - a. May be helpful in the acute phase of disease (<13 weeks).
5. Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) may be helpful in CRPS, but studies are lacking.
6. Lidocaine IV does decrease the pain in CRPS.
 - a. Intravenous lidocaine 2 mg/kg with 0.5 mg/kg of ketamine, given in a Bier block to the affected extremity, has been reported to give resolution of symptoms in CRPS.
 - b. Lidocaine PO (mexiletine) does not appear to be effective.
 - i. It was previously thought that the membrane stabilizing effects might be advantageous to people with CRPS.
 - ii. This, however, has not been proven.
 - c. There have been reports of Lidoderm patches (5%) giving good pain relief with CRPS.
7. Some experimental evidence has shown Gabapentin (Neurontin) to reduce pain associated with CRPS.
 - a. A typical starting dose would be 300 mg at night, slowly increasing over the course of one week to three times a day dosing.
 - b. Titration of the medication should continue in a similar fashion to minimize side effects and optimize analgesic properties.
 - c. Daily maximum dosing should not exceed 3,600 mg/day.
 - d. Decreased dosing is necessary for patients with renal impairment.
8. Clonidine patches are also effective when smaller areas of pain are present.
 - a. The improvement of the pain is believed to be due to a reduction of the sympathetic nervous system activity.
 - b. A typical starting dose should be a patch of 0.1 mg/day applied weekly.
 - c. The patch should be applied to the affected area.
 - d. Caution should be taken when using clonidine as it may cause hypotension; also if used for a prolonged period of time and abruptly stopped it may lead to rebound hypertension.
 - e. The dose may be increased as tolerated if it is effective in relieving pain in a given small area and the patient's blood pressure can tolerate it.

9. Ketamine

- a. The only potent N-methyl-D-aspartate (NMDA)-blocking drug currently available for clinical use
- b. Ketamine is given IV 10–90 mg/hour over several treatment days.
- c. Some studies show promising results.

10. Sympathetic blocks

- a. Stellate ganglion (upper extremity) block
 - i. Should be performed in a series of 3–6 injections.
 - ii. Should give immediate pain relief.
 - iii. Physical therapy should be planned in conjunction with the improved pain control.
 - iv. This block can be repeated weekly, or delayed longer if pain does not return.
 - v. The block should result in good pain relief and the patient should have improved function in the extremity. If not, the block series should be discontinued.
 - vi. The usual dose is 15 cc of bupivacaine 0.5%. This should give definite results and longer lasting pain relief.
 - vii. Horner's syndrome (miosis, enophthalmos, ptosis, and anhidrosis) is often seen as a side effect of the successful block.
 - viii. Omnipaque is injected before the bupivacaine to ensure that the needle is in the proper position. Also, the omnipaque is injected with the bupivacaine to ensure that the medication travels inferiorly toward the T1 level where the stellate ganglion lies.
 - ix. The stellate ganglion block is typically performed at the C6 level due to the increased risk of vertebral artery puncture and pneumothorax at lower levels. The bupivacaine is injected caudally in the direction of the stellate ganglion.
 - b. Lumbar sympathetic (lower extremity) block
 - i. Effective at reducing pain compared with saline control injections up to 24 hours after the injection.
 - ii. Treatment goals are similar to those with the stellate ganglion block.
 - iii. Bupivacaine 0.5% about 20 cc with omnipaque would be a typical choice for injection.
 - iv. The omnipaque would be injected before the bupivacaine to make sure the needle is in the proper position. The omnipaque would also be injected and mixed with the bupivacaine to watch its spread, ensuring that the injection is not intravascular.
- ## 11. Other blocks that may be helpful depending on the site of pain include
- a. Celiac plexus block
 - i. Bupivacaine 0.5%, 15 cc per side can be used to determine if the cause of the pain can be relieved with this block.
 - ii. Usually used for pancreatic pain.
 - (1) Palliative treatment, in cases such as terminal pancreatic cancer, can be performed and would include neurolytic block after the local anesthetic block has confirmed that the pain has been relieved with this block.

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- (2) Neurolytic agents, including phenol and alcohol
 - (a) Alcohol is painful on injection.
 - (b) Phenol is painless on injection.
 - (c) Alcohol and phenol have about the same efficacy.
 - (d) Neurolytic blocks are reserved for patient at the end of life as the pain can return in weeks to months.
- b. Splanchnic nerve block
 - i. Again, one may use bupivacaine for the injections with the goal of reducing the pain acutely.
 - ii. If more permanent pain relief is desired based on the clinical situation, then alcohol or phenol may be used with a local anesthetic.
- c. Hypogastric plexus and ganglion impar blocks
 - i. May be performed with a local anesthetic such as bupivacaine or with neurolytic agents.
12. Transcutaneous electrical nerve stimulation (TENS)
 - a. This is a small, battery-operated unit; electrodes are placed by the patient over the affected area.
 - b. The electrical current stimulation is mild; however, it can stop pain impulses from being transmitted to the central nervous system.
 - c. The TENS unit can also cause endorphins to be released centrally, which will modify the patient's perception of pain.
 - d. The unit is controlled by the patient; stimulation can be increased or decreased as desired.
13. Spinal cord stimulator (SCS)
 - a. The SCS leads are implanted dorsally in the epidural space.
 - b. The electrical impulses generate a tingling sensation that helps prevent painful impulses from being perceived by the patient.
 - c. Typically, a six-lead electrode is implanted for a trial period of one week.
 - d. The permanent implant surgery is performed in the operating room under sedation and the electrode is initially adjusted intra-operatively with the patient

confirming that the electrical stimulation is covering the areas of pain.

e. Once the electrode wire is secured in place, the wires are attached to an external generator and the patient is taken to the recovery area for further adjustments of the stimulator.

14. Psychiatric treatment

a. Psychiatry should be involved with these chronic pain patients at the beginning of their treatment.

b. Often these patients are frustrated and depressed.

c. The depression may set in early if their limb function does not improve with therapy.

d. They will also need to be involved before a SCS can be placed.

15. Surgical sympathectomy does not have good results for the vast majority of patients and therefore is not usually recommended.

Prognosis

1. Follow-up studies show that the majority (62%) of patients are limited in daily activities at 5 years.

2. The majority (60%) of patients with CRPS II, even with intensive therapy, are still in pain at one year.

3. Patients with CRPS I are much more likely to have a resolution of their symptoms at one year (74%) compared to patients with CRPS II.

4. Some degree of functional dysfunction is likely to be present in the affected extremity for at least 13 months after the CRPS is diagnosed.

BIBLIOGRAPHY

1. Waldman SD. *Pain Management*, Vol. 1. Philadelphia: Saunders Elsevier, 2007, pp 283–303.
2. Benzon HT, Raja SN, Molloy RE, et al. *Essentials of Pain Medicine and Regional Anesthesia*, 2nd ed. Philadelphia: Elsevier, 2005, pp 376–85.
3. <http://www.nationalpainfoundation.org>. Diagnosing Complex Regional Pain Syndrome (CRPS).

88. Opioid Addiction^{1,2,3,4}

Brendan J. Astley, MD

SAMPLE CASE

A 47-year-old female presents for a total knee arthroplasty. Her medical history is positive for hypertension, rheumatoid arthritis, and a previous history of drug abuse. Her current medications include hydrochlorothiazide (HCTZ), lisinopril, and buprenorphine hydrochloride (Subutex). The patient states that if possible she would like to avoid narcotics. What is your anesthetic plan? What is Subutex? How will it affect your anesthetic medications? Is it possible to completely avoid narcotics in the peri-operative period?

CLINICAL ISSUES

Definition of Opioid Addiction

As defined by the World Health Organization, “a state, psychic and sometimes also physical, resulting from the interactions between a living organism and a drug, that includes a compulsion to take the drug on a continuous or periodic basis in order to avoid the discomfort of its absence. Tolerance may or may not be present.”

Definition of Tolerance

1. A condition in which the typical effect of a drug is diminished due to previous drug exposure.
2. The previous exposure has caused a change in the way a person deals with the drug either physically, mentally, or both.

Opioid Receptor Types

1. Mu (μ_1, μ_2, μ_3): analgesia, respiratory depression, euphoria, sedation, and gastrointestinal dysmotility
2. Kappa ($\kappa_1, \kappa_2, \kappa_3$): analgesia, dysphoria, diuresis, and psychotomimetic effects including hallucinations and delusions
 - a. This is believed to be very important in spinal analgesia.
3. Delta (δ_1, δ_2): analgesia and other unknown effects
 - a. Activation causes analgesia with minimal respiratory suppression.

Properties of the μ , κ , and δ Receptors

1. These receptors are located in the central nervous system and the peripheral nervous system.
2. The analgesic components of these receptors are the primary target of drug therapy, with the μ receptor being the most important target.
3. Approximately 82% of patients on opioid therapy report opioid-related side effects.
 - a. Constipation
 - b. Nausea and vomiting
 - c. Sedation
 - d. Pruritis
 - e. Respiratory depression
 - f. Muscle rigidity
 - g. Antitussive effect (This is usually a positive side effect.)

Appropriate Usage of Opioids

1. Opioids may be used for acute or chronic pain.
2. Opioids are very safe and effective pain killers with minimal short- or long-term side effects and tolerance profiles.
3. Opioids may be used in all patients, from pediatric to the elderly.
4. Opioids can be used in former addicts although alternative techniques to alleviate pain should be tried first.
 - a. See alternative techniques below.
 - b. If opioids are chosen for former addicts, then great caution and close follow-up care should be taken in this population.
 - i. Effective communication with the rehabilitation physician and the patient’s psychiatrist should be implemented.
 - ii. The treatment plan should be coordinated with these officials and the pain management services.
 - c. Opioids should not be avoided solely due to “former addict” status.

Algorithm for Determining the Appropriate Opioid Usage for a Patient

1. Consider all of the potential consequences if a patient has any of the expected side effects. Our primary goal is to do no

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harm. How will you treat any of the associated side effects? Develop a plan.

2. Does the patient have any previous opioid usage?
 - a. Use the patient's daily opioid dose as the baseline of opioid needs.
 - b. Start the acute pain regimen on top of that baseline dose.
 - c. Know the signs of withdrawal.
3. Decide on the appropriate starting dose as mentioned above based on the patient's needs and previous opioids daily dosage.
4. Use a pain scale scoring system to assess the adequacy of pain control.
5. Monitor for oversedation.
 - a. Patients with a history of opioid usage are usually more sedated with higher pain scores post-operatively.
6. Use multiple techniques in addition to opioids.
 - a. Regional/neuraxial blocks
 - b. Anti-convulsants
 - c. Sodium channel blockers
 - d. NMDA receptor antagonists
 - e. Non-steroidal anti-inflammatory drugs (NSAIDs)
7. Prepare for daily routine events that typically cause increased pain (i.e., dressing changes).
 - a. Compensate for these times with an appropriate breakthrough dose of a short-acting medication.
8. Be prepared to treat side effects, such as nausea and vomiting, and respiratory depression.
9. Patients with chronic opioid dependence should be monitored for compliance with therapy by using random urine toxicology screens.
10. Always believe the patient is in pain, but always be vigilant for any signs of complications, addiction, or manipulation.

Signs of Addiction/Diversion

The signs of addiction should be watched for in every patient. Often, as physicians we are prescribing powerful medications that, in the hands of the wrong person, can do great harm – not only to the patient but also to others who may obtain access to the drug inappropriately and to the community at large.

1. Multiple physicians are prescribing these patients pain medications.
2. The patients frequently report losing their prescriptions.
3. Requests for early refills
4. Unable to provide a urine toxicology screen when requested
5. The patient is requiring escalating doses of medication.
6. The patient inappropriately focuses on the narcotics during office visits.
7. The patient is not interested in a non-narcotic way of pain relief, such as interventional methods, NSAIDs, and psychiatric treatment.
8. Diversion: borrowing/selling opioid medications from/to others.

Withdrawal and the Medications Used for Its Treatment

Withdrawal can be a frightening experience for a patient coming off opioids. However, this experience is usually not life threatening.

Signs and Symptoms of Withdrawal

Physiological Symptoms

1. Increased lacrimation
2. Rhinorrhea
3. Sneezing
4. Nausea
5. Vomiting
6. Abdominal cramps
7. Diarrhea
8. Fever
9. Chills
10. Tremor
11. Tachycardia
12. Aches and pains, often in the joints
13. Elevated pain sensitivity
14. Elevated blood pressure
15. Piloerection
16. Pupillary dilation
17. Sweating
18. Dysphoria

Cognitive Symptoms

1. Suicidal ideation
2. Depression
3. Adrenal exhaustion
4. Adrenal fatigue
5. Spontaneous orgasm
6. Prolonged insomnia leading up to delirium
7. Auditory hallucinations
8. Visual hallucinations
9. Enhanced olfactory sense
10. Decreased sexual drive
11. Agitation
12. Panic disorder
13. Anxiety/irritability
14. Paranoia
15. Delusion
16. Opioid cravings

TKO: Only a physician licensed in drug rehabilitation may prescribe medications for the sole purpose of detoxification from opioids. For the oral boards it may be important to recognize these drugs and some of their general properties in case they are included in a given case.

Commonly Prescribed Withdrawal Medications

Agonists

1. Methadone
 - a. Trade name: Dolophine

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- b. Pharmacokinetics**
 - i.** Oral peak concentration is 1–7.5 hours.
 - ii.** Elimination half-life is 7–59 hours.
 - iii.** These times are shorter for pregnant patients.
 - iv.** Variable bioavailability with resultant different duration of action and dosing regimens
- c.** Used for drug detoxification, opioid abuse, neuropathic pain, and somatic pain.
- d.** The usual maintenance dose for abuse is 80–120 mg/day orally (PO).
- e.** The usual dose for pain treatment in an opioid-naïve patient can range from 2.5 to 10 mg PO every 3–6 hours as needed.
- f.** Renal and hepatic impairment may increase the accumulation of methadone and therefore dosing should be adjusted.
- g.** Black box warning: can cause QT prolongation and serious arrhythmias (torsades de pointes) in doses higher than 200 mg/day.
- h. Contraindications**
 - i.** Respiratory depression
 - ii.** Bronchial asthma
 - iii.** Hypercarbia
 - iv.** Paralytic ileus
- i. Precautions**
 - i.** Large daily doses
 - ii.** QT prolongation
 - iii.** Abuse
 - iv.** Electrolyte abnormalities may augment torsades de pointes.
 - (1)** Hypomagnesemia
 - (2)** Hypokalemia

Agonist/Antagonist

1. Buprenorphine

- a.** Buprenorphine is a semi-synthetic opiate with partial agonist and antagonist properties. Buprenorphine hydrochloride was first marketed in the 1980s as an analgesic, available generally as Buprenex in a 0.3 mg/mL injectable formulation.
- b.** Buprenorphine itself is a mixed agonist/antagonist. It blocks the activity of other opiates and induces withdrawal in opiate-dependent individuals who are currently physically dependent on another opiate. This is why users must wait until they are in withdrawal before beginning treatment with buprenorphine.
- c.** Elimination half-life: intravenous (IV) 1.2–7.2 hours and sublingual 37 hours
- d.** Used for opioid dependence and pain control.
- e.** This is a schedule III drug that is a partial μ -opioid receptor agonist and κ -opioid receptor antagonist.
 - i.** Since this drug is a partial μ -opioid receptor agonist, it makes reversing the overdose with naloxone difficult.

2. In 2002, the Food and Drug Administration (FDA) approved Suboxone and Subutex, high-dose sublingual pill preparations of buprenorphine for the treatment of opioid addiction.

a. Subutex has a greater attraction to the opiate receptors than other drugs such as heroin and methadone, and it binds more tightly to these receptors.

i. This drug is used most commonly in drug treatment programs to discourage further opioid abuse because it has a “ceiling effect,” meaning that only a certain, reduced level of pain control can be achieved and therefore a reduced feeling of reward for the patient taking this drug.

ii. This ceiling effect not only limits its maximal analgesic effect but it also limits its associated amount of respiratory depression.

iii. The dependence dose is 12 to 16 mg/day.

iv. Precautions include

(1) Compromised pulmonary function

(2) Head injury or increased intracranial pressure (ICP)

(3) Acute alcohol abuse

(4) Addison’s disease

(a) Opioid receptor actions at the level of the hypothalamus can affect hormone secretion on the hypothalamic-pituitary axis and cause further adrenal-cortical insufficiency leading to hypotension.

(5) Delirium tremens

(6) Infant risk with breast-feeding

(a) Decreased maternal milk production

(b) Buprenorphine is passed into the breast milk.

(7) Severe dysfunction of the hepatic, renal, or pulmonary systems. Ingestion of high doses of buprenorphine can result in severe hepatitis and acute renal failure.

(8) Severe opioid withdrawal may result with high doses.

v. Side effects

(1) Nausea and vomiting

(2) Dizziness and vertigo

(3) Sedation

(4) Arrhythmias

(5) An increase or decrease in blood pressure

(6) Dyspnea

(7) Respiratory depression

vi. Naloxone may not reverse the respiratory depression caused by buprenorphine.

b. Suboxone

i. A combination of buprenorphine and naloxone

ii. This combination is effective in reducing the misuse of this drug because, if crushed and injected (IV), the naloxone will precipitate withdrawal.

iii. If taken as directed (sublingual), then very little to no naloxone will be absorbed.

Alternative Analgesics:

Although implied, the following is specifically for chronic opioid users or those that have a previous abuse history. These patients have a more complicated background of pain and may require several extra therapies to get them through the perioperative period successfully.

1. Regional anesthesia
 - a. Especially useful in orthopedics and vascular procedures
 - b. Regional anesthesia is easy to deliver to the upper and lower extremities, and usually has good results.
 - c. May be contraindicated in a patient with impending neurological complications: compartment syndrome.
2. Neuraxial anesthesia
 - a. A spinal or epidural may be an option depending on the site of surgery.
 - b. The epidural may have the added advantage of being used in the post-operative period for pain relief.
 - c. Bupivacaine is commonly used for spinal anesthesia.
 - i. Longer duration of action compared with lidocaine
 - ii. Less likely to cause transient neurologic syndrome when compared to lidocaine
 - (1) Patients with transient neurologic syndrome typically have complaints of pain and a burning sensation in the lower extremities and buttock.
 - (2) The pain may last for several hours to days after the procedure has been completed.
 - (3) Occasionally the pain may last for years.
 - (4) Transient neurologic syndrome has been associated with
 - (a) Higher concentrations of local anesthetics (e.g., lidocaine 5%)
 - (b) Prone and lithotomy positions
 - d. Opioids and clonidine are effective in combination with bupivacaine or independently.
 - e. Lidocaine or bupivacaine can be used for epidural anesthesia.
 - i. A combination of the two may be advantageous as lidocaine will have quicker onset of anesthesia and bupivacaine will have a longer duration of action.
3. NSAIDs and acetaminophen (Tylenol)
 - a. This is highly effective pain relief for the majority of pain that people experience on a day-to-day basis; however, these should be used in combination with other techniques for peri-operative care.
4. Muscle relaxation
 - a. These may be good additions in certain situations that involve muscle spasticity as the cause of pain.
 - b. Methocarbamol (Robaxin), carisoprodol (Soma), baclofen, metaxalone (Skelaxin), and diazepam (Valium) are examples of medications that cause central muscle relaxation.
5. Anxiety
 - a. This can be the main source of pain problems.
 - b. Often this can stem from social issues or a personality disorder.

c. Psychiatry can help sort out some of these issues over time, and in the acute peri-operative period, benzodiazepine supplementation may be advantageous.

6. Neuropathic pain
 - a. Pain that is initiated or caused by a primary lesion or dysfunction in the nervous system
 - b. This may be considered if there is a burning component, numbness, or “electric shock” feeling to a patient’s pain.
 - c. Medications that are useful supplements for this pain include gabapentin (Neurontin) and pregabalin (Lyrica).
7. Sleep
 - a. It is very important for all of us to sleep well; however, for a patient in pain (chronically or acutely), this can be a major source of concern and sleeping poorly may accentuate the patient’s pain.
 - b. Lack of sleep should be treated aggressively.
 - c. A good pharmacological choice for the chronic pain patient would be amitriptyline. This medication can help relieve neuropathic pain, is not addictive, and is a sleep aid.
 - d. Other selective serotonin reuptake inhibitors (SSRIs)/antidepressants that have sedative properties may also be useful: i.e., trazadone.
 - e. Care should be taken to stay away from substances that have addictive potential, especially in patients with histories of addiction or who are at high risk for addiction.

**KO TREATMENT PLAN****Pre-operative**

1. Obtain a detailed history concerning previous drug use/abuse, length of use, treatment medications, and the length of the treatment.
2. Focus on a non-narcotic method of caring for the patient, if feasible.
3. Emphasize that if narcotics are used, it will only be in the peri-operative period and not for chronic therapy.
4. Be aware that some chronic users have decreased pain thresholds and increased opioid tolerance. Therefore, doses that would be effective normally may not be effective in this population.
5. If the patient is currently on an agonist/antagonist such as buprenorphine (Subutex), then she may experience a ceiling effect with narcotics peri-operatively. However, care should be taken when giving narcotics because side effects such as over-sedation and respiratory depression are still possibilities. Ideally, if opioids will be required in the peri-operative period, agonist/antagonists should be discontinued 1-2 weeks before any surgery and supplemented with another opioid agonist such as methadone. Therapy can be restarted after the surgery.
6. Choose a regional anesthetic for intra-operative and/or post-operative pain relief, if possible.

Intra-operative and Post-operative

1. Close monitoring of the respiratory rate if ventilation is not controlled may be valuable as this should give you an idea of the patient's anesthetic requirements and pain tolerance. Titrate the opioids to a goal respiratory rate of about 10 respirations per minute.
2. Monitoring for cardiac dysrhythmias is important with patients on high-dose methadone (>200 mg/day). This is especially important if other agents or conditions are present that could cause cardiac issues such as
 - a. Tricyclic antidepressants, secondary to the potential for an associated QT prolongation
 - b. Electrolyte deficiencies, particularly calcium, potassium, and magnesium, because of their associated torsades de pointes or QT prolongation
 - c. Lasix can lead to a further decrease in potassium concentrations, which can lead to dysrhythmias, and/or decreased magnesium, which can also lead to torsades de pointes.
 - d. Amiodarone and procainamide can also cause QT prolongation and worsen a patient's condition acutely.
 - e. Hypothyroidism can also cause QT prolongation.
3. Titrate short- to medium-acting medications such as fentanyl until intra-operative pain is under control. There is still a risk of over-sedation in patients who are not opioid naïve.
4. Consider using a low dose propofol/ketamine infusion during the case, such as propofol/ketamine in a 4:1 concentration. Opioids can often be avoided entirely in this scenario.

- a. Ketamine helps with pain control by N-methyl-D-aspartic acid antagonism, instead of activation at the opioid receptors.
- b. Ketamine will help attenuate the decrease in blood pressure caused by propofol.
- c. Propofol will be useful as an anesthetic agent and as an anti-emetic.
5. Titrate in longer acting agents at the end of the case for continued pain coverage in the post-anesthesia care unit (PACU) and afterward.
6. Consider an early regional block during the immediate post-operative period unless there are contraindications.
7. Keep in mind that, especially in this population, it is better to prevent severe pain than it is to treat it after the patient is very uncomfortable. Be proactive.

BIBLIOGRAPHY

1. Waldman SD. *Pain Management, Vol. 1*. Philadelphia: Saunders Elsevier, 2007, pp 939–61 and 997–1001.
2. Benzon HT, Raja SN, Molloy RE, et al. *Essentials of Pain Medicine and Regional Anesthesia*, 2nd ed. Philadelphia: Elsevier, 2005.
3. Thomson Micromedex Healthcare Series. [http:// www.micromedex.dk/](http://www.micromedex.dk/).
4. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 353–79.

89. Peri-Operative Temperature Regulation^{1,2,3,4,5,6,7}

Alma Hoxha, MD

TEMPERATURE REGULATION

Why Measure a Patient's Temperature during the Peri-operative Period?⁵

1. Typical clinical doses of all general anesthetics reduce the threshold for vasoconstriction to 33–35°C.
2. General anesthetics increase the threshold for sweating and active vasodilatation by 1°C.
 - a. Anesthesia profoundly alters the thermoregulatory system, markedly reducing cold-response thresholds while slightly increasing warm-response thresholds.
 - b. This results in an approximately 4°C range of core temperatures not triggering thermoregulatory deficiency intra-operatively.

Temperature Monitoring and Thermal Management Guidelines

1. The patient's core temperature should be measured if he or she will have a general anesthetic for longer than 20–30 minutes.
2. The patient's temperature should also be measured during any regional anesthetic when changes in temperature are intended, anticipated, or suspected.
 - a. Mechanism for hypothermia under regional anesthesia⁷
 - i. Spinal anesthesia leads to an internal redistribution of heat from the core to the peripheral compartment.
 - ii. Loss of thermoregulatory vasoconstriction below the level of the spinal block
 - (I) There is increased heat loss from body surfaces.
 - iii. Altered thermoregulation under spinal anesthesia is characterized by a decrease in vasoconstriction and shivering threshold and a slight increase in the sweating threshold.
3. Unless hypothermia is specifically indicated (e.g., for protection against cerebral ischemia), effort should be made to maintain a normothermic intra-operative core temperature.
 - a. Normal body temperature is 36.7°C to 37.0°C ± 0.2°C to 0.4°C.
4. Temperature should always be monitored in any surgical patient who
 - a. Is receiving blood products
 - b. Has a pre-existing fever

c. Has an overt infection

d. Has autonomic instability

5. Continuous monitoring of temperature is recommended; however, 15 minute intervals are acceptable for most patients.

Temperature Monitoring Sites⁵

1. Core
 - a. Pulmonary artery
 - b. Distal esophagus
 - c. Tympanic membrane
 - d. Nasopharynx
2. Intermediate
 - a. Mouth
 - b. Axilla
 - c. Bladder
 - d. Rectum
 - e. Forehead skin surface: 1–2°C less than the core temperature

HYPERTHERMIA^{1,2}

SAMPLE CASE

A 63-year-old male with three vessel disease is brought to the operating room for a coronary artery bypass graft (CABG) surgery. The patient has a history of hypertension, gastroesophageal reflux diseases, diabetes mellitus, and hyperlipidemia. He weighs 113 kg and is 169 cm tall. His pre-operative blood pressure is 130/66 mm Hg, heart rate is 72, oxygen saturation is 98%, respiratory rate is 18, and temperature is 36.7°C. The patient has no prior surgical history. He is induced with etomidate and succinylcholine and intubated with a #8 endotracheal tube. The surgeon plans to perform this as an off-pump CABG. Anesthesia is maintained with isoflurane, fentanyl, and a vecuronium drip. During the procedure, your resident notes the patient to have an incremental increase in his temperature up to 38.3°C. Is this a concern for you? What are the potential causes of this increase in temperature? What would you do?

CLINICAL ISSUES

TKO: Hypothermia is by far the most common peri-anesthetic thermal perturbation. However, hyperthermia is more dangerous than a comparable degree of hypothermia.

Definitions

1. Hyperthermia: Core body temperature exceeds normal values (>37.4°C).
 - a. The body produces or absorbs more heat than it can dissipate.
 - b. The body temperature is raised without the consent of the body's heat control centers.
 - c. Body temperatures greater than 40°C can be life threatening.
 - d. At 41°C brain death begins to occur.
 - e. At about 45°C, death is imminent.
 - f. Internal temperatures above 50°C cause muscle rigidity and death.
2. Passive intra-operative hyperthermia:
 - a. Excessive patient heating
 - b. Most common in infants and children
 - c. Frequently seen when effective active warming is used without adequate body temperature monitoring
3. Malignant hyperthermia (MH)
 - a. Results secondary to an enormous increase in metabolic heat produced by both internal organs and skeletal muscle in response to a volatile anesthetic or succinylcholine
 - b. Central thermoregulation presumably remains intact during an acute crisis. (Please see Chapter 84 on MH for further information.)
4. Fever: Regulated increase in the core temperature targeted by the thermoregulatory system
 - a. Fever results when endogenous pyrogens increase the thermoregulatory target temperature ("set point").
 - b. The body sets its core temperature higher: anterior hypothalamus.
 - c. Fever is relatively rare during general anesthesia.
 - i. Volatile anesthetics and opioids inhibit the expression of fever.

Differential Diagnosis of Hyperthermia

1. Iatrogenic
 - a. Increased room temperature
 - b. Warming devices
 - c. Airway humidifiers and warmers
 - d. Excessive re-warming after cardiopulmonary bypass
2. Infectious
 - a. Pre-operative fever associated with an upper respiratory tract infection or condition related to the surgical indication
 - b. Sepsis

- c. Bacteremia or sepsis associated with surgical manipulation (i.e., oral surgery)
- d. Infusion of blood components with infectious contamination
3. Pulmonary
 - a. Aspiration pneumonia
 - b. Atelectasis
 - c. Deep venous thrombosis or pulmonary embolus
4. Metabolic
 - a. Pheochromocytoma
 - b. Thyrotoxicosis or thyroid storm
 - c. Adrenal insufficiency
5. Central nervous system
 - a. Status epilepticus
 - b. Hypothalamic pathology
 - c. Parkinson's disease
6. Drug induced
 - a. Malignant hyperthermia
 - b. Neuroleptic malignant syndrome
 - c. Anticholinergic effect
 - d. Cocaine
 - e. Tricyclic antidepressants
 - f. Antibiotic-induced drug fever
 - g. Monoamine oxidase inhibitors interacting with opioids, especially meperidine
7. Miscellaneous
 - a. Monitoring error
 - b. Connective tissue disease
 - c. Hematoma
 - d. Transfusion reactions

Signs and Symptoms of Hyperthermia

1. Skin
 - a. Perspiration
 - i. Draws heat out from the body.
 - ii. Allows the heat to be carried off by evaporation.
 - iii. When the body is no longer capable of sweating secondary to dehydration, the patient's core temperature will rise rapidly.
 - b. The skin initially becomes red as the blood vessels dilate in an attempt to increase heat dissipation.
 - c. Once the patient's blood pressure begins to drop, cutaneous blood vessels will begin to contract and the skin may start to look pale and bluish. (This is a bad sign.)
2. Cardiac
 - a. Blood pressure may drop significantly from dehydration.
 - i. Syncope or dizziness can be seen in an awake patient.
 - b. Increased cardiac output
 - c. Increased heart rate
 - d. Increased metabolic rate and therefore increased oxygen consumption
 - i. Myocardial ischemia occurs if the cardiac demand exceeds the oxygen delivery.

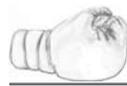
89. Peri-Operative Temperature Regulation

- e. Decreased intravascular volume and pre-load due to the hyperthermia associated perspiration and vasodilatation
- 3. Respiratory
 - a. Increased respiratory rate
 - b. If the cellular demand exceeds the oxygen delivery, acidosis occurs.
 - c. Rightward shift of the oxygenation hemoglobin dissociation curve
- 4. Neurologic
 - a. If awake, hyperthermic patients may become confused and hostile.
 - i. Headache
 - ii. Appear intoxicated
 - iii. Seizure
 - iv. Temporary blindness
 - v. Unconsciousness
 - b. Cerebral blood flow increases by 5–7% for each degree celsius increase in body temperature.
 - c. Reduces the latency of the somatosensory evoked potentials
- 5. Electrolytes
 - a. Electrolyte abnormalities occur due to perspiration and the associated nausea and vomiting.
 - i. Loss of electrolytes
 - ii. Loss of free water

Treatment of Hyperthermia

1. Treatment of hyperthermia depends on the etiology of the elevated temperature with the critical distinction between fever and other causes of hyperthermia.
2. Review patient's history and examine the patient for clues to the possible causes of hyperthermia.
 - a. Medications
 - b. Surgical procedure
 - c. Peri-operative complications
 - d. Iatrogenic
3. First line of treatment: treat the underlying cause.
 - a. Passive cooling
 - i. Remove any external warming devices: heating blankets, pads, fluid warmers, etc.
 - ii. Lower the room temperature.
 - iii. Remove excess blankets, clothing, or surgical drapes.
 - b. Stop any blood transfusions: transfusion reaction.
 - c. Stop the volatile anesthetic and change to a total intravenous anesthetic if the patient has signs of hypermetabolism and you are concerned about malignant hyperthermia.
 - d. Urine culture
 - e. Chest X-ray
4. Second line of treatment: administration of the antipyretic medications (This step is not always possible during the surgical procedure.)
 - a. Acetaminophen
 - b. Aspirin
 - c. Non-steroidal anti-inflammatory drugs (NSAIDs)

5. Third line of treatment: active cooling should be considered when temperature exceeds 39°C or the patient is believed to be hemodynamically unstable secondary to temperature perturbation.
 - a. Turn off warming devices and blow cool air over the patient.
 - b. Cooled intravenous fluids
 - c. Ice packs on the torso, head, neck, and groin
 - d. A fan can be used to aid evaporation.
 - e. Flush cool water into the nasogastric tube and Foley catheter.
 - f. In desperate situations, use of cardiopulmonary bypass provides rapid cooling.
 - g. A hyperthermia vest can be placed if available.



KO TREATMENT PLAN

Pre-operative

1. Careful pre-operative history and physical examination
 - a. History of malignant hyperthermia
 - b. Known sepsis
 - c. Pheochromocytoma
 - d. Thyroid dysfunction
2. What is the patient's pre-operative temperature?
3. The sample patient does not have an elevated pre-operative temperature, and he has no history of any diseases associated with hyperthermia.

Intra-operative

1. Iatrogenic hyperthermia is prevented by monitoring the patient's core temperature after the establishment of a baseline temperature and using a blanket cooling or warming device as necessary.
2. If hyperthermia occurs, one should review the recent intra-operative events:
 - a. Blood transfusion
 - b. Malignant hyperthermia-triggering agents: volatile anesthetics and succinylcholine
 - c. Where is the site of the surgery?
 - d. Recent medications given
3. Differential diagnosis for the sample patient

TKO: On the oral board exam, you should be able to list this differential quickly and efficiently, and in the order of most to least likely. The oral board examiners want to know that you have a complete differential in your mind, but they also really want to know what you think is going on, and that you have a plan to treat it quickly.

- a. Iatrogenic
 - i. Increased room temperature
 - ii. Warming devices

- iii. Airway humidifiers and warmers
 - iv. Excessive re-warming after cardiopulmonary bypass (if the case was performed on-pump).
 - b. Drug induced
 - i. Malignant hyperthermia
 - ii. Neuroleptic malignant syndrome
 - iii. Anticholinergic effect
 - iv. Antibiotic-induced drug fever
 - v. Monoamine oxidase inhibitors interacting with opioids, especially meperidine
 - c. Pulmonary
 - i. Atelectasis
 - ii. Deep venous thrombosis or pulmonary embolus
 - iii. Aspiration pneumonia
 - d. Miscellaneous
 - i. Monitoring error
 - e. Infectious
 - i. Sepsis
 - (1) Low likelihood of occurring acutely, but worth keeping on your extended differential
4. Diagnosis
- a. Review the patient's medical history.
 - b. Verify the temperature reading and check all other vital signs.
 - i. Look especially for changes in the end-tidal CO₂.
 - (1) Potential clue to malignant hyperthermia or a pulmonary embolism
 - ii. Look for signs of hypermetabolism.
 - (1) Tachycardia
 - (2) Acidosis
 - (3) Hypercarbia
 - (4) Hypoxemia
 - (5) Hyperthermia
 - c. Review all of the pre-operative and intra-operative medications that the patient has received.
 - d. Send an arterial blood gas immediately.
 - e. Chest X-ray
 - f. Culture any possible infected areas.
 - i. Wound
 - ii. Urine
 - iii. Sputum
 - (1) This will not give you an immediate answer, but it can be used for diagnosis if other possibilities are ruled out.

Post-operative

1. Although some mild hyperthermia or fever may be beneficial with an infection, the potential physiologic effects on the heart and brain can be detrimental, especially in the elderly.
2. Hyperthermia should be treated aggressively in at-risk patients.

HYPOTHERMIA^{3,4,6}

SAMPLE CASE

A 67-year-old male is brought to the operating room for an emergency surgery to repair his perforated esophagus. The patient was found lying in bed by his family members. He was cold and diaphoretic. His chest X-ray showed capsules in his stomach. The patient has a history of hypertension, hypothyroidism, gastroesophageal reflux disease, and depression. Vital signs: blood pressure 96/55 mm Hg, heart rate 61, respiratory rate 20, and temperature 33.8°C. What do you think about the patient's body temperature? Is this a concern for you? How will the patient's hypothermia affect your anesthetic? Does it affect the muscle relaxant reversal? What are the effects of hypothermia on cardiovascular function? What are the effects of hypothermia on coagulation? What physiologic effects are you concerned about in a hypothermic patient?

CLINICAL ISSUES

Definition

1. Hypothermia: defined as a core body temperature less than 36°C
 - a. The thermoregulatory system in an awake patient usually maintains a core body temperature within 0.2°C of normal (37°C).
 - b. Usually caused by prolonged exposure to low temperatures
2. Mild hypothermia: about 1-2°C below normal
 - a. Triples the incidence of a morbid cardiac outcome.
 - b. Triples the incidence of wound infections.
 - c. Directly impairs immune function.
 - i. Impairs resistance to some bacteria in vitro.
 - d. Prolongs hospitalization by 20%.
 - e. Significantly increases surgical blood loss and the need for allogenic transfusion.
 - f. Can lead to cold-induced direct platelet dysfunction.

TKO: Hypothermia during anesthesia by far is the most common peri-operative thermal disturbance. Hypothermia results from a combination of anesthetic-impaired thermoregulation and exposure to the cold operating room environment.

Clinical Hypothermia

1. Mild: 32-35°C
2. Moderate: 26-31°C
3. Deep: 20-25°C
4. Profound: 14-19°C

Incidence

1. More common than hyperthermia
2. Less dangerous than hyperthermia

89. Peri-Operative Temperature Regulation

3. The core temperature usually decreases by 0.5–1.5°C in the first 30 minutes after the induction of anesthesia.
 - a. Internal redistribution of heat

General Causes of Hypothermia

1. Decreased heat production
2. Decreased metabolic rate
3. Increased heat loss
4. Heat transfer
 - a. Radiation
 - b. Convection
 - c. Conduction
 - d. Evaporation

Mechanisms of Heat Loss

1. Radiation loss
 - a. Largest component of heat loss
 - b. Occurs in the form of infrared electromagnetic waves
2. Convection loss
 - a. Secondary to “wind chill”
 - b. Transfer of heat between the body and the air
 - c. Heat rises from the skin which creates a convection current.
3. Conduction loss
 - a. Due to direct skin contact
 - b. Exchange of heat between objects in direct contact
 - c. Conductors pull heat away from the body.
 - i. Water
 - ii. Metals
4. Evaporative loss
 - a. Usually less significant
 - b. Important with premature babies
 - c. Conversion of water from a liquid to a gas requires energy in the form of heat that is taken from the body.

Differential Diagnosis of Hypothermia

1. Environmental
 - a. Wind chill
 - b. Cold water immersion
2. Impaired thermoregulation
 - a. Extremes of age: neonates, elderly
 - b. Prolonged immobilization
 - c. Drugs
 - i. Alcohol intoxication
 - ii. Central nervous system depressants
 - iii. Drug overdose
3. Medical conditions
 - a. Hypothyroidism
 - b. Large body surface area burns
 - c. Malnutrition
 - d. Hypoglycemia
 - e. Hypothalamic stroke or tumor
 - f. Unconsciousness

4. Iatrogenic causes of hypothermia
 - a. Prolonged anesthesia and surgery without proper monitoring and regulation
 - b. Prolonged cardiopulmonary resuscitation
 - c. Blood or blood product transfusions (especially if the blood products are given rapidly and not warmed properly)
 - d. Large-volume fluid resuscitation (especially without a proper warming device)

Physiologic Effects of 33°C

1. Hypertension, tachycardia, and increased cardiac output: initial response to hypothermia
 - a. Followed by cardiovascular collapse at colder temperatures
2. Increased oxygen consumption
3. Hyperventilation
4. Shivering
5. Piloerection
6. Muscle miscoordination
7. Confusion

Physiologic Effects of 30°C

1. Metabolism is reduced 50% at 30°C.
2. Significant neural and organ depression
 - a. Cold narcosis
3. Loss of consciousness

Physiologic Effects of 28°C

1. Hyperglycemia: secondary to a plasma fall in insulin and increased catecholamines from the stress of hypothermia
2. Hypovolemia: secondary to cold diuresis
 - a. Physiologic increase in the hematocrit
 - b. Hypotension
3. Cardiac arrhythmias
 - a. Bradycardia
 - i. Decreased cardiac output secondary to a decreased heart rate
 - b. Nodal rhythm
 - c. Premature ventricular contractions
 - d. Atrio-ventricular block
 - e. Ventricular fibrillation
4. EKG changes
 - a. Prolonged PR interval
 - b. Widening of the QRS complex
 - c. Prolongation of the QT interval
 - d. ST segment elevation
 - e. Wave rising steeply from the S wave
 - f. EKG shows a J wave.
5. Oxy-hemoglobin dissociation curve shifts to the left.
6. Oxygen consumption is decreased unless shivering is present; then it is increased.
7. Thrombocytopenia

89. Peri-Operative Temperature Regulation

8. Metabolic acidosis
9. Prolonged drug metabolism

Physiologic Effects of <25°C

1. Metabolism is reduced 60% at 25°C.
2. Ventricular fibrillation
3. Shivering ceases
4. Cessation of spontaneous respiration
5. Acid-base disturbances, unless adequate ventilation is maintained

Physiologic Effects of 20°C

1. Prolonged bleeding and clotting times
 - a. Usually reversible with re-warming

Organ Function during Hypothermia

1. Metabolism
 - a. Hypothermia causes a complex array of physiologic changes.
 - b. Abnormal response to drug administration
 - i. Drug responses are often exaggerated.
 - ii. The minimum alveolar concentration (MAC) for a volatile anesthetic decreases by 5–7% per °C fall in body temperature.
 - c. Hypothermia decreases the whole body metabolic rate by approximately 8% per °C.
 - d. Reduced metabolism of carbohydrates with hypothermia
 - i. Hyperglycemia is usually proportional to cooling.
2. Brain
 - a. Cerebral blood flow (CBF) decreases in proportion to the metabolic rate during hypothermia because of an autoregulatory increase in cerebrovascular resistance.
 - b. Cerebral function is well maintained until the patient's core temperature falls below 33 °C.
3. Heart
 - a. Initial increase in heart rate, stroke volume, cardiac output, and blood pressure secondary to sympathetic stimulation
 - i. If the sympathetic response is blunted by anesthesia or other medications, there is a proportional decrease in cardiac output, heart rate, and mean arterial pressure, with little change in stroke volume, and an increase in peripheral vascular resistance.
 - ii. Decrease in the heart rate, cardiac output, and blood pressure with extremes of hypothermia
 - b. Increase in contractility
 - c. Well-maintained stroke volume
 - d. At 28°C the SA node pacing becomes erratic and ventricular irritability increases.
 - e. At 25°C, ventricular fibrillation usually occurs.
 - i. Electrical defibrillation is generally ineffective at these temperatures.

4. Kidney
 - a. Renal blood flow decreases.
 - b. Renal vascular resistance increases.
 - c. Inhibition of tubular reabsorption maintains normal urinary volume.
 - d. As the temperature decreases, reabsorption of sodium and potassium is progressively inhibited and an anti-diuretic hormone mediated "cold diuresis" results.
5. Respiratory
 - a. Increase in oxygen consumption with an associated increase in respiratory rate with the initial fall in temperature
 - b. Ultimately, hypothermia leads to respiratory depression.
 - c. There is a 6% fall in oxygen consumption per °C fall in body temperature as hypothermia progresses.
 - d. Respiratory strength is decreased at temperatures less than 33°C, but ventilatory CO₂ response is minimally affected.
 - e. Increased physiologic and respiratory dead space
6. Liver
 - a. Hepatic blood flow and function decrease.
 - i. Significantly inhibits the metabolism of some drugs.
 - (1) Propranolol
 - (2) Verapamil
 - (3) Prolongs elimination of nondepolarized muscle relaxants.
7. Tissues
 - a. The tolerance to tissue hypoxemia is increased during hypothermia.
8. Blood
 - a. Rise in hematocrit
 - b. Increase in viscosity
 - c. Leukopenia
 - d. Thrombocytopenia

Treatment of Hypothermia

1. Surface re-warming
 - a. Warmed blankets
 - b. Forced air heating blankets
2. Heated inspired gases
3. Fluid warmer
4. Warm the operating room temperature.
5. Warm body cavity lavage: bladder, stomach, chest, and abdominal cavity
6. An arterial line should be placed for continuous pressure monitoring and frequent blood gas determinations.
7. A pulmonary artery catheter may be helpful to follow cardiac output, but there is a risk of cardiac arrhythmia due to its placement.
8. Bladder catheterization
 - a. Monitoring urine output reflects the adequacy of intravascular volume and organ perfusion.

9. Continuous core temperature monitoring
10. Warm the patient slowly.

TKO: A patient is not dead until he or she is warm and dead!



KO TREATMENT PLAN

Pre-operative

The management and prevention of hypothermia are inseparable and include the following:

1. Pre-operative skin warming
2. Adjusted ambient temperature in the operating room
3. Intra-operative temperature monitoring
4. Heated and humidified anesthesia circuits
5. Forced-air warming blankets and other devices
6. Warmed intravenous fluids and blood products
7. Prevention and treatment of post-operative shivering
 - a. Meperidine: 25 mg IV (treatment)
 - b. Clonidine: 0.15 mg IV (treatment)
 - c. Tramadol: 1 mg/kg IV (treatment and prophylaxis)
 - d. Ondansetron: 8 mg IV (prophylaxis)
8. Anticipation and treatment of re-warming vasodilatation

Intra-operative

1. During general anesthesia there is an initial rapid decrease in core temperature (0.5–1.5°C during the first hour) followed by a slow linear reduction.
2. The core temperature stabilizes and subsequently remains unchanged after 3–4 hours.
 - a. The plateau phase is associated with peripheral thermoregulatory vasoconstriction triggered by core temperatures of 33–35°C.
3. Treatment of intra-operative hypothermia
 - a. Preventing redistribution hypothermia
 - i. Active pre-warming for as little as 30 minutes helps prevent redistribution hypothermia.
 - b. Airway heating and humidification
 - i. Even though airway heating and humidification are effective in infants/children, cutaneous warming still remains more effective and transfers more than 10 times as much heat as airway heating.
 - c. Intravenous fluids
 - i. One unit of blood or 1L of crystalloid solution administered at room temperature will decrease the mean body temperature by approximately 0.25°C.
 - ii. Fluid warmers minimize this loss and should be used when large amounts of intravenous fluid or boluses are administered.
 - d. Cutaneous warming
 - i. Room temperature 23°C for adults and 26°C for children is needed to maintain normothermia.
 - ii. Operating room temperature is the most critical factor influencing heat loss because it determines the rate

at which metabolic heat is lost by radiation and convection from the skin and by evaporation from within surgical incisions.

iii. The easiest way of decreasing cutaneous heat loss is to apply passive insulation to the skin surface.

(1) Insulators readily available in most operating rooms include cotton blankets, surgical drapes, plastic sheeting, and reflective composites.

(2) A single layer of each reduces heat loss approximately 30%, with no clinical difference noted among the insulation types.

iv. Cutaneous heat loss is roughly proportional to the surface area throughout the body.

(1) 90% of metabolic heat is lost through the skin surface.

(2) Only cutaneous warming will transfer sufficient heat to prevent hypothermia.

v. For intra-operative use, circulating water and forced-air cutaneous heating devices should be considered.

4. Predisposition of hypothermia during general and regional anesthesia

a. Heat loss is common in all patients during general anesthesia because anesthetics alter thermoregulation, prevent shivering, and produce peripheral vasodilation.

i. Volatile anesthetics impair the thermoregulatory center in the hypothalamus and also have direct vasodilatory properties.

ii. Opioids reduce the vasoconstrictive mechanism of heat conservation.

iii. Barbiturates cause peripheral vasodilation.

iv. Muscle relaxants reduce muscle tone and prevent shivering thermogenesis.

b. Regional anesthesia also may produce sympathetic blockade, muscle relaxation, and sensory blockade of thermal receptors, thus inhibiting compensatory responses.

5. Attention should be paid to the effect of hypothermia on the patient's cardiac physiology.

a. Vasoconstriction causes hypoperfusion and hypoxia.

b. Shivering increases oxygen consumption by up to 300%.

i. Results in increased myocardial oxygen consumption

c. As hypothermia worsens, the heart rate, cardiac output, and oxygen consumption decrease.

d. Later symptoms include ventricular arrhythmias and myocardial depression.

6. Hypothermia leads to decreased platelet function, visceral sequestration of platelets, and platelet aggregation resulting in thrombocytopenia.

7. Drug effects are prolonged by decreased hepatic blood flow, metabolism, renal blood flow, and clearance.

a. Protein binding increases as body temperature decreases.

b. The MAC of inhalation agents is decreased about 5–7% per °C fall in core temperature.

- c. Hypothermia may prolong the duration of neuromuscular blocking agents because of decreased metabolism.

Post-operative

1. Hypothermia delays discharge from the post-anesthetic care unit and may prolong the need for mechanical ventilation.
 - a. Delayed emergence from volatile anesthetics (decreased MAC) and prolonged neuromuscular blockade
2. Shivering (see the pre-operative section for treatment).

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1576–7, 1580–4, 1589–93.
2. Atlee JL. *Complications in Anesthesia*, 2nd ed. Philadelphia: Saunders Elsevier Health Sciences, 2006, pp 423–5.
3. Atlee JL. *Complications in Anesthesia*, 2nd ed. Philadelphia: Saunders Elsevier Health Sciences, 2006, pp 419–22.
4. Faust RJ, Cucchiara RF. *Anesthesiology Review*, 3rd ed. Philadelphia: Churchill Livingstone, 2001, pp 91–2.
5. www.mhaus.org.
6. Wong KC. Physiology and pharmacology of hypothermia. *West J Med*, 1983;138(2):227–32.
7. Ben-David B, Solomon E, Levin H. Spinal anesthesia, hypothermia, and sedation: A case of resection with forced-air warming. *Anesth Analg*, 1997;85:1357–8.

90. Myasthenia Gravis^{1,2,3}

Ryan J. Gunselman, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 45-year-old male is scheduled for a resection of a tumor in his colon. He has a history of myasthenia gravis. He takes pyridostigmine 360 mg/day. What are your anesthetic concerns with this patient? How would you assess the adequacy of his therapy? How would you manage his pre-operative medications? Should he have pulmonary function tests (PFTs) pre-operatively? Would a spinal/epidural be a good choice for this case? Will the patient need or not need muscle relaxant during this case? Would succinylcholine be a good choice? If you used a non-depolarizing muscle relaxant (NDMR), would you reverse it at the end of the case?

CLINICAL ISSUES

Definition of Myasthenia Gravis (MG)

1. Autoimmune disease in which nicotinic acetylcholinesterase receptors (nAChRs) are attacked by IgG antibodies
2. Functionally, MG will decrease the available number of nAChRs, causing muscle weakness and fatigue.
3. It is the most common disorder of neuromuscular transmission.

Incidence

1. 0.25 to 2 per 100,000
2. Peak of onset

- a. Females: second and third decades of life
- b. Males: sixth to eighth decades of life

Classification

1. Seropositive: antibodies to nAChRs found: 80% of MG patients
2. Seronegative: no antibodies to nAChRs: 20% of MG patients
 - a. 70% of seronegative patients have antibodies against muscle-specific tyrosine kinase, an enzyme mediating the arrangement of nAChRs.

Anatomical Origin

1. Thymus:
 - a. Hyperplasia found with production of antibodies to nAChRs
 - b. Thymectomy, however, does not cure MG: the patient must have the production of antibodies in other locations as well.

Clinical Signs

1. Two major types of symptoms
 - a. Ocular
 - i. Weakness of the eyelids and extra-ocular muscles: ptosis and diplopia
 - b. Generalized (bulbar)

90. Myasthenia Gravis

- i.** Weakness of the face, limbs, and respiratory muscles in addition to the ocular muscles
 - (1)** Weakness with chewing
 - (2)** Expressionless look
 - (3)** Head drop
 - (4)** Respiratory insufficiency
- ii.** The most common complaint is fluctuating weakness in the muscles.
- c.** Symptoms usually are worse later in the day or after exercise and are often transient early in the disease process.
- d.** 50% of patients present with ocular symptoms.

Differential Diagnosis

1. Generalized fatigue
2. Graves' disease
3. Amyotrophic lateral sclerosis (ALS)
4. Lambert-Eaton myasthenic syndrome
5. Congenital myasthenic syndrome
6. Penicillamine-induced myasthenia
7. Botulism
8. Statin medication side effects

Diagnostic Testing

1. Primarily based on the patient's clinical symptoms
2. Tensilon test
 - a.** Can be used as a screening test at the bedside in patients with suspected MG.
 - b.** A small dose of a fast-onset acetylcholinesterase inhibitor will increase the Ach in the neuromuscular junction and should improve muscle weakness in patients with MG.
 - i.** Classically, a 10mg dose of edrophonium is used.
3. Ice pack test
 - a.** Used in patients when the muscarinic effects of edrophonium may be deleterious (i.e., patients with heart block, bradycardia, etc.)
 - b.** In patients with ptosis, cooling the eyelid often improves neuromuscular transmission.
4. Serologic testing for AChR antibodies
5. Electrophysiologic testing
 - a.** Repetitive nerve stimulation (RNS)
 - b.** EMG studies

Treatment

1. Usually starts with the use of anticholinesterase therapy.
2. Steroids and other immunosuppressants
3. Plasmapheresis
4. Intravenous immunoglobulin
5. Thymectomy

Anesthetic Implications

1. Muscle relaxants
 - a.** Patients with MG who are not receiving anticholinesterase treatment
 - i.** Depolarizing muscle relaxants
 - (1)** Due to an overall decrease in nAChRs, patients with MG are more resistant to succinylcholine.
 - (2)** ED95 of succinylcholine in a patient with MG is 2.6 times that of a patient without MG.
 - ii.** Non-depolarizing muscle relaxants (NDMR)
 - (1)** More sensitive to NDMRs
 - (2)** Avoid long-acting agents (i.e., pancuronium).
 - (3)** Sensitivity, however, will vary.
 - b.** Patients receiving treatment for MG with anticholinesterase therapy
 - i.** Depolarizing muscle relaxant
 - (1)** More sensitive to succinylcholine
 - (2)** May increase the duration of action of succinylcholine
 - ii.** NDMR
 - (1)** More resistant to NDMRs
 - (2)** Decreased sensitivity to NDMRs compared to an MG patient who is not receiving anticholinesterase treatment.
 - (3)** Remember that since these patients are prone to respiratory insufficiency, the smallest dose of NDMR that provides relaxation should be used despite the associated resistance.
- c.** Reversal of NDMRs
 - i.** May result in a cholinergic crisis due to an excess of Ach in the MG patient treated with anticholinesterase therapy.
 - ii.** May be ineffective in patients who are receiving anticholinesterase treatment because much of the anticholinesterase inhibition already exists.
 - iii.** Many clinicians will avoid neuromuscular blockade completely, if possible.
 - iv.** If muscle relaxation is necessary, clinicians often allow the patient to recover from blockade without reversal while maintaining mechanical ventilation.
 - (1)** Atracurium and cisatracurium are often preferred neuromuscular-blocking drugs for the MG patient because the patient's metabolism can obviate the need for reversal.
 - v.** Train-of-four monitoring can be used to assess the degree of blockade
2. Inhaled anesthetics
 - a.** In normal patients, volatile agents provide some degree of muscle relaxation; in MG patients this may be more pronounced.
3. Regional anesthesia
 - a.** Epidural/spinal anesthesia may be used, but at lower doses.
 - i.** Caution must be used in patients with respiratory insufficiency.

91. Obesity^{1,2,3,4}

Jeffrey A. Brown, DO and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 48-year-old, 223 kg female is scheduled for a laparoscopic cholecystectomy. Her medical history is significant for cholelithiasis, osteoarthritis, hypertension, type II diabetes mellitus, and gastroesophageal reflux disease. She uses a continuous positive airway pressure (CPAP) machine at home. What are your concerns? How would you approach this patient? Are there any tests you would like to have before surgery? How would you induce anesthesia in this patient? What would you use for maintenance of anesthesia?

CLINICAL ISSUES

Now more than ever, the United States is obese. These patients represent a challenge from a medical standpoint as well as an increased risk of a difficult airway. The wise anesthesiologist will know how to manage these patients effectively.

Definitions

1. Overweight: body mass index (BMI) ≥ 24 kg/m²
2. Obesity: BMI ≥ 30 kg/m²
3. Extreme (“morbid”) obesity: BMI ≥ 40 kg/m²

Physiologic Effects of Obesity

1. Cardiovascular
 - a. Due to their often sedentary nature, obese patients may not complain of angina or dyspnea with exertion.
 - b. Careful history taking may prompt further testing such as EKG, echocardiography, or cardiac stress testing.
 - c. Cardiac output (CO) is increased 0.1 L for every 10 kg of extra body fat.
 - d. Chronic hypoxemia may lead to pulmonary hypertension and later cor pulmonale.
 - e. Increased absolute blood volume
 - f. Increased risk of arrhythmias
 - i. Cardiac hypertrophy
 - ii. Hypoxemia
 - iii. Fatty infiltrate of the cardiac conduction system
 - iv. Coronary artery disease
 - v. Associated sleep apnea

2. Respiratory
 - a. Functional residual capacity (FRC) is reduced, resulting in rapid hypoxemia during induction of general anesthesia.
 - b. Increased oxygen consumption
 - c. Patients should be kept in a semi-recumbent state as long as possible prior to induction.
 - d. Ventilation with 100% oxygen is mandatory.
 - i. At least four vital capacity breaths should be sufficient for denitrogenation.
 - e. At least 5% of obese patients suffer from obstructive sleep apnea (OSA).

- i. The STOP questionnaire:

- (1) Easy, concise tool for screening for OSA

- (2) Consists of four questions

S: Do you snore loudly?

T: Do you often feel tired or sleepy during the daytime?

O: Has anyone ever observed you stop breathing during sleep?

P: Do you have or are you being treated for high blood pressure?

If patients answer “yes” to two or more of these questions, they are considered at high risk for OSA.

The sensitivity increases greatly when combined with other known risk factors for OSA: high BMI, age over 50, large neck circumference, and male gender.

- f. Patients who use CPAP at home should bring their machines with them to the hospital on the morning of surgery.
3. Airway
 - a. Neck circumference alone is an independent predictor of a difficult airway.
 - i. Neck circumference greater than 60 cm represents a 35% chance of difficulty with intubation.
 - ii. These patients may often require an awake fiber optic intubation.
 - b. Many authors now recommend “ramping” patients’ shoulders and neck so that the external auditory meatus is in line with the sternal notch prior to induction to aid in visualization of the vocal cords, especially in the setting of direct laryngoscopy.

91. Obesity

4. Gastrointestinal

- a. Gastric volume is increased, even in a fasted obese patient.
- b. The pH of the gastric juice is reduced, predisposing the patient to aspiration pneumonitis should aspiration occur.
- c. Patients should be pre-medicated with H₂ blockers and metoclopramide prior to surgery.
- d. All obese patients should be considered to have a “full” stomach.

5. Endocrine

- a. Obese patients have a much higher incidence of type II diabetes mellitus than non-obese patients.
- b. They may require exogenous insulin to maintain normal glucose levels during the peri-operative period.

TKO

1. Have a high index of suspicion for obstructive sleep apnea (OSA).
2. An awake fiber optic intubation should be considered in many obese patients.
3. Neck circumference is an independent indicator of a difficult airway.
4. Pulmonary embolism is the major cause of 30-day mortality in bariatric surgery patients.
 - a. Measures to prevent deep vein thrombosis must be taken.
5. Drugs with a short duration of action should be used during maintenance of anesthesia.
 - a. If long-acting narcotics must be used, then the obese patient may require hospitalization overnight with continuous oxygen saturation monitoring.



KO TREATMENT PLAN

Pre-operative

1. Complete history and physical
2. Determine the severity of her OSA.
 - a. This will help you decide if she should stay overnight with continuous pulse oximetry monitoring and help determine your anesthetic plan.
3. A pre-operative EKG should be obtained if deemed necessary after a careful history.
 - a. Chest pain
 - b. Shortness of breath
 - c. Orthopnea
 - d. Poor functional capacity
4. Chest X-ray: heart size and pulmonary vasculature (evidence of pulmonary hypertension)
5. Pre-operative blood glucose should be checked and treated.
6. Evaluate her airway.
7. The patient in the above sample case should be pre-medicated with intravenous (IV) metoclopramide and ranitidine the morning of surgery.

Intra-operative

1. I would intubate the patient via an awake fiber optic intubation.
 - a. “Full stomach”
 - b. Risk for rapid desaturation
 - i. Pre-medication
 - (1) Glycopyrrolate 0.2 mg IV
 - (2) Conservative doses of sedation
 - (a) 1–2 mg IV versed
 - (b) 50 micrograms of IV fentanyl
 - (3) Adequate topicalization either via nebulized lidocaine 2–4% for 10 minutes or having the patient gargle with 5 ml of 2% viscous lidocaine.
 - (4) The patient should have the standard ASA monitors applied prior to any sedation or topicalization.
2. Once the patient is successfully intubated, I would induce with propofol 1–2 mg/kg because of its rapid onset and short duration of action.
3. If paralysis is required (it is not always needed) full reversal and demonstration of return of muscular strength, (i.e., with 5 second head lift) is mandatory prior to extubation.
4. Pharmacological considerations
 - a. Lipophilic drugs
 - i. Dose according to actual body weight.
 - ii. The loading dose should be higher.
 - iii. It will take longer to reach a steady-state concentration compared to hydrophilic drugs due to the patient’s excessive adipose tissue acting as a diluting reservoir for lipophilic drugs.
 - iv. It will take longer for the drug to be cleared from the body once it is stopped compared to hydrophilic drugs due to the patient’s excessive adiposity.
 - v. Examples
 - (1) Thiopental: expect a long duration of action.
 - (2) Propofol
 - (3) Benzodiazepines: highly lipid soluble
 - (4) Fentanyl
 - b. Hydrophilic drugs
 - i. Dose according to an adjusted body weight.
 - (1) Weight between the actual and ideal body weight.
 - (2) Adjusted body weight = $40\% \times (\text{actual body weight} - \text{ideal body weight}) + \text{ideal body weight}$
 - (a) Ideal body weight
 - (i) Males: $0.9 \times \text{height in cm} - 88$
 - (ii) Females: $0.9 \times \text{height in cm} - 92$
 - ii. Examples
 - (1) Vecuronium: recovery may still be prolonged.
 - (2) Pancuronium: low lipid solubility
 - (3) Succinylcholine: with a maximum dose of 120–140 mg IV
5. I would use a remifentanyl infusion and desflurane for maintenance anesthesia because their effects are short-lived.
 - a. There is very little, if any, residual respiratory depression from these agents.

- b. Metabolism of remifentanyl is via non-specific plasma esterases.
- c. Desflurane is a very insoluble agent; ideal for a patient with a large amount of adipose tissue.

Post-operative

1. Extubation criteria are the same as for non-obese patients.
 - a. I would be much stricter in following the established criteria:
 - i. Vital capacity greater than 15 cc/kg
 - ii. Respiratory rate 10–20 breaths per minute
 - iii. Reasonable arterial blood gas results.
 - iv. Negative inspiratory force greater than 20 cm H₂O

2. I would extubate the patient and apply her CPAP.
3. She should be monitored overnight with continuous oxygen saturation monitoring, secondary to her OSA.

BIBLIOGRAPHY

1. Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill Medical, 2005, p 813.
2. Chung F, Yegneswaran B, Liao P, et al. STOP Questionnaire: A tool to screen for obstructive sleep apnea. *Anesthesiology*, 2008;108:812–21.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, p 1048.
4. Gounden R, Blackman M. Clinical pharmacology: Dosing in the obese patient. *Clinical Pharmacology* 2006;24(7):399–400.

92. Sickle Cell Anemia^{1,2,3,4,5}

Sheila Chiu-Miller, DO and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

An 11-year-old male with sickle cell disease is scheduled for a repair of his ankle fracture. What are the anesthetic implications of sickle cell disease? How would you anesthetize this child? Would you use regional or general anesthesia? Can the surgeon use a tourniquet?

CLINICAL ISSUES

Definition

1. Sickle cell disease is a hereditary hemoglobin disorder that stems from structural deformation or sickling of the hemoglobin in the red blood cell.
2. Most common forms of the disease include sickle cell anemia (SS disease), sickle hemoglobin C disease (SC disease), sickle β -thalassemia, and sickle cell trait (SA disease).
3. The pathologic hallmark of this disease is vaso-occlusive crisis that arises when erythrocytes with irreversibly sickled hemoglobin aggregate in the blood vessels leading to venous thrombosis, organ infarction, and severe pain.

Pathophysiology of Sickle Cell Anemia

1. Normal adult hemoglobin (Hb A) is a protein with two α -globin and two β -globin chains.
2. Sickle hemoglobin (Hb S) has two normal α -globin chains and two sickle β -globin chains.

3. Genetic mutation in the β -globin gene where the neutral amino acid (valine) substitutes for an acidic amino acid (glutamic acid) at the sixth position
4. When hemoglobin S deoxygenates, a gel polymer of hemoglobin forms resulting in sickling of the red blood cell molecule.
5. Physiologic conditions that affect the rate of deoxygenation determine the extent of gel formation.
6. Low arterial oxygen tension, acidosis, and vascular stasis are powerful stimuli of sickle cell formation.
 - a. Sickling occurs at a PaO₂ <50 mm Hg and is most pronounced at a PaO₂ <20 mm Hg.
7. Polymerized sickle hemoglobin is relatively insoluble, rigid, and unstable.
8. Sickling and adhesion of red cells in the blood vessels cause a decrease in oxygen delivery to the tissues and a subsequent ischemia and destruction of end organs.
9. Splenic sequestration (acute and painful enlargement of the spleen) can occur in children less than 6 years old.
10. Bone marrow exhaustion can occur, leading to aplastic crises and severe anemia.

Sickle Cell Anemia (Hb SS)

1. Incidence is 0.2% among African Americans.
2. Homozygous genotype
3. 70–98% of hemoglobin is S and the remainder is hemoglobin F (fetal).

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Sickle Cell Trait (Hb AS)

1. Present in 8–10% of African Americans
2. Heterozygous genotype showing via incomplete dominance
3. One β -globin gene codes for sickle hemoglobin and the other for hemoglobin A.
4. 10–40% hemoglobin S
5. Hemoglobin A ($\alpha 2\beta 2$) and fetal hemoglobin F ($\alpha 2\gamma 2$) can prevent polymer formation and are responsible for the minimal clinical symptoms in sickle cell trait.
6. Patients are not symptomatic unless the PaO_2 is <20 mm Hg when Hb AS begins to sickle.

Sickle Hemoglobin C Disease

1. One β -globin gene codes for sickle globin and the other for hemoglobin C (where lysine replaces glutamic acid on sixth position of the β -globin chain).
2. Hemoglobin C can likewise form polymers with Hb S resulting in SC disease.

Sickle β -Thalassemia

1. One β -globin gene has a sickle cell mutation and the other has a thalassemia mutation.
2. The synthesis of the β -globin chain can be completely or partially suppressed.

Triggers of Sickle Cell Crisis

1. Low oxygen saturation/desaturated hemoglobin S
2. Acidosis
3. Hypothermia/hyperthermia
4. Infection
5. Emotional stress
6. Physical exertion
7. Alcohol consumption
8. Dehydration
9. Surgery

Clinical Signs and Presentation (% equals the incidence per lifetime)

1. Neurological
 - a. Pain crisis: $>70\%$ of patients
 - b. Stroke: 20% of patients
 - c. Proliferative retinopathy, peripheral neuropathy, and chronic pain syndrome are rare.
2. Cardiovascular
 - a. Myocardial infarction
3. Pulmonary
 - a. Acute chest syndrome (ACS)
 - i. Occurs in 40% of patients
 - ii. Pneumonia-like
 - iii. Clinically presents as chest pain, fever $>38.5^\circ\text{C}$, tachypnea, wheezing, or cough.

- iv. ACS diagnosis is based on a new pulmonary infiltrate involving at least one lung segment.
 - b. Airway hyper-reactivity: 35% of children
 - c. Restrictive lung disease: 10–15% of patients
4. Genitourinary
 - a. Chronic renal insufficiency: 5–20%
 - b. Urinary tract infection
 - c. Priapism: 10–40% men
 - d. Obstetric complications
 - i. Higher number of vaso-occlusive events
 5. Gastrointestinal
 - a. Cholelithiasis: 70% adults
 - b. Liver disease: 10%
 - i. Viral hepatitis from transfusion (up to 10% of adults)
 - ii. Liver failure ($<2\%$ patients)
 6. Hematological
 - a. Hemolytic anemia
 - b. Acute aplastic anemia
 - c. Splenic enlargement/fibrosis
 7. Orthopedic
 - a. Osteonecrosis: 50% adults
 - b. Osteomyelitis
 - c. Dactylitis
 8. Vascular
 - a. Ischemic leg ulcer: 20%
 9. Immunologic
 - a. Increased susceptibility to infection
 - b. Increased incidence of transfusion erythrocyte alloimmunization and therefore hemolytic transfusion reaction

Peri-operative Complications

1. Sickle cell patients have a high incidence of peri-operative problems including pain crisis, ACS, transfusion reactions, and other non-specific complications such as fever, infection, bleeding, thrombosis, embolism, and even death.
2. Peri-operative management should include stratifying risks for complications.
 - a. High surgical risk, longer duration of general anesthesia, increased age, recent crisis, frequent hospitalizations, abnormal chest X-ray, pregnancy, pre-existing infections, and organ failures increase risk.
 - b. Since sickling cannot be reversed, the best type of management is prevention of sickling.



KO TREATMENT PLAN

Pre-operative

1. Obtain a detailed history and physical examination including
 - a. Sickle cell trait or anemia or other variant hemoglobinopathy
 - b. Previous transfusions and related complications
 - c. Recent sickle cell crisis and/or hospitalization

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- d. Presence of any chronic organ dysfunction
 - e. Surgical history (e.g., splenectomy) that predisposes the patient to infections by encapsulated organisms
2. Labs and other tests
- a. Hematocrit (Hct) and the percentage of hemoglobin S (via electrophoresis) to determine whether simple or exchange transfusions (apheresis used to remove a person's red blood cells or platelets and replace them with a transfused blood product) are needed prior to the procedure
 - i. A patient with sickle cell disease should have a hemoglobin S ratio less than 40% prior to any elective surgery.
 - b. BUN, creatinine for baseline kidney function
 - c. Urinalysis to detect hematuria, proteinuria, and the presence of an infection
 - d. Pulse oximetry as a non-invasive measure of oxygenation
 - e. Chest X-ray
 - i. For patients with recent upper or lower respiratory tract symptoms or asthma exacerbation
 - ii. To detect the presence of pulmonary fibrosis, calcified spleen, and calcified marrow
 - f. Type and screen for antibodies
 - i. Leukocyte-reduced/filtered, sickle hemoglobin (Hb S) negative and phenotype/antigen-matched red cells are preferred to minimize alloimmunization and transfusion reactions.
 - g. Pre-operative transfusion guidelines
 - i. Intravenous (IV) fluids with no transfusion (even with a Hct < 30%):
 - (1) Local anesthetic
 - (2) Minor and brief procedure
 - (3) General anesthetic less than 60 minutes
 - (4) No pulmonary compromise
 - ii. Simple transfusion
 - (1) Hct < 30% with a goal of 30–35%
 - (2) Higher risk patients
 - (3) Non-tonsillectomy, non-laparotomy, non-thoracotomy procedures
 - iii. Repeated simple transfusion
 - (1) Elective procedures of the airway, open thoracotomy or general anesthetic > 90 minutes
 - (2) Goal for Hb S is less than 30%.
 - iv. Exchange transfusion
 - (1) Any Hct
 - (a) Urgent procedures of the airway, open laparotomy, thoracotomy, or anesthesia > 90 minutes
 - (b) Elective procedures with a goal of reducing the % of Hb S to 30%
 - (c) Patients with a history of severe ACS and/or chronic pulmonary disease
 - h. Other tests such as pulmonary and liver function tests, arterial blood gas, EKG, neurological imaging if indicated for a specific organ disease process

3. Avoid pre-operative sedative medications that may affect ventilation if patients have a pulmonary component to their sickle cell disease.

- a. Remember that these patients may have an increased tolerance for sedatives due to their prior treatment of pain crisis.

Intra-operative

1. Maintain normothermia with convection thermoregulation devices and fluid warmers.
 - a. Hypothermia depresses sickling but promotes peripheral vasoconstriction and vascular stasis.
 - b. Hyperthermia can promote hemoglobin S gel formation and sickling.
 - c. Your goal is to maintain normothermia.
2. Circulatory stasis and acute hypovolemia can be avoided with adequate hydration and anticipation of intra-operative volume loss.
 - a. IV fluids up to 1–1.5 × maintenance fluids especially if the kidneys are unable to concentrate urine
 - b. Must use fluids with caution if cardiac function is compromised to avoid pulmonary edema.
3. Correct acidemia and hypoxia.
4. Avoid areas of stasis such as pressure points that promote vascular stasis and sickling.
5. While no single anesthetic technique is considered more favorable in reducing sickle cell associated complications, I would choose
 - a. Pre-oxygenation to minimize hypoxia
 - b. Induction with fentanyl for pre-emptive analgesia and propofol for rapid onset of sedation (in the presence of intravenous access)
 - c. **Inhalation induction with sevoflurane if no pre-operative intravenous access**
 - i. Inhaled, halogenated anesthetics accelerate precipitation of hemoglobin S in vitro but no known clinical significance has been shown.
 - d. Maintenance with isoflurane
 - i. Potential neuroprotective and cardioprotective effects of isoflurane, especially in the event of ischemic episodes during anesthesia
 - e. Muscle relaxation
 - i. There is no contraindication for using a depolarizing or non-depolarizing muscle relaxant in sickle cell anemia patients.
 - ii. One study by Dulvadestin *et al.*⁵ showed that the onset time for atracurium is delayed in sickle cell anemia patients, but not the duration of the neuromuscular blockade.
 - f. Choice of anesthetic technique can be general or regional anesthesia as long as perfusion and oxygenation are maintained during the surgery.
 - i. General anesthesia may be preferred for a young and anxious child; however, regional analgesia

(e.g., epidural anesthesia) has been used safely in the pediatric population.

ii. Regional anesthesia may lead to compensatory vasoconstriction and decreased oxygen delivery to non-blocked areas resulting in red cells in non-blocked areas more vulnerable to sickling.

6. The use of an extremity tourniquet is controversial.
 - a. There is no definitive study to prove the safety or danger of the use of a tourniquet in a sickle cell patient.
 - b. The anesthesiologist and surgeon must weigh the benefits of a tourniquet (if necessary for the success of the surgery) and risks associated with induced vaso-occlusive and vascular stasis effects of the tourniquet.

Post-operative

1. Early mobilization to prevent venous stasis
2. Pulmonary toilet such as frequent suctioning and incentive spirometry to prevent hypoxia and atelectasis

3. Early effective analgesia using a variety of techniques such as opioids, non-narcotic analgesics, and epidural analgesia
4. Supplemental oxygen in the presence of hypoxia
 - a. Avoid the routine use of supplemental oxygen, which can suppress erythropoiesis.
5. Prompt recognition and treatments for possible complications such as pain crisis or ACS

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 1111-13.
2. Firth PG, Head CA. Sickle cell disease and anesthesia. *Anesthesiology*, 2004;101:766-85.
3. Humes HD. *Kelley's Textbook of Internal Medicine*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2000, pp 1788-94.
4. Blood transfusion for pediatric patients with sickle cell disease. New England Pediatric Sickle Cell Consortium. December 2003.
5. Dulvadestin P, Gilton A, Hernigou P, Marty J. The onset time of atracurium is prolonged in patients with sickle cell disease. *Anesth Analg*, July 2008;107(1):113-6.

93. Stress Dose Steroids^{1,2,3,4,5}

Michael Dale Bassett, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 65-year-old male is admitted to the emergency department and found to have an acute appendicitis. His medical history is significant for rheumatoid arthritis. The patient has taken prednisone 10 mg/day for several years. After an uneventful appendectomy, the patient becomes hypotensive with a blood pressure of 76/40 mm Hg in the post-anesthesia care unit. The patient received a 2L normal saline intravenous (IV) bolus without improvement. Would you recommend that the patient receive stress dose steroids in this situation?

CLINICAL ISSUES

Normal Physiology

1. Glucocorticoids (including cortisol) are produced in the cortex of the adrenal gland.
2. Glucocorticoid production is regulated by adrenocorticotropic hormone (ACTH) secreted from the anterior pituitary under the influence of corticotropin-releasing hormone (CRH) from the hypothalamus.
3. ACTH and CRH are controlled by negative feedback through cortisol.

4. Normal daily production of cortisol is about 15-20 mg/day.
5. Acute physical or psychological stress activates the hypothalamic-pituitary-adrenal (HPA) axis resulting in increased levels of CRH, ACTH, and ultimately cortisol.

Actions of Cortisol

1. Stimulation of gluconeogenesis
 - a. Increase protein catabolism
 - b. Increase lipolysis
 - c. Decrease insulin sensitivity of adipose tissue
2. Anti-inflammatory effects
 - a. Induce the synthesis of lipocortin (an inhibitor of phospholipase A₂)
 - b. Inhibit production of interleukin-2 (IL-2)
 - c. Inhibit release of histamine and serotonin from mast cells and platelets
3. Calcium homeostasis
 - a. Stimulate osteoclasts; inhibit osteoblasts
 - b. Reduce intestinal calcium absorption
 - c. Stimulate parathyroid hormone release
 - d. Increase urinary calcium excretion
 - e. Decrease reabsorption of phosphate

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4. Suppress immune response by inhibition of IL-2 and T-lymphocytes
5. Maintain vascular response to catecholamines by up-regulating α_1 adrenergic receptors on arterioles
6. Increase cardiac output
7. Cause loss of collagen and connective tissue

Effect of Surgery

1. Surgery is a potent activator of the HPA axis.
2. The degree of activation depends on the type of surgery, age of the patient, medication usage, and concurrent illness.
3. Cortisol release enhances survival by improving
 - a. Cardiac output
 - b. Responsiveness to catecholamine
 - c. Work capacity of skeletal muscles
 - d. Ability to mobilize energy sources
4. Plasma ACTH and cortisol
 - a. Increase at time of surgical incision
 - b. Levels continue to increase during the surgery.
 - c. Greatest increase occurs during reversal of anesthesia, extubation, and in the immediate post-operative period, primarily due to pain.
5. Cortisol levels are measured at
 - a. 50 mg/day during minor procedures
 - b. 75–100 mg/day during major procedures
 - c. Can reach between 200 and 500 mg/day with severe stress
 - d. Rare to be greater than 200 mg/day 24 hours after surgery

Chronic Glucocorticoid Therapy

1. Suppresses the HPA axis
2. Impairs wound healing
3. Increases friability of skin, superficial blood vessels, and other tissues
4. Increases risk of fractures, infections, gastrointestinal hemorrhage, and ulcers

Acute Adrenal Insufficiency Crisis

1. Chronic glucocorticoid therapy, including inhaled and topical steroids, exerts negative feedback on the HPA axis by suppressing CRH and ACTH release and can cause a secondary adrenal insufficiency.
2. The adrenal gland atrophies and can no longer secrete cortisol.
3. These patients cannot increase their cortisol levels during surgery and can suffer from an acute adrenal insufficiency crisis.
4. Adrenal insufficiency crisis
 - a. Fatigue
 - b. Nausea and vomiting
 - c. Fever
 - d. Abdominal pain
 - e. Anorexia

- f. Hyponatremia
- g. Hypoglycemia
- h. Hypotension
- i. Shock

5. After cessation of steroid therapy, recovery of the HPA axis can take up to 12 months or longer.

Criteria Regarding HPA Axis Suppression

1. The following patients can be considered to not have a suppressed HPA axis, and therefore do not require stress dose steroids in the peri-operative period.
 - a. Patients receiving morning doses of prednisone (or its glucocorticoid equivalent) 5 mg/day or less for any length of time
 - b. Any dose of a glucocorticoid for less than 3 weeks
 - c. Patients treated with alternate-day glucocorticoid therapy
2. The following patients should be considered to have suppression of their HPA axis and therefore require stress dose steroids in the peri-operative period.
 - a. Any patient who has received more than 20 mg/day of prednisone (or its glucocorticoid equivalent) for more than 3 weeks within the previous year
 - b. Any patient who has clinical Cushing's syndrome
3. Intermediate category patients include those taking more than 5 mg/day but less than 20 mg/day of prednisone (or its glucocorticoid equivalent) within the previous year. They may or may not have HPA axis suppression.

HPA Axis Evaluation

1. In patients whose HPA axis status is uncertain, one can test for responsiveness of the adrenal gland to stress.
2. Measurement of cortisol levels during insulin-induced hypoglycemia
 - a. Most sensitive test of response to stress
 - b. Not commonly used as it is difficult to perform and risky to the patient
3. 1 μ g ACTH (cosyntropin) stimulation test
 - a. Cosyntropin 1 μ g is given intravenously.
 - b. Plasma cortisol is measured before and 30 minutes after cosyntropin administration.
 - c. Adrenal function is considered normal if the plasma cortisol level is at least 18 μ g/dL at 30 minutes.
 - d. ACTH levels obtained with the 1 μ g test are equivalent to ACTH levels after stressful situations, such as surgery or hypoglycemia.
4. 250 μ g cosyntropin stimulation test
 - a. Cosyntropin 250 μ g is given intravenously or intramuscularly.
 - b. The plasma cortisol is measured before and 60 minutes after the cosyntropin administration.
 - c. Adrenal function is considered normal if the plasma cortisol level is at least 20 μ g/dL at baseline or 60 minutes after cosyntropin administration.

- d.** Limitations to this test are that the dose of cosyntropin is much higher than that often observed in the body in response to most stressful situations. This explains why patients with a partially suppressed HPA axis fail to respond to the 1 µg test but respond normally to the 250 µg cosyntropin test.
- 5.** In each of these stimulation tests, cross-reaction between exogenous glucocorticoids and the test can occur.
- May lead to falsely elevated cortisol values
 - Glucocorticoids should be stopped 24 hours prior to testing.
 - Dexamethasone does not affect these tests.



KO TREATMENT PLAN

Peri-operative Management

- Patients who are considered not to have functional suppression of their HPA axis by the criteria outlined above should continue on their usual dose of glucocorticoids peri-operatively if they are still taking them. If they are no longer on glucocorticoids, they do not require any additional steroid prior to surgery.
- Patients who are considered to have HPA axis suppression by the defined criteria or through testing should be treated based on the surgery being performed.

- Minor procedures or surgery under local anesthesia: take the usual morning steroid dose. No extra supplementation is necessary.
 - Moderate surgical stress: take the usual morning steroid dose. Give 50 mg hydrocortisone IV just before the procedure and 25 mg every 8 hours for 24 hours. Resume the usual dose thereafter.
 - Major surgical stress: take the usual morning steroid dose. Give 100 mg hydrocortisone IV before induction of anesthesia and 50 mg every 8 hours for 24 hours. Taper the dose by half per day until the maintenance level is reached.
- 3.** Patients who fall into the intermediate category regarding their HPA axis can be treated with glucocorticoids, or if time permits, can be tested for responsiveness of the adrenal gland to stress.

BIBLIOGRAPHY

- Brown, CJ, et al. Perioperative stress dose steroids: Do they make a difference? *J Am Coll Surg*, Dec 2001;193(6):678–86.
- Andreoli TE, Carpenter CCJ, Griggs RL, et al. *Cecil Essentials of Medicine*, 6th ed. Philadelphia: Saunders, 2004, pp 603–14.
- Jabbour SA. Steroids and the surgical patient. *Med Clin North Am*, Sep 2001;85(5):1311–7.
- Reed A, Yudkowitz F. *Clinical Cases in Anesthesia*, 3rd ed. Philadelphia: Elsevier, 2005, pp 165–9.
- Welsh, GA, et al. The surgical patient taking glucocorticoids. Up to Date (Online) July 2007. <http://www.utdol.com>.

94. Transfusion Therapy^{1,2,3,4}

Vera V. Borzova, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 23-year-old man sustained a gunshot wound to the abdomen. The patient was taken to the operating room for an emergency laparotomy. His heart rate was 140, blood pressure 100/60 mm Hg, and respiratory rate was 28. Upon surgical opening of the abdomen, the patient's blood pressure dropped to 80/40 mm Hg. The estimated blood loss is at least 4 L. What are your anesthetic concerns? What are your plans for his massive blood transfusion? Which blood components would you use and why? What complications are associated with a massive blood transfusion?

CLINICAL ISSUES

Blood Products

- Whole blood: rarely used to allow better utilization of blood bank resources
- Red blood cells (RBC)
- Platelets
- Fresh frozen plasma (FFP): contains all of the coagulation factors including labile factors V and VIII.
- Cryoprecipitate: contains concentrated amounts of fibrinogen, von Willebrand factor, and factor XIII.

6. Factor concentrates: factors VIII, IX, and antithrombin III
7. Synthetic factors: factor VIIa and erythropoietin

Indications for Transfusions of RBC

1. Low oxygen-carrying capacity with impaired tissue oxygenation
2. RBC is usually administered when the hemoglobin is less than 6 g/dL and usually unnecessary when the hemoglobin (Hb) is more than 10 g/dL.
 - a. Trigger for transfusion for the majority of surgical patients is around 7 g/dL (hematocrit (Hct) of 21%).
 - b. Important exception is a patient with a recent acute myocardial infarction/unstable angina. A minimal threshold of 9.5–10 g/dL is used for these patients.
3. Do not use blood products to replace intravascular fluid volume. Anemia is often well tolerated if intravascular volume is maintained (isovolemic anemia).
4. One unit of packed RBC increases the Hb by 1 g/dL and Hct by 2–3% in a euvolemic patient.
5. Use ABO compatible red blood cells.

Indications for Transfusion of Platelets

1. Low platelet count and/or platelet dysfunction
2. Platelet transfusions are rarely indicated if the platelet count is more than 100,000/uL in the absence of platelet dysfunction.
3. Usually indicated when the count is less than 50,000/uL in the presence of excessive bleeding
4. Prophylactic transfusion is indicated for a platelet count <10,000 - 20,000/uL to prevent spontaneous bleeding.
5. A threshold of 80,000/uL is often used for spinal and epidural anesthesia.
6. One unit of platelets increases the platelet count by 5,000–10,000/uL.
7. ABO compatible platelets are preferred but not required for transfusion

Indications for Transfusion of FFP

1. Correction of factor deficiencies for which specific concentrates are unavailable
2. Correction of multiple factor deficiencies including
 - a. Disseminated intravascular coagulation (DIC)
 - b. Liver disease
 - c. After massive blood transfusions
 - i. FFP should be given in doses sufficient to achieve a minimum of 30% of plasma factor concentration. This usually requires administration of 10–15 mL/kg of FFP.
3. Urgent reversal of warfarin therapy usually requires 5–8 mL/kg of FFP.
 - a. If time permits and serious bleeding is absent, withdrawal of warfarin and administration of vitamin K is the preferred treatment.

4. Consider administering FFP in antithrombin III deficiency (heparin resistance) in patients requiring heparin when an antithrombin concentrate is unavailable.
5. Guide the transfusion of FFP by following the INR/prothrombin time (PT)/partial thromboplastin time (aPTT) or by using clinical evidence of bleeding if INR/PT/PTT are not available in a timely manner.
 - a. PT greater than 1.5 times normal, INR greater than 2.0, and a PTT greater than 2 times normal warrant transfusion of FFP to correct excessive microvascular bleeding.
6. Use ABO compatible FFP.
 - a. Group AB plasma is suitable for all blood types.

Indications for the Transfusion of Cryoprecipitate

1. Congenital fibrinogen deficiency: normal level of fibrinogen is 200–400 mg/dL
2. Microvascular bleeding with a fibrinogen level <80–100 mg/dL.
3. Ongoing microvascular bleeding after a massive transfusion when fibrinogen levels can not be obtained in a timely manner
4. Consider for treatment of hemophilia A or von Willebrand disease when concentrates are unavailable.
5. 1 unit of fibrinogen per 10 kg of body weight increases fibrinogen by 50 mg/dL in the absence of continued consumption or active bleeding.
6. ABO compatibility is not required but preferable for transfusion of cryoprecipitate.

TKO: The three leading causes of transfusion-related death:

1. Transfusion-related acute lung injury (TRALI)
2. ABO incompatibility
3. Bacterial sepsis

Complications of Blood Transfusions

1. Infectious
 - a. Bacterial contamination, especially of platelets stored at room temperature
 - b. Viral diseases
 - i. Cytomegalovirus (CMV)
 - ii. Hepatitis B and C
 - iii. Human immunodeficiency virus (HIV)
 - iv. Human T-cell lymphotropic virus
 - v. West Nile virus
2. Non-infectious
 - a. Acute hemolytic transfusion reactions
 - i. Usually a result of ABO incompatibility
 - ii. Represents immunologic destruction of transfused red cells most often due to incompatibility of the antigen of the transfused cells with an antibody in the recipient's circulation.

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- iii. Signs are non-specific in a patient under general anesthesia
 - (1) Tachycardia
 - (2) Hypotension
 - (3) DIC with diffuse bleeding
 - (4) Hemoglobinuria
 - (5) Elevated peak airway pressure
 - iv. Diagnosis
 - (1) High index of suspicion
 - (2) Direct Coombs test: definitive test
 - (3) Additional laboratory tests
 - (a) Decreased serum haptoglobin
 - (b) Plasma and urine hemoglobin
 - (c) Increased serum bilirubin
 - (d) Decreased platelet count
 - (e) Prolongation of PT, aPTT, INR
 - (f) Decreased serum fibrinogen and elevation of the fibrin degradation products
 - v. Treatment
 - (1) Stop the transfusion, identify the patient, recheck the blood, and repeat a cross-match.
 - (2) Support the blood pressure with fluids, pressors, and inotropes.
 - (a) 0.9% saline given intravenously to maintain a urine output of 100 mL/hour.
 - (b) Dopamine is used to support blood pressure.
 - (3) Support renal function
 - (a) Maintain volume and blood pressure.
 - (b) Diuretics: mannitol and furosemide
 - (c) Sodium bicarbonate to alkalinize the urine
 - b. Delayed hemolytic transfusion reactions
 - i. Extravascular hemolysis of donor RBCs by recipient antibodies upon re-exposure to an RBC antigen (anamnestic response)
 - ii. Diagnosis
 - (1) Index of suspicion
 - (2) Unexplained reduction in hemoglobin concentration
 - (3) Low-grade fever, mild jaundice, and elevated bilirubin
 - (4) Positive Coombs test: definitive test
 - 3. Allergic reactions
 - a. Reaction to the proteins in the donor plasma that occurs after the transfusion of plasma (most often) as well as RBCs and platelets (see the anaphylaxis chapter, Chapter 85)
 - b. Treatment is supportive.
 - i. Stop the transfusion.
 - ii. Intravenous (IV) crystalloid fluids
 - iii. IV epinephrine
 - iv. Methylprednisolone
 - v. Diphenhydramine
 - 4. Febrile reactions to donor leukocytes are usually mild and self-limited.
 - a. Treat with acetaminophen.
 - 5. Transfusion-related acute lung injury
 - a. Form of non-cardiogenic pulmonary edema as a result of activation of host leukocytes by donor antibodies
 - b. Clinical picture of ARDS with hypoxia, dyspnea, and fever
 - c. Treatment is supportive with mechanical ventilation as needed.
 - 6. Graft versus host disease
 - a. Passenger donor lymphocytes establish an immune response against an immunocompromized host.
 - b. Pancytopenia develops rapidly.
 - c. Irradiation of blood products is the only way to prevent graft-versus-host disease.
 - 7. Post-transfusion purpura
 - a. Thrombocytopenia is due to the destruction of the patient's own platelets by alloantibodies.
 - b. Plasmapheresis is used to remove the antibodies.
 - c. Intravenous immunoglobulin is the treatment of choice.
 - 8. Immunosuppression may be associated with blood transfusion and may be responsible for cancer recurrence, activation of viral infections, and increased mortality.
 - a. Use leukoreduction as some of the reactions are linked to lymphocytes and limit the amount of transfusions in susceptible patients.
- TKO:** Complications of Massive Blood Transfusion: transfusion of more than one patient blood volume.
- 1. Hypothermia
 - 2. Volume overload
 - 3. Dilutional coagulopathy: decrease in fibrinogen, factors II, V, and VIII, and platelets
 - 4. Left shift of the oxygen-Hb dissociation curve due to a decrease in the 2,3-DPG of the stored RBC
 - 5. Citrate intoxication can occur with a rate of transfusion of 1 unit of RBC every 5 minutes in an adult.
 - a. Watch for signs of hypocalcemia: hypotension, narrow pulse pressure, elevated central venous pressure (CVP), and prolonged QT interval on ECG.
 - 6. Hyperkalemia is possible when large volumes of stored blood are given rapidly.
 - a. ECG shows peaked T waves, prolonged PR interval, and a widened QRS.
 - b. If ECG changes are observed, stop the transfusion, administer calcium, bicarbonate, insulin, and glucose.
 - 7. Acid-base disturbances
 - a. Metabolic acidosis with transfusion of stored blood followed by metabolic alkalosis as the lactate and citrate in the transfused blood are being converted to bicarbonate in the liver
- ### Compatibility Testing
- 1. ABO, Rhesus typing
 - a. Most fatal hemolytic transfusion reaction result from ABO incompatibility
 - b. Transfusion of type-specific blood has a 99.8% probability that the transfusion will be compatible.
 - 2. Antibody screen
 - a. It identifies recipient antibodies against donor RBC antigens.

94. Transfusion Therapy

- b.** When the plasma screen is positive, the blood bank will proceed to identify the antibody.
 - c.** Use appropriate antigen-negative donor units for transfusion.
 - d.** If blood was screened for antibodies, the probability of a compatible transfusion becomes 99.94%.
- 3. Cross-match**
- a.** Donor cells get mixed with recipient serum. It is performed in three phases.
 - i.** The first phase takes 1–5 minutes and confirms ABO and Rh type.
 - ii.** The second phase takes 30–45 minutes and detects antibodies to different blood group systems.
 - iii.** The third phase is performed only when the screening is positive and it takes 60–90 minutes. It detects antibodies in low titers and antibodies that do not agglutinate easily.
 - iv.** The cross-matched blood has 99.95% probability of compatible transfusion.

Emergency Transfusion

- 1.** When cross-matched blood is unavailable, use type-specific, partially cross-matched blood.
- 2.** If type-specific, partially cross-matched blood is unavailable, use type-specific, uncross-matched blood.
- 3.** If type-specific blood is unavailable, use type O Rh-negative blood: the universal donor.
 - a.** Do not withhold Rh-positive blood from an exsanguinating Rh-negative patient if Rh-negative blood is unavailable. In other words, it is OK to give O-positive blood to an O-negative recipient in an emergent situation.
- 4.** If more than 10–12 units of O-negative blood were administered to non-group O patient, continue with O negative blood even if type specific blood becomes available unless the blood bank confirms that anti-A or anti-B antibodies are present in low titers only.
 - a.** Anti-A and anti-B antibodies when combined with group O red blood cells can cause hemolysis of A, B, or AB red blood cells.



KO TREATMENT PLAN

Pre-operative

- 1.** At least two large-bore IV lines
- 2.** Rapid transfusion device
- 3.** Activate massive transfusion protocol if available.
 - a.** Anticipate the need to transfuse RBC, FFP, platelets, and possibly cryoprecipitate.
- 4.** Obtain blood samples.
 - a.** Blood type and cross-match
 - b.** Baseline coagulation studies (INR, PT, aPTT)
 - c.** Complete blood count (CBC)
 - d.** Blood chemistry

Intra-operative

- 1.** Rapid sequence induction and intubation
- 2.** Placement of arterial line
- 3.** Insert a Foley catheter to monitor urine output and check for hemoglobinuria.
- 4.** Start volume resuscitation with crystalloids and blood using a rapid transfusion device and fluid warmer.
 - a.** Can use O negative blood if type and screen are unavailable.
 - b.** Switch to cross-matched blood as soon as possible.
- 5.** Monitor the patient's vital signs, temperature, acid-base status, electrolytes, and hemoglobin/hematocrit.
 - a.** Keep in mind that hemoglobin/hematocrit are affected by the patient's fluid status and may be an inaccurate indicator of the blood oxygen content in the presence of active bleeding.
- 6.** Treat hypothermia aggressively.
- 7.** In the presence of ongoing microvascular bleeding, consider dilutional coagulopathy or the development of DIC.
 - a.** Draw coagulation studies, CBC, and send blood for fibrinogen and fibrin split products.
- 8.** Consider correcting coagulopathy with platelets, FFP, and cryoprecipitate as needed, especially if more than 1.5–2 blood volumes have been transfused.
- 9.** Consider recombinant factor VIIa therapy.
 - a.** Used for uncontrolled bleeding in hemophilia patients
 - b.** Increasingly used to treat uncontrolled hemorrhage
 - i.** It has an associated increased risk of deep vein thrombosis, pulmonary embolism, and myocardial infarction.
- 10.** Follow electrolytes closely, paying special attention to calcium (hypocalcemia) and potassium (hyperkalemia); correct as needed.

Post-operative

- 1.** Anticipate the need for an intensive care unit bed and continuing resuscitation.
- 2.** Prepare to transport a critically ill, intubated patient.

BIBLIOGRAPHY

- 1.** Practice guidelines for perioperative blood transfusion and adjuvant therapies: An updated report by the American Society of Anesthesiologists Task force on Perioperative Blood Transfusion and Adjuvant Therapies. *Anesthesiology*, July 2006;105(1):198–208.
- 2.** American Red Cross. *Practice Guidelines for Blood Transfusion*, 2nd ed. A compilation from recent peer-reviewed literature, April 2007.
- 3.** Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 208–21.
- 4.** Dunn PF, ed. *Clinical Anesthesia Procedures of the Massachusetts General Hospital*, 7th ed. Philadelphia: Lippincott Williams and Wilkins, 2206, pp 603–20.

95. Tourniquet Physiology^{1,2}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 30-year-old female underwent surgery for a left total knee replacement lasting 2 hours. After the tourniquet was released, her blood pressure rapidly fell to 70/40 mm Hg. How would you manage this drop in blood pressure? What if her oxygen saturation fell to 86%? How would you manage this drop in oxygenation?

CLINICAL ISSUES

Indication for Tourniquet

1. Applied to upper or lower extremities to decrease intra-operative bleeding.
2. Improve the operative conditions.

Relative Contraindications

1. Sickle cell disease
 - a. Possible precipitation of sickle cell crisis secondary to the low concentration of oxygen in the ischemic limb.
 - b. If a tourniquet is considered mandatory to decrease the blood loss and improve the operative visualization, maintenance of adequate hydration, warmth, and blood volume is critical.

Physiologic Changes¹

1. Neurologic
 - a. Somatosensory evoked potentials and nerve conduction are ablated within 30 minutes.
 - b. Pain and hypertension can be seen at 45–60 minutes.
 - c. Post-operative neurapraxia occurs at 2 hours.
 - d. Nerve injury may occur at the skin level under the edge of the tourniquet. Direct pressure from the cuff is more damaging than the distal ischemia.
2. Muscle
 - a. Cellular hypoxia occurs within 8 minutes.
 - b. Cellular creatinine levels decline.
 - c. Progressive cellular acidosis
 - d. Endothelial capillary leak develops in 2 hours.
 - e. The limb becomes progressively colder.
3. Systemic effects of tourniquet inflation
 - a. Expansion of central venous blood volume
 - b. Arterial and pulmonary artery pressure increases.

4. Systemic effects of tourniquet release
 - a. Transient fall in core temperature by 0.7 degrees centigrade occurs within 90 seconds of deflation.
 - b. Transient metabolic acidosis
 - c. Transient fall in central venous oxygen tension by about 20% within 30–60 seconds (a decrease in arterial oxygen saturation is unusual).
 - d. Acid metabolites are released into the central circulation.
 - e. Transient fall in pulmonary and systemic arterial pressures
 - f. The fall in blood pressure can be significant and has resulted in cardiac arrest.
 - g. 10–15% increase in heart rate
 - h. 5–10% increase in serum potassium
 - i. Transient increase in end-tidal carbon dioxide
 - j. Increased oxygen consumption
5. Local effects of tourniquet inflation
 - a. Mitochondrial partial pressure of oxygen decreases to zero within 8 minutes.
 - b. Anaerobic metabolism begins.
 - c. Tissue edema begins with inflation lasting longer than 60 minutes.
 - d. Secondary to edema, closure of the wound may be more difficult.
 - e. Injury to the muscle beneath the tourniquet may delay rehabilitation.
6. Prolonged inflation
 - a. Hypertension occurs after 45 to 60 minutes.
 - i. Treat with deepened anesthesia.
 - ii. The refractory treatment is IV hydralazine, nifedipine, or labetalol.
 - b. Neurological problems may occur if inflated greater than 2 hours.
 - c. Deflate the cuff every 90–120 minutes to minimize the risk of neurapraxias.



KO TREATMENT PLAN

Intra-operative

1. A transient fall in blood pressure is common with the deflation of the tourniquet. Monitor closely and treat as needed.

2. A persistent drop in blood pressure and pulse oximetry may be ominous.

- a. Verify the patient's vital signs.
- b. Check for a pulse.
- c. Look at the cardiac rhythm and end-tidal CO₂.
- d. Consider a differential diagnosis that includes fat embolism, anaphylaxis, and cardiac arrest.

- e. Treat accordingly.
- f. ACLS

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2423–5.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1123–4.

96. Electroconvulsive Therapy (ECT)^{1–12}

Kelly P. Jones, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 57-year-old male with known hypertension presents on the day of his ECT with an elevated blood pressure of 180/95 mm Hg. All other vitals are within normal limits. This is not his first treatment. Would you go ahead with the procedure?

CLINICAL ISSUES

Definition of Electroconvulsive Therapy (ECT)

1. A procedure in which a programmed electrical stimulus is passed through the brain, deliberately triggering a brief seizure¹
2. A seizure of at least a 25-second duration is necessary for a therapeutic effect.
3. The current is administered to both hemispheres or only the non-dominant hemisphere.
 - a. Stimulation of only one hemisphere is associated with reduced memory loss.
4. The stimulus produces a grand mal seizure: brief tonic phase followed by a more prolonged clonic phase.
5. A patient usually requires 6–12 treatments during a hospitalization and then continued treatments weekly or monthly.

Indications for ECT²

1. Definitely effective
 - a. Major depression refractory to antidepressant therapy
 - b. Need exists for a rapid treatment response, such as in pregnancy.
 - c. Medical co-morbidities prevent the use of antidepressant medication.
 - d. Previous response to ECT

- e. Depression with psychotic features
- f. Catatonic stupor
- g. Severely suicidal
- h. Food refusal leading to nutritional compromise
- i. Bipolar disorder
- j. Schizophrenia
- k. Mania
- l. Atypical psychosis
2. May be effective in
 - a. Neuroleptic malignant syndrome
 - b. Organic delusional disorder
 - c. Organic mood disorder
 - d. Obsessive-compulsive disorder
 - e. Neuroleptic-induced Parkinsonism
 - f. Neuroleptic-induced tardive dyskinesia
 - g. Catatonia secondary to medical conditions

Relative Contraindications for ECT

1. Unstable or severe cardiovascular disease
2. Space-occupying intracranial lesion or elevated intracranial pressure
3. Recent cerebral hemorrhage or stroke
4. Bleeding or unstable intracerebral aneurysm
5. Severe pulmonary disease
6. ASA class 4 or 5

Morbidity and Mortality

1. Approximately 4 deaths per 100,000 treatments

Physiologic Effects of ECT

1. Cardiovascular

- a. Initiation of the electrical stimulus triggers a brief parasympathetic discharge that can lead to a 10–15 second period of bradycardia with or without hypotension, premature atrial or ventricular contractions, or asystole.
 - b. The onset of the seizure often leads to a catecholamine surge that produces tachycardia and hypertension. The duration of the hemodynamic changes correlates with the length of seizure.⁶
 - c. The postictal state may present with compensatory bradycardia that can be effectively treated with anticholinergics if needed.
2. Central nervous system
- a. Increased cerebral blood flow⁷
 - b. Increased cerebral oxygen consumption
 - c. Increased intracranial pressure⁷
 - d. Possible increased blood-brain permeability⁷
 - e. Memory loss is a common side effect of ECT.
 - f. Disorientation can last from minutes to hours and appears to be related to the treatment parameters.
 - g. Delirium occurs in approximately 10% of patients recovering from anesthesia.
 - i. Several conditions have been identified that increase the risk for delirium post-ECT.
 - (1) Stroke within 1 year
 - (2) Parkinson's disease
 - (3) Advanced age⁹
 - h. Headache is also a common complaint after ECT.
 - i. Ibuprofen has been shown to be effective in prophylaxis of post-ECT headache.



KO TREATMENT PLAN

Pre-operative

A complete pre-surgical evaluation should be performed with special attention to the areas listed below.

1. Pre-existing medical conditions
 - a. Cardiac disease: arrhythmia, ischemia, congestive heart failure
 - i. 2007 American Heart Association and the American College of Cardiology consider ECT a low-risk procedure.⁵
 - ii. It is well tolerated even in at-risk patients because the duration of hemodynamic changes is brief.⁵
 - b. Neurologic disease: space-occupying lesion, stroke, dementia
 - c. Osteoporosis or other causes of fragile bones
 - d. Medications: One study showed 54% of patients with psychiatric illnesses use alternative medicine in addition to physician-prescribed pharmacotherapy.¹
 - e. Labs: the American Psychiatric Association suggest no routine medical labs are required unless indicated by other medical conditions such as hypertension treated with diuretics.

Intra-operative

1. Anesthesia considerations

Many IV anesthetics have been used for ECT.¹⁰

 - a. Methohexital (0.5–1.0 mg/kg IV) is the most commonly used drug for ECT and is considered the gold standard.
 - i. Rapid onset and rapid recovery
 - ii. Short duration of action
 - iii. Minimal anticonvulsant side effects
 - iv. Methohexital provides longer seizure duration when compared to propofol.
 - v. Fewer side effects than etomidate
 - vi. Faster wake-up than thiopental
 - b. Propofol 0.75 mg/kg
 - i. Decreased seizure duration compared to methohexital
 - ii. No difference in outcome was demonstrated when compared to methohexital.
 - iii. Lower blood pressure and heart rate response to ECT
 - c. Thiopental 1.5–2.5 mg/kg
 - i. Produced tachycardia and hypertension more often than propofol.
 - ii. Avoids pain at the injection site.
 - iii. No advantage over methohexital
 - iv. Prolonged recovery time when compared to methohexital.
 - d. Etomidate
 - i. May prolong seizure duration
 - ii. Good choice for patients when seizure duration was deemed too short with other agents
 - iii. It may also prolong recovery.
 - e. Ketamine
 - i. Less desirable agent because of the expected hemodynamic changes
 - ii. It does not prolong seizure duration or post-procedure agitation.
 - f. Succinylcholine (0.3–0.5 mg/kg IV)
 - i. Most commonly used neuromuscular blocker for ECT.
 - ii. It has a short duration of action and low frequency of side effects.
 - iii. Other non-depolarizing muscle relaxants may be used but may require prolonged ventilatory support.
 - g. Glycopyrrolate 0.2–0.4 mg
 - i. Given pre-operatively as an antisialagogue
 - ii. Decreases the bradycardia associated with ECT
 - h. Caffeine is given 3 to 5 minutes before initiation of ECT in varied doses.
 - i. Shown to increase seizure length
 - i. Beta-blockers
 - i. Used to control tachycardia and hypertension post-stimulus.
 - (1) Esmolol 1 mg/kg IV
 - ii. Has been shown to decrease the seizure duration.

- iii. There are reports of asystole after β -blocker treatment and ECT.
- iv. Routine treatment with β -blockers is not recommended because the hemodynamic changes are usually self-limited.¹¹

SAMPLE CASE

I would proceed with this case if there are no signs of unstable cardiac disease. Hypertension alone is not a contraindication. As noted above, this is a low-risk procedure, and hemodynamic changes are usually short-lived and self-limited. I would avoid Ketamine as an induction agent and have β -blockers available at the bedside. I would not pre-treat with β -blockers because of the reported prolonged bradycardia and asystole.

1. Pre-operative evaluation including review of all pertinent areas
2. Always review patient's previous anesthetic ECT records. They are usually a good guide for current anesthetics.
3. Intravenous access
4. Placement of standard ASA monitors
5. Placement of electroconvulsive leads
6. Glycopyrrolate 0.2–0.4 mg IV and caffeine if needed
7. Pre-oxygenate with 100% oxygen.
8. Placement of a second blood pressure cuff on the extremity without the IV to monitor seizure activity
 - a. Seizure activity should also be monitored by an electroencephalogram (EEG).
9. Methohexital or induction agent of choice
10. Bite block placement
11. Inflate the BP cuff on the arm without the IV.
12. Succinylcholine or muscle relaxant of choice
13. Initiation of the ECT
14. Follow the length of seizure by following the arm with the BP cuff and the EEG.
15. Esmolol or labetalol as indicated for prolonged tachycardia or hypertension
16. Assist with ventilation via bag, valve mask until recovery of protective airway reflexes.
17. Transport to recovery for continued post-operative monitoring.

Post-operative

There are three stages of recovery after surgery. Regardless of whether the ECT procedure is done as an outpatient or inpatient procedure, the patient should be monitored in

accordance with standard monitoring guidelines and meet the discharge criteria set by your institution.

1. Early
 - a. Time from emergence to recovery of protective airway responses and early motor activity
 - b. This often occurs in the operating or procedure room of ambulatory centers.
2. Intermediate
 - a. Time when the patient may be moved to a phase 2 recovery.
 - b. There is progression to ambulating, voiding, and oral intake.
3. Late
 - a. Resumption of normal daily activity
 - b. Usually occurs at home

BIBLIOGRAPHY

1. Knaut PR, Conner KM, Weisler RH, Churchill LE, Davidson JR. Alternative therapy use by psychiatric outpatients. *J Nerv Ment Dis*, Nov 1999;187(11):692–5.
2. American Psychiatric Association. Indications for electroconvulsive therapy. *Am J Psychiatry*, 2000;157:1.
3. American Psychiatric Association. *The Practice of Electroconvulsive Therapy: Recommendations for Treatment Training, and Privileging*, 2nd ed. Washington, DC: Author, 2001.
4. Abrams R. The mortality Rate with ECT. *Convuls Ther*, Sep 1997;13(3):125–7.
5. Fleisher, LA, Beckman, JA, Brown, KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines.
6. Larson, G, Swartz, C, Abrams, R. Duration of ECT-induced tachycardia as a measure of seizure length. *Am J Psychiatry*, 1984;14:1269.
7. Abrams, R. *Electroconvulsive Therapy*, 2nd ed. New York: Oxford University Press, 1992.
8. Daniel, WF, Crovitz, HF. Autobiographical amnesia with ECT: An analysis of the roles of stimulus wave form, electrode placement, stimulus energy, and seizure length. *Biol Psychiatry*, 1983;18:121.
9. Martin, M, Figiel, G, Mattingly, G, et al. ECT-induced interictal delirium in patients with a history of CVA. *J Geriatr Psychiatry Neurol*, 1992;5:149.
10. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2654–6.
11. Decina, P, Malitz, S, Sackeim, HA, et al. Cardiac arrest during ECT modified beta-adrenergic blockade. *Am J Psychiatry*, 1984;141:298.
12. Hines RL, Marschall KE. *Stoelting's Anesthesia and Co-Existing Disease*, 5th ed. Philadelphia: Churchill Livingstone, 2008, chapter 22.

97. Remote Anesthesia^{1,2,3}

Jessica A. Lovich-Sapola, MD

SAMPLE CASE

You are called by the neurologist to do the sedation for an MRI on a six month old child. What are your pre-operative concerns? What do you need to take with you to the MRI scanner? What safety concerns do you have for you and the patient? Can you use your usual monitors?

CLINICAL ISSUES

2003 Guidelines for Non-operating Room Anesthetizing Locations

1. It is the responsibility of the anesthesiologist to ensure that the location meets the ASA guidelines for safety. The anesthesiologist must maintain the same high standard of anesthetic care provided in the operating suite.
2. Reliable oxygen source with a full backup E cylinder of oxygen
3. Suction source
4. Waste gas scavenging system if inhalational agents are used
5. Adequate monitoring equipment that complies with the ASA standards
6. Anesthesia machine that has an equivalent function to the machines used in the operating rooms
7. Self-inflating resuscitator bag to deliver positive pressure ventilation
8. Adequate anesthetic drugs and supplies
9. Sufficient safe electrical outlets with isolated electrical power or ground fault circuit interrupters
10. Adequate light and battery-powered backup
11. Sufficient space
12. Emergency cart with defibrillator, emergency drugs, and emergency equipment
13. Means of reliable two-way communication
14. Compliance with safety and building codes
15. Adequately trained staff to support the anesthesia team
16. Adequate equipment for transport
17. Post-anesthesia care facilities

Remote Monitoring¹

1. Qualified anesthesia personnel must be present for the entire case.

2. Continuous monitoring of the patient's oxygenation, ventilation, circulation, and temperature
3. Oxygen concentrations of inspired gas: low oxygen concentration alarm
4. Blood oxygenation: pulse oximetry
5. Ventilation: observation, end-tidal carbon dioxide detection, and disconnect alarm
6. Circulation: EKG, arterial blood pressures every 5 minutes or continuous invasive blood pressures, and pulse oximetry
7. Understand that the standards of anesthesia care and patient monitoring are the same regardless of the location.

Remote Facilities and Equipment¹

1. Know the physical layout of the location, unfamiliar anesthetic equipment, and anesthetic implications of the procedure being performed prior to the induction of anesthesia.
2. Verify the availability of assistance.
3. Check piped-in gases and gas tanks.
4. Check suction.
5. Check power outlets (i.e., grounding and electrical requirements).

Remote Recovery Care¹

1. The patient must be medically stable before transport.
2. The patient must be accompanied to the recovery area by qualified anesthesia staff.
3. Provisions for oxygen delivery and monitoring on the transport cart are required.
4. Appropriate recovery facilities and staff must be provided.

Radiology Suite¹

1. Includes ultrasound (US), computed tomographic scanning (CT), magnetic resonance imaging (MRI), radiofrequency ablation (RFA), and neurosurgical embolization and coiling
2. The rooms are often crowded with bulky equipment.
3. Patients are often required to hold still for long periods of time.
4. Unique hazard
 - a. Radiation exposure
 - i. Leukemia and fetal abnormalities

97. Remote Anesthesia

- ii. Dosimeters are required to measure the level of radiation exposure a staff member receives. (A maximum exposure of 50 millisieverts (mSv) annually, a lifetime of 10 mSv × age, and monthly exposure of 0.5 mSv for pregnant women is allowed.)
- iii. Exposure can be limited by lead aprons, thyroid shields, leaded glass screens, protective goggles, and remote video monitoring.
- b. Iodinated contrast media
 - i. Older ionized contrast media were hyperosmolar and toxic.
 - ii. Newer non-ionized contrast media have lower osmolality and improved side-effect profiles.
 - iii. Overall incidence of an adverse drug reaction from contrast media is about 3%.
 - iv. The incidence of a severe reaction is 0.04%.
- c. High magnetic fields
- d. High-voltage equipment

Reactions to Iodinated Contrast Media

1. Mild: nausea, vomiting, perception of warmth, fever, chills, facial flushing, headache, itchy rash, and mild urticaria
2. Moderate: edema, bronchospasm, hypotension, and seizures
3. Severe: vomiting, rigors, feeling faint, chest pain, severe urticaria, bronchospasm, dyspnea, arrhythmias, and renal failure
4. Life-threatening: glottic edema/bronchospasm, pulmonary edema, prolonged hypotension, loss of consciousness, arrhythmias, cardiac arrest, and seizures/unconsciousness
5. Associated renal dysfunction due to contrast-induced nephropathy
 - a. The contrast media is eliminated by the kidneys.
 - b. Increased risk in patients with pre-existing renal dysfunction, especially if it is related to their diabetes, or patients on chronic non-steroidal anti-inflammatory drugs
 - c. Reduction in nephrotoxicity is associated with proper fluid hydration, careful urine output monitoring, and the use of low-osmolality contrast media.
 - d. Studies have shown benefits with the pre-procedure administration of acetylcysteine or sodium bicarbonate.
6. Pre-treatment: oral methylprednisolone
7. Treatment of a reaction: call for help, stop the injection of the causative agent, place the patient on oxygen, secure the airway, and give bronchodilators, fluids, epinephrine, corticosteroids, and antihistamines.

CT

1. Two-dimensional, cross-sectional image
2. Each cross-section requires a few seconds of radiation exposure.
3. Patient immobility is required.

4. It is often noisy, warm, and can induce claustrophobia.
5. CT can be used for diagnostic and therapeutic purposes.
6. The number one problem is inaccessibility to the patient.

Indications for CT

1. Diagnostic purposes
2. Vascular malformations
3. Tumors
4. Invasive therapeutic procedures
5. CT-guided radiofrequency ablation
6. Head trauma
7. Acute stroke
8. Primary intracerebral hemorrhage
9. Meningioma
10. Aortic dissection screening

MRI

1. Able to obtain images in any plane
2. Excellent soft tissue contrast
3. Does not produce ionizing radiation, is non-invasive, and does not produce biologically deleterious effects
4. MRI is often very time-consuming and any patient movement, including physiologic motion, can produce artifacts.
5. Obese patients can often not fit within the magnet.
6. Hearing protection is mandatory (produces loud noises >90 dB).
7. Thermal injury has been reported at the site of the EKG electrodes and areas where skin contacts the machine.
8. The most significant risk in the MRI suite is the effect of the magnet on ferrous objects.

Indications for MRI

1. MRI can produce better resolution between gray and white matter.
2. Evaluation of blood flow and cerebrospinal fluid
3. Contraction and relaxation of organs
4. Images of tissue surrounded by bone because calcium does not emit a MRI signal
5. Evaluating the posterior fossa and parasellar regions of the brain
6. Diagnosis of multiple sclerosis, epilepsy, and brain tumors
7. Better than CT for small brain tumors
8. Arteriovenous malformations
9. Multiple sclerosis
10. Syringomyelia
11. Congenital spinal cord abnormalities

Contraindications for MRI

1. Shrapnel, vascular clips and shunts, wire spiral endotracheal tubes, pacemakers, automatic implantable cardioverter-defibrillators (AICD's), mechanical heart valves,



Figure 97.1. Chair in the MRI scanner. Source: Courtesy of Moriel NessAiver, PhD, www.simplyphysics.com.

recently placed sternal wire, implanted biological pumps, and intra-ocular ferromagnetic foreign bodies.

2. Tattoo ink with high concentrations of iron-oxide (permanent eyeliner) can cause burns in a rare instance but is not an absolute contraindication.
3. Ferromagnetic items should never be allowed in the vicinity of the MRI magnet, including scissors, pens, keys, gas cylinders, anesthesia machine, anesthesia monitors, syringe pumps, pagers, phones, and steel chairs.
4. Cards with magnetic strips will be de-magnetized, including credit cards and identification badges.

Cerebral Coiling

1. This case should be treated like a cerebral artery aneurysm clipping in the operating room.
2. You should use all of the same intravenous access and monitors.
3. Place a pre-induction arterial line.
4. Always have two large-gauge IV's in place.
 - a. One for drug infusion
 - b. One for rapid fluid administration
5. General anesthesia with an endotracheal tube is usually required. The case can be long and the patient is required to hold still. Consider a muscle relaxant infusion to help ensure patient immobility during critical coiling events. Check for adequate relaxation with a twitch monitor. Communication with the radiologist is important.
6. Have a fluid warmer and forced air/gel pad warmer available to warm the patient after the procedure. Mild hypothermia may be requested during the procedure.
7. Infusion pumps to deliver medications.

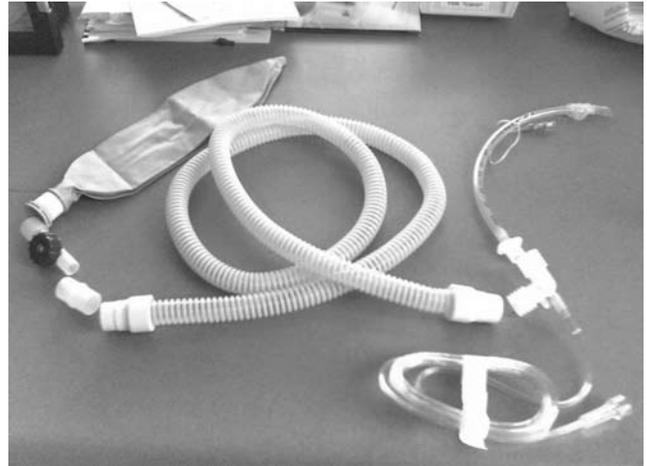


Figure 97.2. Long-distance MRI ventilation set-up. Photo credit: J. Lovich-Sapola, MD.

8. Medications (including but not limited to)

- a. Nitroglycerine
 - b. Nipride
 - c. Esmolol
 - d. Labetalol
 - e. Heparin
 - f. Protamine
 - g. Mannitol
 - h. Phenytoin
 - i. Thiopental
 - j. Dexamethasone
9. An activated clotting time (ACT) machine should be available because heparin is usually given during the procedure.
 10. Stay in constant communication with the operating room in case of an emergency.
 11. The patients are often transported directly to the intensive care unit post-operatively.



KO TREATMENT PLAN

MRI Road Trip; What to Bring

1. Anesthesia cart (pediatric or adult) fully stocked
2. Anesthesia machine/circuits (pediatric or adult)
3. Monitors for transport
4. End-tidal CO₂ monitor
5. Temperature monitor
6. MRI-compatible monitoring equipment is usually available in the MRI suite. Remember that the usual anesthesia monitors are not safe in the MRI scanner.
7. Airway equipment: oral and nasal airways, nasal canula, masks, laryngeal mask airways, and endotracheal tubes
8. Long corrugated ventilation tubing: the anesthesia machine must often be placed a long distance from the patient.
9. Self-inflating resuscitator bag

10. Syringe pump and at least 3 extension sets
11. Medications (including but not limited to)
 - a. Propofol
 - b. Remifentanyl
 - c. Ketamine
 - d. Midazolam
 - e. Fentanyl
 - f. Succinylcholine
 - g. Non-depolarizing muscle relaxants
 - h. Ephedrine
 - i. All other emergency drugs
12. IV tubing and IV fluids
13. Papers needed for charting

MRI Safety

1. Keep all ferromagnetic objects away from the MRI scanner.
2. Always wear ear protection secondary to the loud noises >90 dB.

MRI Anesthetic

1. Initial workup:
 - a. Vital signs
 - b. Pre-operative history
 - c. Physical examination
2. Set up your equipment and familiarize yourself with physical layout and location.
 - a. Verify the availability of assistance
 - b. Check gases, suction, and the MRI monitors.

3. Induce the patient in the holding area on the MRI-safe cart, and then transport the patient to the MRI.
4. Do not take metal into the MRI room!
5. Leave the monitors, anesthesia machine, oxygen cylinder, etc. outside of the MRI room.
6. Place the patient on the MRI table, connect the patient to the inline oxygen in the MRI scanner, and apply the MRI-compatible monitors already available in the MRI suite.
7. ASA standard monitors are required.
 - a. EKG
 - b. Pulse oximetry
 - c. Non-invasive blood pressure
 - d. Capnography
 - e. Temperature
8. Maintain anesthesia either by intravenous medications given with an MRI-compatible pump or inhaled anesthetics given by an MRI-compatible anesthesia machine.
9. At the end of the case, take the patient back to the holding area and extubate there.
10. Transport the patient to the post-anesthesia care unit along with the ASA standard monitors.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2637-63.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 1331-42.
3. What Are the Clinical Indications for MRI, and How Does It Compare to Other Diagnostic Modalities? NIH Consensus Development Program-Outdated Consensus Development Reports. <http://consensus.nih.gov>.

98. Awareness Under Anesthesia^{1,2}

Donald M. Voltz, MD

SAMPLE CASE

A 46-year-old female with temporal-mandibular joint (TMJ) disease presents for a TMJ arthroplasty. This patient is a 155 cm, 62 kg woman with no known allergies or medical problems. She conveys to you that when she underwent a tubal ligation 7 years ago, she remembers waking up during the surgery. As she describes her previous anesthetic misadventure, her anxiety level increases. What do you make of the previous anesthetic history in this patient? What would you tell this patient to alleviate her fears for this surgery? What are your anesthetic plan and management options for

a patient with a prior episode of anesthetic awareness? What patients are at the highest risk of experiencing awareness under anesthesia?

CLINICAL ISSUES

Definitions

1. Awareness under anesthesia
 - a. Awareness under anesthesia occurs when a patient is targeted for general anesthesia but at some point

98. Awareness Under Anesthesia

during this plan, the patient emerges from a plane of surgical anesthesia into one where cortical activity is restored.

- b.** This can occur during a period of
 - i.** Decreased anesthetic delivery: hemodynamic compromise not allowing for adequate level of anesthesia
 - ii.** Increase in surgical stimulation for the given level of anesthetic agents: transition from surgical prep to surgical incision without adequate time for anesthetic deepening
 - iii.** Error in the delivery of anesthetic agents: error in delivery of anesthetic such as vaporizer under-filled or malfunctioning
 - c.** Awareness under anesthesia is a period when patients become aware of their surroundings when they should be amnesic of the events that occur during general anesthesia.
 - d.** This awareness may or may not be remembered by a patient after the surgical procedure is completed.
 - e.** An example of awareness under anesthesia is a patient purposefully reaching for the endotracheal tube immediately after intubation when an inadequate amount of anesthetic medication was delivered or not enough time had passed prior to laryngoscopy. In this situation, a patient demonstrated purposeful movement that resulted from an inadequate level of anesthesia for the given level of stimulation. This episode is not likely to be remembered after the procedure is completed; however, it may be.
 - f.** Patients with awareness appear to be responding appropriately to stimulation or commands. However, they do not remember the circumstances following their anesthetic.
- 2.** Intra-operative recall
 - a.** Intra-operative recall occurs when patients are able to remember specific events during the course of general anesthesia when they should have been completely amnesic.
 - b.** All patients with intra-operative recall also had awareness under anesthesia.

TKO: Remember, you may have awareness under anesthesia without recall!

Memory Types

- 1.** Explicit memory
 - a.** Conscious recollection of information or experiences by a patient
 - b.** This has also been called “direct memory”; it is tested by asking someone to recall what he or she remembers about a specific event.
 - c.** In the context of intra-operative recall, a patient with explicit memory of an event would be able to describe specific events that took place, such as the surgeon splitting the sternum during cardiac surgery or parts of a conversation that took place between operating room personnel.
- 2.** Implicit memory

- a.** The cortical processing of a sensory stimulation without resulting conscious recollection of that stimulus
- b.** During the course of a surgical procedure under general anesthesia, patients can have subconscious processing of auditory or other sensory stimuli that they are later unable to consciously recall.
- c.** In tests when patients were given paired words during various times under general anesthesia, the patients were able to provide the correct word when one of the pair was given to them.
- d.** It has been suggested that implicit memory of intra-operative events occurs more frequently than explicit recall; however, these types of studies are difficult to perform as well as interpret.

Incidence

- 1.** The actual incidence of both intra-operative recall and awareness under anesthesia is unclear due to the difficulty in studying these phenomena as well as not having an objective means of measuring a patient’s level of consciousness.
- 2.** Despite research difficulties, this is a defined complication of anesthesiology with a suggested rate of awareness being between 0.02 and 1.0%.
- 3.** These suggested rates are for true explicit recall. The incidence of awareness under anesthesia (implicit memories) has been suggested to be higher.

Legal Implications

- 1.** ASA Closed Claims Analysis reports 1.9% of cases were related to awareness while British claims related to awareness were 12.2% of all cases brought against anesthesiologists.
- 2.** In some of these cases, cues to a light anesthetic were absent. Hemodynamic cues such as hypertension (15% of cases where awareness was reported) or tachycardia (7% of cases where awareness was reported) were not uncovered during retrospective analysis of the cases.

Stages of Awareness

Transition from an awake state to one of adequate surgical anesthesia is a continuum that varies among patients and is influenced by the amount of surgical stimulation as well as the patient’s physiological state. To obtain a better understanding of the progression from awake to amnesic states, Griffith and Jones define five perceptual stages that occur in general anesthesia.

- 1.** Conscious perception of stimuli with explicit memory of the events
- 2.** Conscious perception of stimuli without explicit memory of the events
- 3.** Dreaming
- 4.** Subconscious perception of stimuli with implicit memory of the events
- 5.** No perception of stimuli and no memory of the events

Risk Factors for Awareness under Anesthesia and Intra-operative Recall

1. Cardiac surgery
2. Emergency cesarean section
3. Trauma
4. Emergency surgery in patients with minimal physiologic reserve
5. Surgery in which neuromuscular blocking agents are used
6. Equipment failures
7. Patients with higher than normal anesthetic requirements: previous drug use and the development of tolerance

Prevention

1. Delivery of anesthetics by well-trained individuals
2. Vigilance of medications delivered and the patient's response to them
3. Repeated checking of the medication delivery systems: infusion pumps, vaporizers
4. Avoidance of neuromuscular blocking agents unless required for the procedure
5. Continual monitoring and trending of anesthetic delivery and patient's physiologic variables
6. In situations when anesthesia may be light (cardiac, cesarean sections), inform the patient about the risk and ensure the patient that you will do everything possible to prevent awareness during these periods.

TKO: Depth of anesthesia/bispectral index monitors (BIS) are not completely reliable for all individuals and all cases. In the future, the reliability of these monitors may improve.

Treatment

1. Intra-operative awareness with recall is very disconcerting to the patients who experience this.
2. The feeling of helplessness and an inability to move or communicate this to their anesthesiology provider results in a high incidence of post-traumatic stress disorder (PTSD).
3. Review of these cases post-operatively often does not reliably uncover when or why the patient might have been aware.
4. In most cases of awareness, the classic signs of hypertension or tachycardia are absent.
5. In light of this, it is important to take every case seriously and initiate steps to help these patients.

6. Directly address a patient's fears and concerns in a sympathetic manner as well as arrange for these patients to be seen by a psychiatrist who has expertise in the area of PTSD treatment.



KO TREATMENT PLAN

Pre-operative

1. A discussion of the patient's experience might be helpful in gaining additional information about her prior episode of anesthesia awareness and recall.
2. Reassure the patient that you will closely monitor her clinical condition and ensure that all steps are being taken to prevent awareness from occurring this time.
3. You might consider modifying your anesthetic plan from the previous one.
4. A review of her current medications as well as an investigation into what was administered during the previous case when awareness occurred could factor into the formulation of the new anesthetic plan.
5. Supplementing a volatile anesthetic with a propofol infusion with midazolam might lead to a more memory-suppressant anesthetic.
6. Currently, the ASA Practice Guidelines do not recommend the routine use of intra-operative depth of anesthesia (BIS) monitors for all cases of general anesthesia.
 - a. The ASA does support the use of these monitors. However, the ASA also recommends multimodality monitoring such as heart rate, blood pressure, and expired volatile anesthetics.
 - b. Diligent follow-up and intervention in cases of suspected or confirmed intra-operative recall and or awareness
7. Vigilant clinical signs monitoring with the addition of depth of anesthesia monitoring will provide data for intra-operative clinical management of this patient.
8. Following the anesthetic, it is important to speak with the patient to ensure that no recollection occurred during this case and provide support since her earlier experience might require further intervention, even if no recall or awareness occurred during this procedure.

BIBLIOGRAPHY

1. Griffith D, Jones JB. Awareness and memory in anaesthetized patients. *Br J Anaesth*, 1990;65:603-7.
2. Sebel PS. Awareness and Memory during General Anesthesia. ASA Annual Meeting Refresher Course Lectures, 2007.

99. Laser Safety^{1,2,3,4,5}

Aditya Reddy, MD and Jessica A. Lovich-Sapola, MD

SAMPLE CASE

A 24-year-old male professional recording artist presents to an otolaryngologist with the complaint of hoarseness. On exam he was found to have vocal cord nodules and a laser laryngoscopy was scheduled. This patient has no significant medical history. His surgical history is significant for a tonsillectomy and adenoidectomy as a child under general anesthesia without any complications. What type of laser may be used? What safety precautions will be necessary in the operating room?

CLINICAL ISSUES

Definition of laser: an acronym that stands for *Light Amplification by Stimulated Emission of Radiation*

Indications for Laser Treatments

1. Vocal cord nodules and polyps
2. Malignant neoplasm: vocal cord cancers
3. Laryngoceles and benign cyst in the laryngoceles
4. Recurrent respiratory papillomatosis
5. Subglottic hemangioma
6. Laryngomalacia
7. Congenital and acquired anterior glottic webs
8. Subglottic and glottic stenosis
9. Condyloma acuminatum of external genitalia and urethra
10. Ureteral stricture or bladder neck contracture
11. Carcinoma of the penis, bladder, ureter, and renal pelvis
12. Laser resurfacing for facial cosmetic procedures (such as perioral, periorbital creases and wrinkles)
13. Laser ablation of endometriosis
14. Coagulation of small or superficial blood vessels

Different Types of Medical Lasers and Their Specific Applications

1. Argon Laser
 - a. It is absorbed by hemoglobin.
 - b. It has a modest tissue penetration between 0.05–2 mm.
 - c. It provides the ability to penetrate skin or ocular structures and selectively coagulate vascular or pigmented regions.
 - d. It is mainly used in ophthalmologic and dermatologic procedures.

2. Potassium-titanyl-phosphate (KTP) or yttrium aluminum garnet (YAG) laser
 - a. Absorbed by hemoglobin
 - b. Has a penetration similar to the argon laser
3. Neodymium-doped yttrium aluminum garnet (Nd-YAG) laser
 - a. Very powerful
 - b. Tissue penetration between 2 and 6 mm
 - c. It can be used for tumor debulking – particularly the upper airway, trachea, and main-stem bronchi.
4. Carbon dioxide (CO₂) laser
 - a. Very little tissue penetration
 - b. Best precision
 - c. Can be used for cutting
 - d. It is absorbed by water; hence, minimal heat is dispersed to the surrounding tissues.
 - e. It is primarily used for procedures in the oropharynx and around the vocal cords.
5. Helium-Neon (He-Ne) laser
 - a. Produces an intense red light
 - b. Used for aiming the CO₂ and Nd-YAG lasers
 - c. It has very low power and will not cause any significant damage.
6. Ruby laser
 - a. Primarily used for tattoo and nevi removal
 - b. Absorbed well only by cells containing dark pigment

Complications and Hazards of Laser Surgery

1. Vaporization of tissue by lasers can produce a plume of smoke and fine particulates that can be deposited in lungs.
 - a. Viral DNA has been detected in smoke from condylomas and skin warts.
2. Perforation of larger blood vessels that are not coagulable by the laser
3. Laser-induced pneumothorax: after laryngeal procedures
4. Venous gas embolism: especially with Nd-YAG lasers during hysteroscopic surgery
5. Corneal injury: CO₂ lasers
6. Retinal injury: Argon, KTP, Nd-YAG, and ruby laser
7. Thermal injury to the skin
8. Endotracheal tube fires

Anesthetic Technique/Management for Laser Surgery

1. The most effective way to prevent operating room personnel from exposure to the viral and chemical content of the laser plume is to use a smoke evacuation system with the suction being held close to the tissue being vaporized.
 - a. In addition, operating room personnel should wear gloves and high-efficiency filter masks.
2. The operating room staff and the patient should have proper eye protection during laser surgery.
 - a. Safety goggles or lenses specific for the specific laser in use
 - b. Protective covers over the patient's eyes
3. One of the major caveats during laser surgery (laryngoscopy) is to supply a gas mixture with the lowest potential for combustion.
 - a. Ideally the lowest inspired oxygen concentration required to safely oxygenate the patient should be used.
 - b. The ideal mixture would be air and oxygen or helium and oxygen.

TKO: Remember, nitrous oxide (N₂O) is as combustible as oxygen.

4. Total intravenous anesthesia (TIVA) is preferred because volatile anesthetics can potentially decompose into toxic compounds when exposed to airway fires.
 - a. Modern volatile anesthetics are non-flammable and non-explosive.
5. Protect the endotracheal tube with metallic tape, or use a laser-resistant endotracheal tube.
 - a. Traditional endotracheal tubes
 - i. Polyvinyl chloride (PVC)
 - (1) Inexpensive and non-reflective
 - (2) Low melting point and highly combustible
 - ii. Silicone rubber
 - (1) Non-reflective
 - (2) Combustible and turns into toxic ash with fire
 - iii. Red rubber
 - (1) Puncture resistant and non-reflective
 - (2) Highly combustible
 - b. Metal endotracheal tubes
 - i. Combustion and kink resistant
 - ii. Cumbersome, flammable cuff; transfers heat and reflects the laser
 - iii. Types
 - (1) Bivona Fome-Cuff
 - (a) Aluminum spiral tube with a silicone polyurethane foam cuff in a silicone envelope
 - (b) Recommended for use with the CO₂ laser
 - (c) The foam part of the cuff is self-inflating and remains inflated if the envelope ruptures.
 - (d) High incidence of sore throat
 - (2) Xomed Laser-Shield
 - (a) Silicone elastomer tube containing metallic powder
 - (b) To be used with the CO₂ laser only

- (c) Risk of perforation and fragmentation into silica ash
- (3) Mallinkrodt Laser-Flex
 - (a) Airtight stainless steel, spiral wound endotracheal tube
 - (b) It has 2 separate PVC cuffs.
 - (i) The cuffs should be filled with colored saline.
 - (ii) Recommended for CO₂ and KTP and Nd-YAG lasers (not the original YAG laser)
- c. Wrapped endotracheal tubes
 - i. Aluminum foil, copper foil, or a thin metal coated plastic tape
 - ii. Disadvantages
 - (1) No cuff protection
 - (2) Adds thickness to the endotracheal tube
 - (3) Not FDA approved
 - (4) Reflective
 - (5) Rough edges may damage the mucosa
6. Inflate the endotracheal tube cuff with water or saline (+ methylene blue to facilitate leak detection).

Treatment Plan for Airway Fires

In case of an airway fire, the surgeon (usually the first person to recognize an airway fire) and anesthesiologist must act quickly, decisively, and in coordinated fashion. The following steps must be taken:

1. Remove the source of the fire.
2. Stop ventilation by disconnecting the breathing circuit from the anesthesia machine.
3. Remove the object on fire (usually the endotracheal tube).
4. Ventilation with 100% oxygen should be provided by mask.
 - a. Stop all volatile anesthetics.
 - b. Continue intravenous anesthetics.
5. Direct laryngoscopy and rigid bronchoscopy should be performed to examine the airway and remove any debris if present.
6. Reintubate if needed.
7. Evaluate the face and oropharynx.
8. Chest X-ray
9. Distal fiber optic bronchoscopy as needed
10. Severe damage may require a low tracheostomy.
11. Consider steroids.

**KO TREATMENT PLAN****Pre-operative**

1. The CO₂ laser is widely used for vocal cord surgery since it can precisely vaporize superficial tissue.
2. The patient's eyes must be protected with moistened gauze and taped.
3. All of the operating room personnel must wear protective goggles.

4. The laser should always be placed in “stand by” mode when not being used.

Intra-operative

1. Induction of anesthesia should be performed in a standard manner.
 - a. Standard ASA monitors
 - b. Intravenous propofol can be used as an induction agent because of its rapid onset and short duration of action.
 - c. Rocuronium is a good choice for muscle relaxation since it is very important that the vocal cords must remain motionless during laser surgery.
2. A smaller diameter, laser-safe endotracheal tube (Mallinkrodt Laser-Flex) should be placed to facilitate visualization of the larynx and vocal cords.
3. Place methylene blue-dyed normal saline in the endotracheal tube cuff(s).
 - a. This will help the surgeon to identify if he or she has ruptured the cuff with the laser.
4. Use the lowest possible FiO₂ (<0.3–0.4) and avoid nitrous oxide.
 - a. Both oxygen and nitrous oxide support combustion.
5. Maintenance of anesthesia
 - a. TIVA
 - i. Propofol and remifentanyl infusion

- ii. Especially useful with jet ventilation and/or intermittent apnea techniques.
- b. When the endotracheal tube is used for the entire case, then volatile anesthetic gases may be used in addition to the propofol and remifentanyl infusions.

Post-operative

1. Be vigilant for complications resulting from the laser use
 - a. Eye trauma
 - c. Airway compromise
 - d. Esophageal perforation
 - e. Pneumothorax/mediastinal air: may present as hypotension and cardiovascular collapse.

BIBLIOGRAPHY

1. Miller Ronald D, Fleisher Lee A, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 2542–3, 2573–85.
2. Reed P. Allan, Yudkowitz S. Francine. *Clinical Cases in Anesthesia*, 3rd ed. Philadelphia: Churchill Livingstone, 2005, pp 265–8.
3. Jaffe A. Richard, Samuels I. Stanley. *Anesthesiologist's Manual of Surgical Procedures*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2004, pp 150, 235–8, 434, 606–8, 685, 882, 962.
4. Barash G. Paul, Cullen F. Bruce, Stoelting K. Robert. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 91, 170–71, 1032–33.
5. Faust J. Ronald, et al. *Anesthesiology Review*, 3rd ed. Philadelphia: Churchill Livingstone, 2002, pp 250–51.

100. Electrical Safety in the Operating Room^{1,2,3,4,5}

Sheila Chiu-Miller, DO and Jessica A. Lovich-Sapola, MD

SAMPLE CASES

A broken equipment ground wire that is leaking 0.1 mA of current ends up conducting through an indwelling right ventricular catheter of a patient undergoing coronary artery bypass graft (CABG) surgery and causes ventricular fibrillation. Is this an example of microshock or macroshock? How could it have been prevented?

CLINICAL ISSUES

Definitions

1. Ohm's law: $I = V/R$
 - a. I = Current (ampere)
 - b. V = Potential (volt)
 - c. R = Resistance (ohms)

Most Common Electrical Hazards in the Operating Room

1. Electrical shock
 - a. Macroshock
 - i. Current >1 mA
 - ii. Very rare occurrence secondary to the following safety measures:
 - (1) Isolation transformers
 - (a) The operating room electricity is separated from the hospital's main power source through a secondary circuit (see Figure 100.1).
 - (b) The line source from the electrical provider is grounded but the secondary circuit is not.
 - (c) In order to complete a circuit and cause a shock, both conductors must be contacted; if

only one conductor is contacted, no electrical current will pass through the body.

- (2) Line isolation monitor (LIM)
 - (a) Keeps the integrity of an isolated power system in check.
 - (b) It is only a monitor; it does not prevent a shock.
 - (c) Detects short circuits or current leakage and sets off an alarm.
 - (d) Monitors the isolation of the transformer from the ground.
 - (e) The LIM reads 0 Amp when there is no leakage.
 - (f) The monitor is usually set to alarm at 2 to 4 mA
 - (i) This alarm is not useful for leakage currents in the microshock range.
 - (g) If the ungrounded side of the circuit becomes grounded due to a short in the circuit, touching any part of the circuit will make a complete path and allow the current to pass through the body, causing a shock.
 - (i) Severity of the shock correlates with the intensity of the current (Table 100.1).
 - (3) Ground fault circuit interrupter (GFCI)
 - (a) A special circuit breaker that helps to avoid electrical shock in a grounded power system
 - (b) It cuts off electrical supply to faulty equipment without warning.
 - (i) It is rarely found in an operating room setting secondary to the danger of life-saving equipment shutting off without warning.
 - (ii) A GFCI is often used in residential homes: kitchens and bathrooms.
- b. Microshock**
- i. Small current (i.e., 0.05–0.1 mA)
 - ii. Can place susceptible patients with direct external connection to the heart (e.g., intracavitary or epicardial pacemaker wires or indwelling central access catheters) at risk of cardiac dysrhythmias including ventricular fibrillation.
 - iii. Patients are not protected from microshock by isolation transformers.
 - iv. Microshock only occurs when skin resistance is bypassed.
 - v. Only a hazard when there is a direct external electronic device connecting a current through a patient to the ground AND the device has a faulty ground.
 - vi. National code requires less than 0.01 mA maximum permissible leakage through electrodes or catheters that directly contact the heart.
- 2. Electrical or thermal burn**
- a. There are three requirements for a fire:
 - i. Oxygen
 - ii. Fuel
 - iii. Ignition source
 - b. Major ignition sources in the operating room are electrosurgical units (i.e., bipolar or unipolar electrocautery) and the laser.

- c. On rare occasions, the end of a fiber optic light cord can get hot enough to ignite a fire on the paper drapes.
- d. Fires can also result from improper grounding of equipment.

Determinants of Electrical Hazards

1. Type of current
 - a. Direct current (DC)
 - i. One direction
 - ii. Non-oscillating current
 - iii. A constant voltage crosses a resistor (e.g., DC through skeletal and cardiac muscle causes a sustained contraction).
 - b. Alternating current (AC)
 - i. Current that changes polarity at a specific rate (frequency)
 - ii. Lower frequency AC (standard 60 Hz) causes skeletal muscle tetany and likely ventricular fibrillation even with a small current.
 - c. Current density
 - i. The extent of disruption by an electric current is inversely proportional to the area at which the current is applied (a smaller caliber catheter has a higher current density and requires a lower amount of current to cause fibrillation).
 - d. Current duration
 - i. 2–4 microseconds: cardiac muscle can recover fast.
 - ii. >1 second(s): the muscle can become depolarized with a decreased possibility of recovery.
 - e. Skin resistance
 - i. Intact dry skin = 1 million ohms of resistance
 - ii. When skin is wet, the resistance can drop to 1,000 ohms.
 - iii. A central venous catheter penetrating the skin lowers the resistance to 500 ohms, increasing the risk for microshock.
 - (1) Decreased resistance equals an increased risk for an electrical shock.
 - f. Current threshold
 - i. An AC current frequency of 60 Hz or greater is required to stimulate ventricular fibrillation.

CASE DISCUSSION

1. The sample case is an example of a microshock.
 - a. A small current leak such as 0.1 mA (100µA) can cause a microshock to a susceptible patient, such as this patient with a direct central access to the right ventricle via the catheter.
 - b. This current is sufficient to cause ventricular fibrillation.
 - c. Prompt recognition of the problem and early removal of the catheter causing the fibrillation can facilitate more effective resuscitation intra-operatively.

100. Electrical Safety in the Operating Room

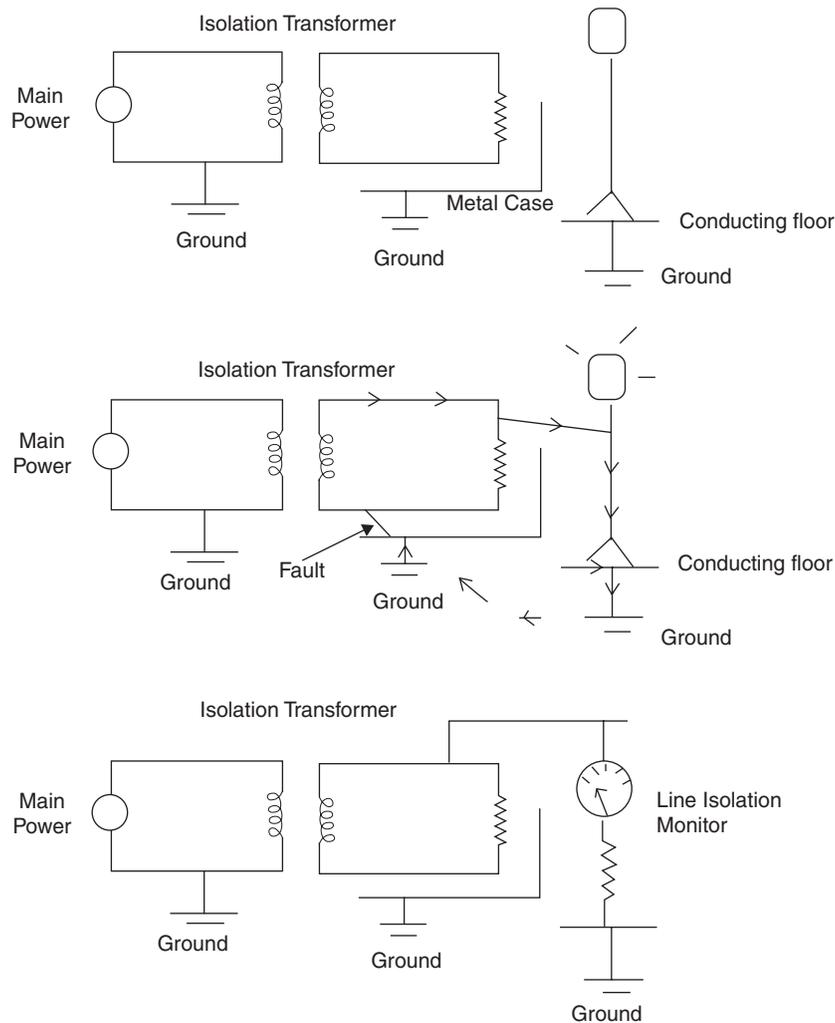


Figure 100.1. A. No electric shock occurs if an isolated power line is touched. B. An electric shock does occur if a faulted secondary power line is touched. C. A line isolation monitor can watch for a fault. *Source:* This figure was published in *Anesthesia, 6th Edition*, RD Miller, LA Fleisher, RA Johns, et al., Page 3142, Copyright Elsevier (2005). Adapted with permission.

Table 100.1. Effects of 60 Hz AC Current on an Average Human for 1 Second Duration of Contact

Current (milliamperes, mA)	Event	Shock level
0.01 mA	Maximum recommended 60 Hz leakage current	Microshock
0.1 mA	Ventricular fibrillation	Microshock
1 mA	Threshold of perception	Macroshock
5 mA	Maximum harmless current intensity	Macroshock
10–20 mA	“Let go” value exceeded, sustained muscle contraction next	Macroshock
50 mA	Pain, exhaustion, mechanical injury, possible fainting, cardiorespiratory functions continue	Macroshock
100–300 mA	Ventricular fibrillation, respiratory center intact	Macroshock
>6000 mA	Sustained myocardial contraction followed by normal rhythm, transient respiratory center paralysis, burns when current density >100mA/cm ²	Macroshock

Adapted from Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 149–74.

2. Prevention of current leakage pre-operatively is the key to avoiding this adverse event.
 - a. Regular maintenance of the equipment in the operating room can be very helpful.



KO TREATMENT PLAN

Pre-operative

1. Understand the electrical construction pattern of the operating room.
2. Know what the alarms mean and don't ignore them (e.g., when the LIM alarm sounds, a low resistance between the isolated power lines and ground is detected and should not be ignored).
 - a. When the LIM alarm sounds, each piece of electrical equipment should be unplugged one at a time, starting with the most recently plugged in, until the faulty piece of equipment is identified.
3. Always identify the nearest fire extinguisher in the operating room.
4. Always have a plan on how to react in case of a real fire.
5. Regular maintenance of equipment is important to ensure proper functioning.

Intra-operative

1. Be vigilant.
2. Make sure that a grounding pad is placed properly on the patient if electrocautery is used.
 - a. If the pad is folded, the patient can be burned.
 - b. The pad should be rechecked during long cases and should be replaced if necessary.
3. Emergency equipment should be immediately available in case of an electrical shock or fire.

BIBLIOGRAPHY

1. Hensley FA, Martin DE, and Gravlee GP. *A Practical Approach to Cardiac Anesthesia*, 4th ed. Lippincott Williams & Wilkins, 2008, pp 138–40.
2. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 3139–49.
3. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 149–74.
4. Erie, JK. Line isolation monitors: Leakage currents. In Faust, RJ et al., eds. *Anesthesiology Review*, 3rd ed. New York: Churchill Livingstone, 2002, pp 247–8.
5. Johnson, ME. Microshock hazards. In Faust, RJ, Cucchiara, RF, Rose, SH, Spackman, TN, WedelDJ, Wass, CT, eds., *Anesthesiology Review*, 3rd ed. New York: Churchill Livingstone, 2002, p 249.

101. Ethics^{1,2}

Jessica A. Lovich-Sapola, MD

CLINICAL ISSUES

Definitions

1. Informed Consent
 - a. The cornerstone of the patient-physician relationship
 - b. It is a legal condition whereby a person gives consent based upon an appreciation and understanding of the facts, implications, and future consequences of an action.
 - c. The person must have adequate reasoning faculties and be in possession of all of the relevant facts at the time that consent is given.
 - d. The goal is to maximize the ability of the patient to make substantially autonomous informed decisions.
 - e. It is unreasonable to expect a patient with no prior medical training to be fully informed.
2. Negligence
 - a. Conduct that is culpable because it falls short of what a reasonable person would do to protect another individual from a foreseeable risk of harm
 - b. May occur if the anesthesiologist provides a disclosure that is insufficient to allow a patient to make an informed decision and an injury subsequently occurs, even if the injury was foreseeable and in the absence of medical error.
3. Causation
 - a. The causal relationship between conduct and the result.
 - b. Assesses whether the omitted information would have caused the patient to choose a different option.
4. Exception to the obligation of full disclosure occurs with emergencies and situations of therapeutic privilege.

5. A competent patient has the right to refuse life-sustaining medical treatment.
6. All do not resuscitate (DNR) orders should be reevaluated pre-operatively in light of the surgical and anesthetic options.

Malpractice Suit²

The patient must prove all four of the following:

1. Duty: the anesthesiologist owed the patient a duty.
2. Breach of duty: the anesthesiologist failed to fulfill his or her duty.
3. Causation: a reasonably close causal relation exists between the anesthesiologist's actions and the resultant injury.
4. Damages: the actual damage resulted because of the breach of the standard of care.

Organ Procurement

1. Brain death
 - a. The irreversible end of all brain activity, including involuntary activity necessary to sustain life, due to total necrosis of the cerebral neurons following a loss of blood or oxygenation
 - b. The patient has no clinical evidence of brain function upon physical examination.
 - i. No response to pain
 - ii. No cranial nerve reflexes: pupillary response, oculo-cephalic reflex, corneal reflex
 - iii. No spontaneous respirations
 - c. Before an official diagnosis of brain death can be made, conditions that mimic brain death must be ruled out.
 - i. Barbiturate intoxication
 - ii. Alcohol intoxication
 - iii. Sedative overdose
 - iv. Hypothermia
 - v. Hypoglycemia
 - vi. Coma
 - vii. Chronic vegetative states
 - d. Brain death is declared using neurologic criteria, prior to the patient's being taken to the operating room.
 - i. Requires a neurologic exam by two independent physicians
 - ii. The exam must show a complete absence of brain function.
 - iii. The exam may include two isoelectric EEGs 24 hours apart, but this is not mandatory.
 - iv. The patient must have a normal temperature and be free of any medications that can suppress the brain activity.
 - v. A radionuclide cerebral blood flow scan that shows the absence of intracranial blood flow can be used to confirm the diagnosis.
 - e. The non-living donor is kept on ventilator support until the organs have been surgically removed.

2. A more controversial topic is organ donation after cardiac death (DCD).

- a. Cardiac death
 - i. Death declared on the basis of cardiopulmonary criteria
 - ii. Irreversible cessation of circulatory and respiratory function
 - iii. It is determined that the patient has no significant likelihood for survival after the withdrawal of life support.
- b. Examples
 - i. Non-recoverable and irreversible neurological injury resulting in ventilator dependency but not fulfilling the brain death criteria
 - ii. End-stage musculoskeletal disease, pulmonary disease, and high spinal cord injury
- c. Protocol
 - i. A suitable candidate is selected.
 - ii. Consent is obtained from the next of kin to withdraw care and retrieve the organs.
 - iii. Life-sustaining measures are withdrawn under controlled circumstances in the intensive care unit (ICU) or operating room.
 - vi. When the donor meets the criteria for cardiac death, a physician pronounces the patient as dead. This physician can in no way be associated with the organ procurement due to the obvious conflict of interest.
 - v. The time from the onset of asystole to the declaration of death is usually 2-5 minutes.
 - vi. The organs, kidneys, liver, pancreas, lungs, and in some rare cases the heart, are recovered.
 - vii. If a patient does not die quickly enough to permit the recovery of organs, end-of-life care continues and any planned donation is canceled. This occurs in 20% of cases.
- d. This technique increases the number of organs available for donation.

Emergency Situations

1. Presume that the patient wants life-sustaining treatment unless he or she has declared otherwise.

Refusal to Provide Care

1. Anesthesiologists may refuse to provide care when they ethically or morally disagree with the procedure, such as an elective abortion.
2. Anesthesiologists are obligated to make a reasonable effort to find a competent, willing replacement.
3. They may also refuse to provide care if they are not qualified to provide the needed care.
4. An anesthesiologist cannot refuse care based on gender, race, or disease status.

Jehovah's Witness

1. Current doctrine
 - a. Blood is sacred to God.
 - b. Blood must not be eaten or transfused.
 - c. Blood that leaves the human body must be disposed of.
 - d. Therefore, the patient refuses blood products based on his or her religious beliefs.
2. Prohibited blood products
 - a. Red blood cells: allogenic and autologous
 - b. White blood cells
 - c. Platelets
 - d. Blood plasma
3. Usually accepted techniques
 - a. Hemodilution therapy
 - b. Intra-operative blood salvage
 - c. Cardio-pulmonary bypass
 - d. Dialysis
 - e. Epidural blood patch
 - f. Plasmapheresis
 - g. Blood labeling or tagging
4. Most accept synthetic colloid solutions, dextran, erythropoietin, and iron supplementation.
5. A court order can be obtained for a minor to receive blood products, even if the parents refuse.

Emancipated Minor

1. A legal mechanism by which a child is freed from control by his or her parents/guardians, and the parents/guardians are free from any and all responsibility to the child.
2. Patients less than 18 years old who have been given the global right to make their own health care decisions
3. Circumstances under which a person can become emancipated
 - a. Marriage
 - b. Having a child of his or her own.
 - c. Enlisted in the military
 - d. Court order from a judge
 - e. Pregnancy does not necessarily qualify one as an emancipated minor.

SAMPLE CASE #1

A 46-year-old male had an epidural placed for post-operative pain relief after a radical cystectomy. The epidural was

discontinued earlier this morning. You are called to see the patient for progressive bilateral lower extremity motor and sensory loss. You immediately begin testing to rule out an epidural hematoma or abscess. The patient tells you that your colleague never warned him of these potential risks. What do you say?

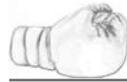


KO TREATMENT PLAN

These are never easy situations. The best approach is to talk openly and honestly with the patient. Tell the patient that while you are not sure what was discussed earlier with your colleague, you plan on doing everything to diagnose the current situation and prevent further progression of his symptoms. Do not say anything bad about your fellow physician. In the event of an ethical dilemma that is beyond your scope of practice, consult the hospital ethics service. The ethics committee will act in an advisory role to resolve the ethical dilemma.

SAMPLE CASE #2

Two weeks ago you were involved in a large trauma case during which 17 units of blood were given. The patient is currently doing well. She has been discharged to the rehabilitation site. The blood bank calls to notify you that one of the units of red blood cells that were given was crossed to a different patient. Do you tell the patient? If so, what do you tell the patient?



KO TREATMENT PLAN

It is your responsibility to notify the patient, despite the fact that no complications occurred. It would be appropriate to consult your legal department prior to the conversation and have a representative from the blood bank present to help answer any questions. It is never a good idea to lie or try to cover up information.

BIBLIOGRAPHY

1. Miller RD, Fleisher LA, Johns RA, et al. *Anesthesia*, 6th ed. New York: Churchill Livingstone, 2005, pp 3175-94.
2. Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006, pp 99-103.

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