PART ONE

General Information
SECTION 1. MONITORS, CIRCUITS, AND MACHINES

1. Standard ASA Monitors

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

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.1
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.2
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

Indications

4. Tool for early warning of hypoxemia.

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.
2. The pulse oximeter measures “functional” saturation.
2. Pulse Oximetry

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 \(\text{SpO}_2\) measurement will be higher than the \(\text{SaO}_2\) 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


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 (\(\text{EtCO}_2\)) 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 \(\text{CO}_2\) concentration versus time or expired volume. Capnography provides three sources of information: numerical \(\text{CO}_2\) values, the capnogram shape, and the arterial and end-tidal \(\text{PCO}_2\) difference.\(^1\)

2. Capnograph: the machine that generates the waveform.\(^1\)

3. Capnogram: the continuous graphic waveform representation of the \(\text{CO}_2\) 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 \(\text{CO}_2\) concentrations during a respiratory cycle.\(^1,3\)

5. Capnometer: the device that performs the measurement and displays the readings.\(^1\)

6. \(\text{EtCO}_2\): end-tidal carbon dioxide; the measurement of the concentration of \(\text{CO}_2\) at the end of exhalation. Normal value of partial pressure ranges between 35 and 45 mm Hg.\(^1\)

7. \(\text{PaCO}_2\): the partial pressure of \(\text{CO}_2\) in the arterial blood.\(^1\)

8. \(\text{a-ADO}_2\): the difference between \(\text{EtCO}_2\) and \(\text{PaCO}_2\). 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 \(\text{EtCO}_2\)
   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 \(\text{CO}_2\) 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

4. Electrocardiogram

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

\textit{Karl Wagner, MD}

\textbf{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?

\textbf{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.

\textbf{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).

\textbf{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\textsuperscript{1,2,3}

\textbf{Rhythm:} sinus or non-sinus

\textbf{Regularity:} regular or irregular

\textbf{P wave:} generated by atrial depolarization.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|}
\hline
Rate & Diagnosis \\
60–100 & Normal \\
<60 & Bradycardia \\
>100 & Tachycardia \\
\hline
\end{tabular}
\caption{EKG Rate}
\end{table}

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.

\textbf{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°.
   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. Additionally, patients who have EKG abnormalities pre-operatively along with chronic cardiac disease have a higher risk associated with non-cardiac surgery.

STRIKE ORAL BOARD 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.
   b. Central nervous system disease

BIBLIOGRAPHY
5. Blood Pressure Monitoring

5. Blood Pressure Monitoring

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.
5. Blood Pressure Monitoring

- Overdamped
- Normal
- Underdamped

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

6. Indications, Complications, and Waveforms

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

6. Indications, Complications, and Waveforms

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.