CHAPTER 3

Monitoring

CONTENTS

3.1 Intraoperative Monitoring Standards

Basic Monitoring Standards

During all anesthetics, the patient's oxygenation, ventilation, circulation, and temperature should be continually evaluated.

- Oxygenation: FiO_2 analyzer; pulse oximetry
- Ventilation: capnography; disconnect alarm
- Circulation: EKG, blood pressure, pulse oximetry
- Temperature: temperature probe

No monitor, however, replaces the presence of a vigilant anesthesia provider. Through the use of visualization, palpation, and auscultation the adequacy of circulation, ventilation, and temperature can be assessed. The use of technology increases your ability to do this monitoring quickly and efficiently, but none of these advances replaces YOU.

3.2 EKG

The EKG may provide the first indication of myocardial ischemia intraoperatively.

Lead placement is important in ischemia detection.

- V5 = 75% sensitive for ischemic event
- II + V5 = 80% sensitive for ischemic event
- II + V4 + V5 = 98% sensitive for ischemic event

3-lead System

Bipolar: one lead positive and the other negative. The 3-lead system can give pictures of the heart's electrical activity from 3 angles. Allows monitoring of leads I, II, and III. Only one lead can be examined at a time. Lead II is best for detecting P waves.

Augmented Limb Leads

Unipolar: right (aVR), left (aVL), and foot (aVF)

Records the electrical difference between the right and left arms and the left foot (all positive leads) utilizing a central negative lead.

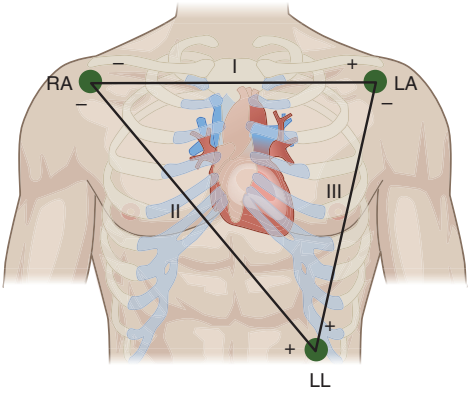


Figure 3-1 EKG Lead System

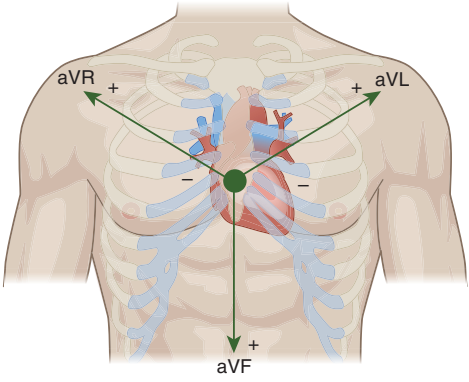


Figure 3-2 EKG Augmented Leads

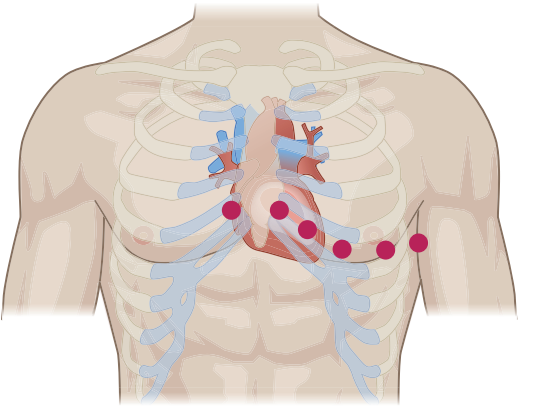


Figure 3-3 EKG Precordial Leads

Precordial Leads (V Leads)

Unipolar: with an electrically neutral center. Each individual V lead is the positive pole. They are placed anatomically over areas of the left ventricle.

Represent the heart's orientation on a transverse plane, providing a three-dimensional view.

Full 12-lead EKG

- Standard limb leads (bipolar)
- Augmented limb leads (unipolar)
- Precordial leads (unipolar)

Imagining the heart's central orientation in relation to the limb and precordial leads and picturing where the positive poles are in each lead help you to understand why the individual leads represent specific areas of the heart.

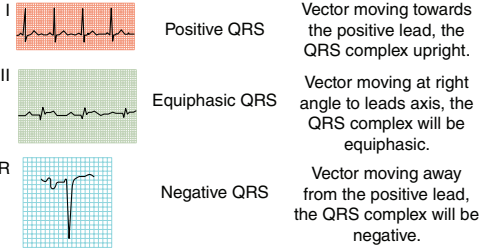


Figure 3-4 Vector and Waveform Direction

It is similar to taking pictures with a camera from different angles.

V1 and V2 represent the intraventricular septum.

V3 and V4 lie approximately over the anterior left ventricle.

V5 and V6 approximate the lateral left ventricular wall.

3.3 Cuff Blood Pressure

Cuff pressures are used often in healthy patients undergoing a short surgery.

Cons: Blood pressure readings by cuff are normally very reliable and accurate, but motion (especially shivering), certain arrhythmias (irregular heart rhythms lead to unidentical pulse waves), or the surgeon leaning against the cuff will cause false readings or failure to get a reading. Other difficulties with using a cuff for blood pressure readings are the interruption of IV infusions, issues with flow, or injury from repeated/frequent cuff inflation.

Formula for mean arterial pressure (MAP) = SBP + (2 × DBP) / 3

Cuff Size

Values may be in error if the cuff is not the proper size.

The width of the cuff should be 40% of the circumference of the extremity

- Too small—falsely high reading
- Too large—falsely low reading

Other problems from overzealous cuff use

- nerve damage
- IV infiltration

Position and Blood Pressure by Cuff

If the sitting patient's pressure is measured by a blood pressure cuff, it is crucial to know the vertical distance between the brain and the blood pressure cuff (difference is the hydrostatic pressure gradient between the heart and the brain). The blood pressure for the base of the brain should be calculated by decreasing the cuff MAP by **0.75 mmHg for every cm of vertical height** above the site of cuff measurement. This will be the MAP the base of the brain "sees." (Additionally, the pressure difference between the base of the brain [Circle of Willis] and the very top of the brain can be as much as 9 mmHg.)

3.4 Arterial Line

Used in any case where wide blood pressure swings are expected or when tight control of blood pressure or multiple arterial blood gas samples will be needed.

Indications for Arterial Line

Cardiac surgery—cardiopulmonary bypass

Major central or peripheral vascular surgery

Pulmonary resections

Intracranial operations

Major trauma procedures

Deliberate hypotension

Patient exhibiting

- pulmonary disease
- cardiac disease
- metabolic derangements
- obesity

Contraindications

Specific to site selection

- Raynaud's disease
- Allen's Test (this is controversial)
  - Allen's Test: Assess collateral circulation of hand
    - occlude both radial and ulnar arteries
    - patient squeezes fist until hand blanches
    - release ulnar artery with hand open
    - normal—color returns in < 5 seconds
    - abnormal—color returns in > 15 seconds
- Systolic Arterial Pressure (SBP): assessment of myocardial oxygen demand

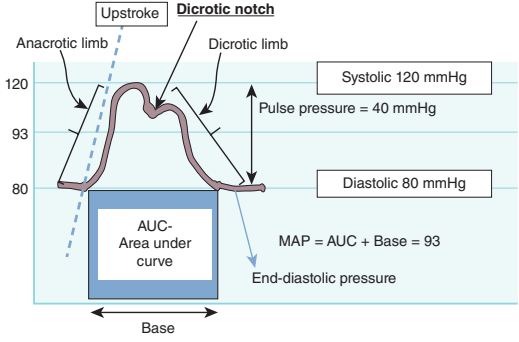


Figure 3-5 Arterial Line Waveform

- Diastolic Arterial Pressure (DBP): assessment of myocardial oxygen supply. The diastolic pressure is what fills the coronary arteries.
- Mean Arterial Pressure (MAP): the MAP perfuses the organs.

Upstroke

Steepness of ascending limb is affected by:

- Heart rate
- Systemic vascular resistance (SVR): through vasopressors (more steep incline) and vasodilators (less steep incline)
- Myocardial contractility (inotropic state): index of left ventricular inotropic state; impaired contractility can prolong the rate of the pressure increase and the angle of the upstroke.
- Anacrotic limb: occurs as ventricles eject blood into arterial tree

Downstroke

Indicates peripheral vascular resistance.

Systolic decline: area between the peak systolic pressure and the dicrotic notch; decline is more rapid when systole comes to an abrupt halt before the left ventricle (LV) is finished with ejection (i.e., LV outflow tract obstruction).

The angle of the dicrotic limb (the rate of fall from end-systole to early-diastole) changes in relation to systemic vascular resistance (SVR). Low SVR shows a steep dicrotic limb due to low pressure in the systemic arterial tree. In patients with increased SVR (aortic stenosis), the dicrotic limb angle is less acute and downstroke time is increased because of the length of time it takes to return to end-diastolic pressure.

Dicrotic limb: descending limb of arterial pressure trace as pressure falls to the diastolic pressure. The dicrotic notch is a part of the limb. Assesses SVR.

Dicrotic notch: occurs as pressure fluctuates when the aortic valve shuts during the descending arterial limb. Assesses systemic vascular resistance. Varies with which artery the line is placed.

- Signifies onset of diastole
- Flat notch: dehydration or valve insufficiency
- Low notch: high pulse pressure due to poor vascular resistance (i.e., low diastole)

Diastolic runoff: area between the dicrotic notch and the end-diastolic pressure—indicates the decline in arterial pressure as ventricular contraction comes to an end.

Area under Curve (AUC): reflects estimation of stroke volume

Pulse pressure (PP): force that the heart generates each time it contracts:

$$PP = \text{systolic} - \text{diastolic pressure}$$

Normal: 40 mmHg

Narrow or low PP: seen with low flow states (drop in left ventricular stroke volume) such as: aortic stenosis, heart failure, shock, tension pneumothorax, cardiac tamponade, pulmonary embolism

Wide or high PP: seen with high flow states such as: aortic regurgitation, warm stage of septic shock, exercise. Arteriovenous malformation, hyperthyroidism/thyroid storm.

Pulse pressure variation (PPV): variations in wave size during respiration, allows for goal-directed fluid management in mechanically ventilated patients. PPV can be measured both by arterial line or non-invasively to estimate stroke volume and cardiac output.

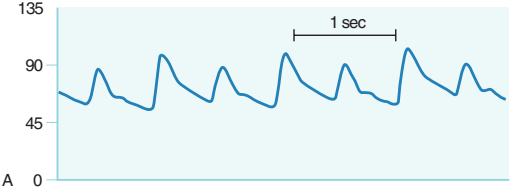


Figure 3-6 Pulse Pressure Variation

Normal: < 13

Respiratory fluctuation seen in:

- mechanical ventilation
- intravascular volume state: dehydration

Bisferiens tracing: Anacrotic notch abnormality

- Second systolic notch can be seen.
- Seen with aortic insufficiency; aortic stenosis; hypertrophic, obstructive cardiomyopathy (HOC)

Arterial Line Placement Locations

Different sites to place an arterial line:
Radial

Ulnar

Axillary

Brachial

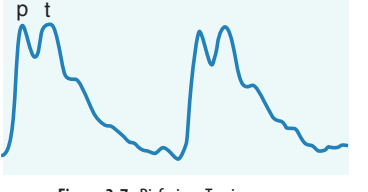


Figure 3-7 Bisferiens Tracing

Femoral

Dorsalis Pedis

Systolic Pulse Amplification

Difference in arterial waveforms according to site of insertion.

Systolic peak is steeper the further down the arterial tree it travels. When compared to the aorta, the systolic blood pressure can increase by as much as 20 mmHg at the radial artery.

From central to peripheral:

- More peaked in amplitude
- Systolic higher
- Diastolic lower
- Lower mean
- Dicrotic notch disappears

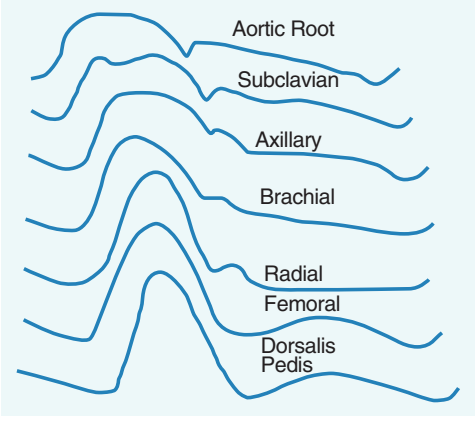


Figure 3-8 Systolic Pulse Amplification

Leveling Transducer and Calibrating Arterial Lines

(See "Leveling Transducer and Calibrating Invasive Lines" for more information)

- Supine position
  - A widely used reference point in leveling is the "***phlebostatic axis***," which corresponds to the level of the atria, is leveled at the 4th intercostal space, mid-axillary line
- Beach chair position (or any position where the head is at a different height from heart)

The horizontal reference point to level and calibrate the arterial blood pressure transducer in a patient with the head different from the level of the heart is the external auditory meatus because it best approximates the pressure delivered to the Circle of Willis in the brain.

Complications with Arterial Line

- Hemorrhage
- Air emboli
- Infection
- Impaired circulation
- Nerve dysfunction
- Thrombosis/ischemia
- Hematoma formation
- Arterial laceration

3.5 Central Venous Catheter (CVP) or CV Catheter (CVC)

Used in any case where intravascular volume status or right ventricular function needs to be monitored or evaluated.

- CVP is measured by coupling the intravascular space to a pressure transducer using fluid-filled tubing.

- CVP pressure is calibrated at the level of the vena cava or the right atrium.
- CVP measurement: patient flat, measure at **end expiration**
- The CVP normal range is 2 to 6 mm Hg

CVP Line Is Required or Useful for

- Measurement of the right heart filling pressures to assess intravascular volume (preload) and right heart function.
- Administration of medications centrally
- Anticipated inotropic medication infusion
- Massive transfusion/large amounts of volume as with surgery on heart, great vessels, mediastinal structures
- Parenteral nutrition
- Venous access—poor peripheral sites
- Aspiration of air embolus
- Insertion of pacemaker

Central Line Contraindications

- Severe coagulopathy
- Infection at site
- Right atrial tumor
- Tricuspid valve vegetation
- Anticoagulation
- Ipsilateral carotid endarterectomy
- Right atrial tumor or clots that could be dislodged
- Bundle branch blocks are a relative contraindication

Leveling Transducer and Calibrating CVP Line

Supine or sitting position

A widely used reference point in leveling for a CVP reading is the "***phlebostatic axis***," which corresponds to the level of the atria—it is leveled at the 4th intercostal space, mid-axillary line.

CVP measured when patient is flat and at end-expiration.

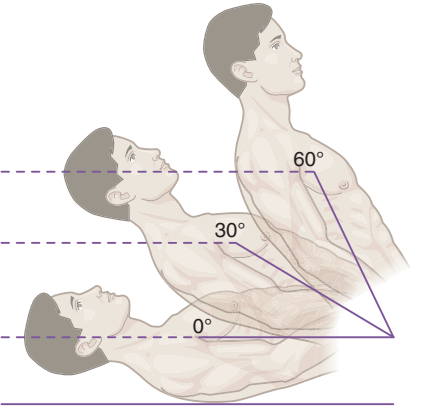


Figure 3-9 Leveling Transducer

CVP Placement

Veins and Anatomy of Upper Chest and Neck

Done in Trendelenburg position when inserting in the jugular or subclavian; this position decreases the risk of embolism and distends the vessels.

Right Internal Jugular (RIJ)

- Most common site for CVP placement. Right-sided access preferred because apical pleura does not rise as high on right and avoids thoracic duct (on left).
- Insertion
  - Patient positioned head down.
  - In low approach, triangle formed by two heads of SCM and clavicle.
  - Cannula aimed down and lateral toward ipsilateral nipple.
- Risk for nerve damage, venous air embolism, and carotid artery puncture severe enough to require surgical intervention.

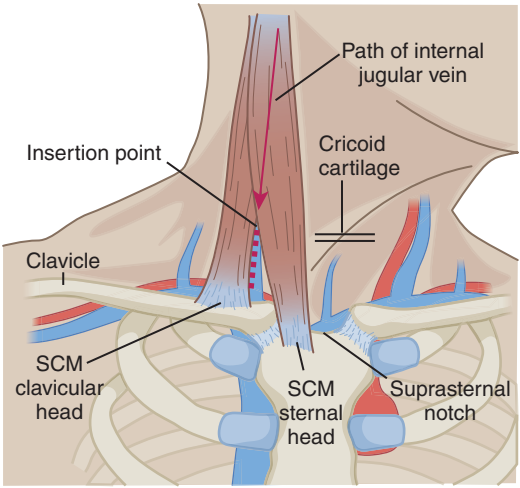


Figure 3-10 CVP Insertion Landmarks for RIJ placement

Left Internal Jugular (LIJ)

- Technically more difficult
- Increased risk of chylothorax (leakage of lymphatic fluid from the thoracic duct into the pleural space) and pleural effusion
- Potential for carotid artery puncture severe enough to require surgical intervention
- Nerve damage
- Venous air embolism

Subclavian Vein

Insertion

- Patient positioned head down
- Insertion at subclavian has a lower infection rate but high rate of pneumothorax

- Needle inserted below junction of medial 2/3 and lateral 1/3 of the clavicle; approached from below clavicle and passes immediately behind clavicle
- Needle aimed toward suprasternal notch
- Vein encountered after 4–5 cm

External Jugular

- Done in Trendelenburg position
- Valves make it difficult to thread
- Stops in axilla (arm extension may help)
- Inadvertent arterial puncture
- Kinking/migration
- Phlebitis

Femoral

- Done rarely, usually only if can't get central line anywhere else
- High incidence of line-related sepsis rate

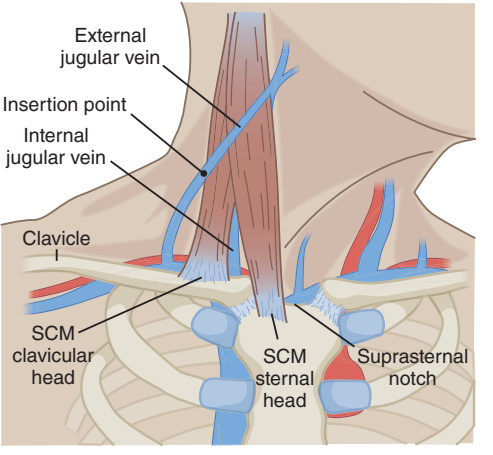


Figure 3-11 External Jugular Landmarks

Brachial

- Percutaneous; difficult to place

CVP Waveform

CVP waves are created by pressure changes in right atrium during both diastole and systole.

The CVP Waveform Interpretation

+ a wave: This wave is due to the increased atrial pressure during right atrial contraction. It correlates with the P wave on an EKG.

+ c wave: This wave is caused by a slight elevation of the tricuspid valve into the right atrium during early ventricular contraction. It correlates with the end of the QRS segment on an EKG.

– x descent: (1st downstroke) This wave is caused by the downward displacement of the ventricle and tricuspid valve during systolic contraction. It occurs before the T wave on the EKG. It signifies atrial relaxation.

+ v wave: This wave arises from the pressure produced when the blood filling the right atrium comes up against a closed tricuspid valve. It occurs as the T wave is ending on an EKG.

– y descent: (2nd downstroke) This wave is produced by the opening of the tricuspid valve in diastole with blood flowing into the right ventricle. It occurs before the P wave on an EKG.

CVP Waveform Abnormalities

- large a wave = tricuspid stenosis, right ventricular failure, pulmonary stenosis, pulmonary hypertension
- cannon "a" waves are abnormalities in the "a wave" that occur when right atrial contraction takes place against a closed tricuspid valve.

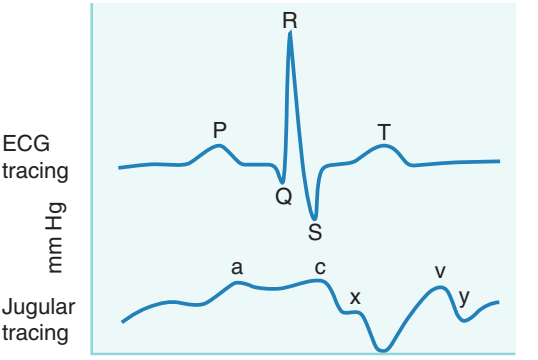
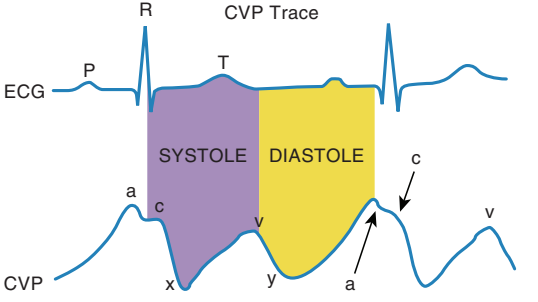


Figure 3-12 CVP Waveform and EKG



5 Components: a, c, v, x, y

Figure 3-13 CVP Waveform Components

- Classically occurs in 3rd degree heart block or AV dissociation, nodal rhythm.
- Unlike "large a waves," which are uniform in height and are observed during *each* cardiac cycle, cannon "a waves" are variable in height and occur sporadically because of the variable relationship of atrial contraction to ventricular systole.

- no a wave = atrial fibrillation
- no x wave = triscuspid regurgitation
- large v wave = tricuspid regurgitation

Complications Placing Central Lines

- Early
  - Hemorrhage
  - Air embolus
  - Pneumothorax
  - Cardiac arrhythmias
  - Pericardial tamponade
- Late
  - Venous thrombosis
  - Infection

3.6 Pulmonary Artery Catheterization (PAC) or PA Pressure (PAP)

Primarily a PAC is inserted to assess left ventricular function, but other information can also be derived regarding valvular disease, myocardial damage, and response to blood pressure interventions.

Indications: Assess cardiac output/index, check preload/volume status, SVO_2.

- Estimate of LA pressure and ultimately LV pressure
- *Estimates of* ***LAP,*** *which is an* estimate *of* ***LVEDP,*** *which is an estimate of* ***LVEDV***.
- LV preload
- LV end diastolic pressure (LVEDP)

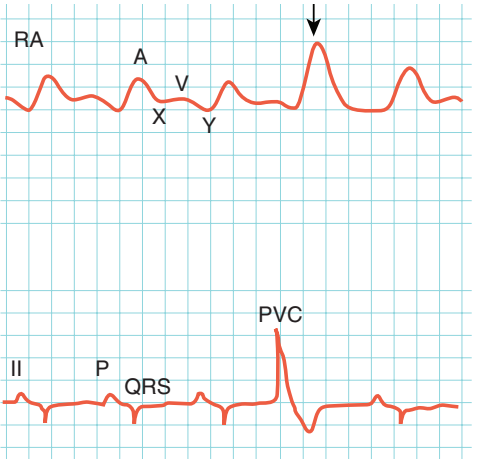


Figure 3-14 CVP Cannon V Wave

Derived Information

- Central venous pressure (CVP)—Right atrial pressure (RAP)
- Right ventricular systolic and diastolic pressures
- Right ventricular stroke work index (RVSWI)
- Pulmonary artery systolic, diastolic, and mean pressures
- Pulmonary artery wedge pressure (PCWP)
- Stroke volume (SV) and SV index (SVI)
- Vascular Resistance
  - Systemic (SVR, SVRI)
  - Pulmonary (PVR, PVI)
- Left ventricular stroke work index (LVSWI)
- Left ventricular end diastolic pressure (LVEDP)
- CO/CI
- SvO_2

Blue = proximal = CVP. . . run CO through blue port
Yellow = PA = distal
Dark blue = RV port

Contraindications to Placing PAP

Absolute

- Tricuspid or pulmonary valve stenosis
- RA or RV masses
- Tetralogy of Fallot
- Severe arrhythmias (or high-risk arrhythmias, i.e., Wolff-Parkinson–White syndrome)

Relative

- Coagulopathy
- Left bundle branch block
- High-risk arrhythmia (i.e., Wolff-Parkinson–White–WPW)
- New pacer wires

Complications of PAC Insertion

- Same as CVP plus some additional
- Dysrhythmias (frequent)
- Cardiovascular stress
- Hemorrhage
- Thromboemboli
- Infection
- Cardiac trauma/PA perforation
- Complete heart block

PA Insertion

Check balloon before inserting—must be concentric and must cover tip when inflated

Usually inserted into right or left internal jugular

Inserted through a large bore introducer (sheath)

Each black mark is 10 cm on PAC

PAC is a flow directed catheter—need heartbeat to insert through heart chambers. Must float catheter with balloon inflated from RA; inflating balloon will protect endocardium and help propel tip through RV output.

Location	Distance to Right Atrium	Inserted to Pulmonary artery	Wedge
Right Internal Jugular (RIJ)	20 cm	40–45 cm	50 cm
RIJ—Most common site	RIJ to right atrium @ 20 cm + 10 cm to RV = 30 cm + 10–15 to PA = 40–45 cm and then + 5 cm for wedge = 50 cm		
Left Internal Jugular	20–25 cm	45–50 cm	55 cm
Femoral Vein	30–40 cm	50–55 cm	60 cm
Subclavian	10–15 cm	30–35 cm	40 cm
R Antecubital	40 cm	60–65 cm	70 cm
L Antecubital	50 cm	70–75 cm	80 cm

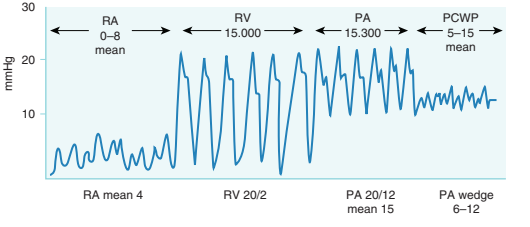


Figure 3-15 Waveform with PAC Insertion and Pressures

3.7 Mixed Venous Oxygen Saturation (SvO_2 or MvO_2)

SvO_2 is the oxygen saturation in venous blood and is also referred to as mixed venous oxygen saturation. It is the measurement of the balance between, and alterations in, O_2 consumption and delivery—represents oxygen "reserve," the amount of oxygen that can be utilized during periods of increased demand. Simply, it is a measurement of the end result of oxygen consumption and delivery at a tissue level. Measured in the pulmonary artery from a mixture of all systemic venous blood draining from all tissue capillary beds (except shunted blood).

SvO_2 = oxygen demand (SaO_2, Hgb, cardiac output) – oxygen consumed (VO_2)

Continuous SvO_2 monitoring (with an oximetric pulmonary artery catheter [PAC], which contains fiberoptic bundles that measure oxygen by reflectance spectrometry) provides a measure of oxygen remaining in the venous blood after passing through the capillary bed. SvO_2 is calculated by the differential absorption of light by saturated and desaturated Hgb (like pulse ox). Best measured from pulmonary artery as adequate mixing has occurred. Sometimes, blood can be drawn from the right ventricle but not from the right atrium as bloods are not adequately mixed that far proximal.

Normal SvO_2: sufficient oxygen supply to tissues (60–80%)

Generally, the tissues of a resting individual use approximately 25% of the available oxygen, leaving an oxygen reserve of 75% for periods of increased activity or physiologic stress.

Mixed venous oxygen saturation (SvO_2) normal range is 60–80%, meaning anywhere from 20–40% of oxygen is extracted by the tissues and 60–80% returned to the right heart.

Measurements outside normal range

Any deviation greater than 10% from patient's baseline should be considered significant if it persists for longer than 3–5 minutes.

Decreased SvO_2 (< 60%)

Either oxygen supply is insufficient or oxygen consumption is high. A drop in SvO_2 is a warning sign that oxygen demand is exceeding oxygen consumption and tissue oxygenation is compromised.

Decreased oxygen supply

- Decreased hemoglobin (bleeding, anemia)
- Arterial hypoxia (suctioning)
- Decreased cardiac output (hypovolemia, shock, cardiac dysrhythmias)

Increased oxygen demand and consumption

- Fever, shivering, thyroid storm, agitation, pain, hyperthermia, seizures, malignant hyperthermia, sepsis

Increased SvO_2 (> 80%)

Either oxygen delivery is increased or oxygen demand is decreased.

Increased oxygen supply and delivery

- Increased FiO_2

Decreased oxygen demand and consumption

- Hypothermia
- Anesthesia (muscle relaxation)

High SvO_2 but organ hypoxia—most common cause is maldistribution of blood flow; arterial blood is shunted past the capillaries and into the venous blood.

- Wedged PA catheter (blood in front of the catheter mixes with capillary blood)
- Sepsis

- Cirrhosis
- Left to right shunts (increased SvO_2 in spite of oxygen tissue deprivation)

Compensatory mechanisms kick in if oxygen delivery is decreased and/or oxygen consumption is increased to ensure adequate oxygenation to tissues. The two most important compensatory mechanisms are:

- When oxygen demand increases, the body attempts to increase delivery of oxygen primarily through an increase in cardiac output.
- If CO does not increase, the tissues will extract a larger amount of oxygen from the available supply.

3.8 Transesophageal Echocardiograph (TEE)

TEE is more accurate than transthoracic techniques.

Used to Evaluate

- Regional wall motion abnormalities indicative of myocardial ischemia
- Stroke volume/ejection fraction
- Global ventricular function
- Intracardiac air embolism
- Intravascular fluid volume

Intraoperative Diagnosis

- Early ischemia and air embolus
- Evaluation of hypotension
- Evaluation of intracardiac valve repairs
- Diagnostic for aortic disease and dissection
- Assess intracardiac mass

Limitations

- Equipment is bulky and expensive
- Requires extensive training and experience
- Qualitative versus quantitative data

Potential Risks

- Esophageal perforation
- Vocal cord paralysis
- Minor bleeding
- Atrial and ventricular arrhythmias

Contraindications

Absolute

- Esophageal pathology
  - Stricture
  - Varices
  - Scleroderma
- Esophagitis
- History of esophageal surgery

Relative

- Coagulopathy or heparinization
- Left atrial myxoma with history of embolization

3.9 Leveling Transducer and Calibrating Invasive Lines

"Leveling" the transducer assigns a zero reference point to a specific point on the patient.

- Level before line insertion or if patient, bed, or transducer moves.
  - Supine position
    A widely used reference point in leveling is the "**phlebostatic axis**," which corresponds to the level of the atria—it is leveled at the 4th intercostal space, mid-axillary line

"Zeroing" exposes the transducer to the air-fluid interface at the stopcock establishing a reference pressure. Can zero at any vertical level in the room without creating

error since atmospheric pressure is the same at floor or ceiling.

- When transducer is open to air (stopcock turned toward patient), the transducer is measuring the pressure of the atmosphere, which is 760 mmHg at sea level.
- Zeroing done before line insertion and first reading.

3.10 Pulse Oximetry

Oxygen saturation (SaO_2) is a measurement of the amount of oxygen bound to hemoglobin (Hgb). Hemoglobin consists of four iron groups (heme) attached to a protein (globin). Each heme molecule can carry one molecule of oxygen, so each molecule of hemoglobin can bind four molecules of oxygen. Oxygen saturation is the ratio of the amount of oxygenated hemoglobin to the total hemoglobin in 100 ml of blood. It is frequently expressed as a percentage.

Factors with no effect on SaO_2

- Bilirubin, fetal hemoglobin (HbF), sickle hemoglobin (HbS), acrylic nails, fluorescein dye

Errors in pulse ox monitoring (falsely low reading)

- Low perfusion states
- Optical interference: Sensor can pick up external light
- Motion
- Anemia Hgb < 5 gm/dL
- Hypothermia
- IV dyes (i.e., methylene blue)
- Electrocautery
- Nail polish (esp. dark reds and blues)
- Cardiac arrhythmias may interfere with the oximeter picking up the pulsatile signal properly

Inaccurate pulse ox reading (falsely high or low reading)

- Abnormal Hgb (i.e., carboxyhemoglobin, methemoglobin

3.11 Capnography and $ETCO_2$

The presence of carbon dioxide in exhaled gas can be measured by capnometry.

Capnometry: the measurement of end-tidal carbon dioxide

Capnography: the display of CO_2; read by infrared light absorption.

- The normal value of $ETCO_2$ is 35–37 mm Hg.
- The $PaCO_2$ is 2–5 mm Hg higher than $ETCO_2$—reflects alveolar dead space and referred to as the gradient between end-tidal and $PaCO_2$

Capnography vs. Pulse Ox:

- Pulse oximetry measures percentage of oxygen on RBCs going out from the heart; reflects changes in oxygenation within minutes.
- Capnography measures ventilation; $ETCO_2$ measures exhaled carbon dioxide; reflects changes in ventilation within seconds.

Capnography Waveform

Analyzed for: height, frequency, baseline, shape

A Inspiration ends
A–B Baseline; devoid of CO_2; dead space
B Expiration begins; rapid rise
B–C Expiratory Upstroke
C–D Expiratory Plateau
D $ETCO_2$ measured
E Inspiration begins

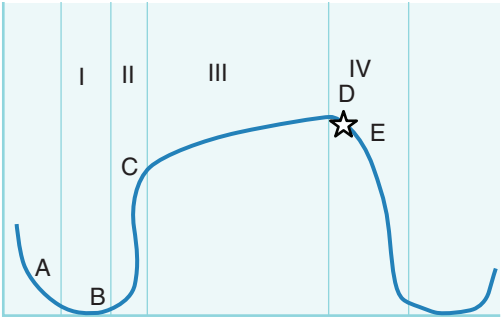


Figure 3-16 $ETCO_2$ Capnography

I steady state – inhalation ends, exhalation begins (A–B)
II rapid rise in CO_2 concentration as anatomical dead space is replaced with alveolar gas (B–C)
III alveolar equilibrium (C–D)
IV inspiration (E–A)

The Alpha Angle

- The angle between phases II and III, which increases as the slope of phase III increases.
- The alpha angle is an indirect indication of V/Q status of the lung.
- Airway obstruction causes an increased slope and a larger angle.
- Other factors that affect the angle are the response time of the capnograph, sweep speed, and the respiratory cycle time.

TABLE 3.1 Normal $ETCO_2$ 35–45 mmHg

Normal $ETCO_2$ 35–45 mmHg	Elevated $ETCO_2$	Decreased $ETCO_2$
Metabolism	Shivering, fever Hyperthermia Pain Malignant hyperthermia	Hypothermia Metabolic acidosis in shock state—*$ETCO_2$ decreases from compensatory increases in minute ventilation due to a decrease in serum bicarb. The more acidic, the lower the bicarb, the higher the respiratory rate, the lower the $ETCO_2$.*
Pulmonary	Rebreathing Decreased respiratory rate Respiratory insufficiency Chronic obstructive airway disease Analgesia/sedation	Hyperventilation Apnea/airway obstruction *Any significant decrease in lung perfusion* Bronchospasm Mucous plugging
Circulatory	Increased cardiac output	Decreased cardiac output Hypotension Hemorrhage/ hypovolemia Cardiac arrest Pulmonary embolism
Other	Tourniquet release Sodium bicarbonate given	

Figure 3-17 Capnograph Alpha Angle

Capnography Interpretation

A. $ETCO_2$ baseline above zero

Rebreathing

- Faulty expiratory valve
- Inadequate inspiratory flow
- Breath stacking (not fully exhaling)
- Exhausted/malfunction of CO_2 absorber system

B. Progressively increasing $ETCO_2$

Anything that causes CO_2 to rise:

Gradual

- Hypoventilation
- Malignant hyperthermia
- Increasing body temperature
- Increased metabolism
- Partial airway obstruction
- Excessive CO_2 production
- Absorption of CO_2 from body cavity

Sudden

- Tourniquet release
- Bicarbonate administration
- Reperfusion during vascular grafting

C. Sudden rapid fall of CO_2 close to zero

- Complete ETT disconnection from circuit
- ETT obstruction or kinked
- ETT cuff leak
- Partial disconnect

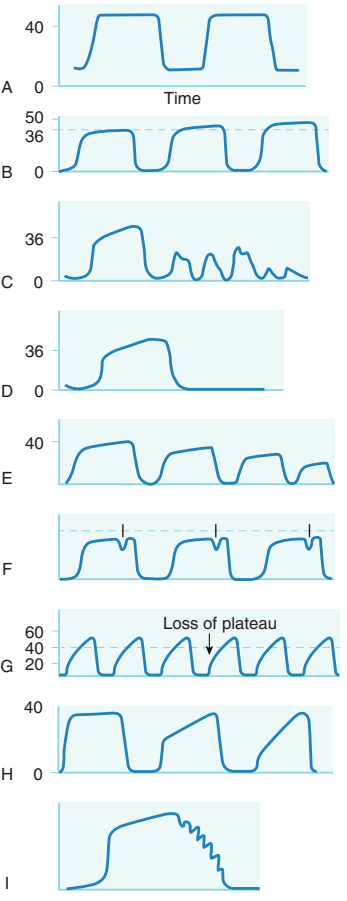


Figure 3-18 Capnography Waveforms

- Leak in circuit or sampling tube
- Room air entrainment
- ETT in hypopharynx

D. Sudden drop to zero

- Cardiac arrest
- Apnea
- Ventilator disconnection or malfunction
- Airway obstruction

E. Decrease in wave size

Exponential decrease in wave size (over 1–2 minutes)

- Circulatory arrest with continued mechanical ventilation
- Severe hypotension
- Massive blood loss
- Pulmonary embolism

Slow, gradual decrease in wave size (over 3–8 minutes)

- Hyperventilation
- Hypothermia

F. Curare Cleft

- Patient attempting to breath over controlled mechanical ventilation causes "curare cleft"
- Surgeon's hands in abdomen and pushing intermittently

***capnography is very sensitive to movements or small changes in airway pressures*

G. Shark fin shape

Continuous slanted upstroke with loss of expiratory plateau—prolonged expiratory time

- COPD
- Wheezing
- Presence of foreign body in the airway
- Obstruction in expiratory limb of vent circuit
- Partially kinked or occluded artificial airway

H. New onset slanted upstroke

- Bronchospasm

- Presence of foreign body in the airway
- Obstruction in expiratory limb of vent circuit
- Partially kinked or occluded artificial airway

I. Cardiac or jet oscillations

3.12 Neuromuscular Blockade Monitoring

- Neuromuscular blockade is monitored during surgery to guide repeated doses of muscle relaxants and to differentiate between the types of block.
- A peripheral nerve stimulator (PNS) is used to stimulate a motor nerve electrically and is a crucial monitor in assessing the patient's strength before a final decision is made to extubate the patient.
- Current output in most stimulators ranges from 0–80 mA. The intensity of the current that reaches the nerve is determined by the voltage generated and resistance of the skin and tissues underlying the electrodes.
- Number of twitches seen or felt can be quantified to number of receptors blocked.

Five Commonly Used Patterns of Stimulation

Train of Four (TOF) ratio: four successive stimuli are delivered at 2 Hz at 0.5 second intervals. At this frequency, the stores of acetylcholine are depleted and the amount released by the nerve decreases with each stimulus.

The ratio of the height of the fourth response to the first has been defined as the "train of four ratio." Twitches progressively fade as nondepolarizing neuromuscular block increases (i.e., the height of the fourth twitch is lower than that of the first twitch). The TOF stimulus provides quantification of the depth of block. At least 12 seconds should pass between TOF stimulations.

Single twitch response: a single stimulus of 0.1 Hz every 10 secs or 1.0 every 1 second at maximum current done

TABLE 3-2 TOF Response

TOF Response	Blocked Receptor	Notes
4/4	< 70%	Amenable to reversal with anticholinesterase May experience weakness even with a TOF 4/4
3/4	75%	Amenable to reversal with anticholinesterase
2/4	80%	Amenable to reversal with anticholinesterase, requires larger dosing than with TOF 3/4 or 4/4
1/4	85–90%	Do not attempt reversal of neuromuscular blockade until TOF > 2/4. Adequate relaxation for short procedures.
0/4	90–100%	95% of receptors blocked—only posttetanic facilitation visible; corresponds to acceptable muscle relaxation for intra-abdominal surgery

to stimulate fibers in a nerve bundle. The intensity of the evoked muscle response depends on how many neuromuscular junctions are unblocked.

Double burst stimulation: to evaluate the difference of the second to the first response, double burst stimulation consists of two short tetani (50 Hz) separated by an interval long enough to allow relaxation (750 msecs) in a series of 3. A minimum of 12 to 15 seconds must elapse between two consecutive double burst stimulations.

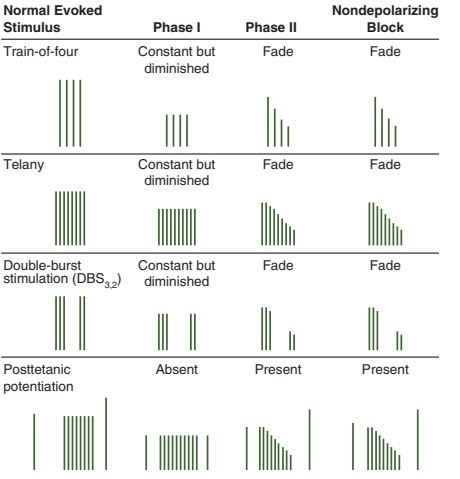


Figure 3-19 PNS Twitch

Tetanic stimulation and posttetanic count: done to evaluate the degree of neuromuscular blockade when there is no reaction to a single twitch or TOF nerve stimulation. Performed by applying tetanic stimulation at 50 Hz for five seconds followed 3 seconds later by single twitch stimulation at 1 Hz. The number of evoked posttetanic twitches detected is called the posttetanic count (PTC).

Fade is first noted at 70% receptor occupancy by neuromuscular blocking drugs and prerelaxant response is not required as the degree of paralysis can be assessed by fade. Should not be repeated more often than every 6 minutes and should not be done on an awake patient.

No twitch is seen with a very profound block but a response will be seen in the early stages of recovery. The number of posttetanic twitches correlates inversely with the time for spontaneous recovery of twitches.

Mobilization and increased synthesis of acetylcholine occur during and after cessation of tetanic stimulation. Thus, after tetanus, there is an increase in the amount of acetylcholine released in response to nerve stimulation and a single twitch evoked after cessation of tetanus may even be stronger than the pretetanic control. This is known as posttetanic facilitation.

Pattern of Twitch Return

Anesthesia providers need to know if the diaphragm and the pharyngeal/laryngeal muscles are strong enough to extubate the patient, but since we cannot test these muscles with a stimulator, we must choose a muscle group with a similar response.

Muscle Relaxants—Most Resistant to Most Sensitive

Diaphragm > muscles of larynx > corrugator supercilii muscles > abdominal muscles > orbicularis oculi > posterior tibial = adductor pollicis > masseter > upper airway muscles

Central Muscles

- Larger muscles that are relatively resistant to muscle relaxants. The dose of muscle relaxant needs to be up to 2 times greater for central muscles than for peripheral muscles to achieve the same degree of blockade.
- Includes the diaphragm, muscles of larynx, corrugator supercilii muscles, abdominal muscles, and orbicularis oculi
- Quicker onset and recovery than peripheral muscles
- If the accurate assessment of muscle relaxation onset is important, monitoring the facial nerve is more useful than a peripheral muscle.

- A central muscle, such as the corrugator supercilii, will reflect the diaphragm more closely than a peripheral muscle.

Peripheral Muscles

- Small, rapidly moving muscles that are sensitive to muscle relaxation but have a slower onset and longer action.
- Includes the adductor pollicis, posterior tibial, adductor pollicis, masseter, upper airway muscles.
- Even total elimination of twitches at the adductor pollicis does not eliminate the possibility of diaphragm movement, such as hiccoughing.
- Monitoring a peripheral muscle such as the adductor pollicis is the best nerve to monitor TOF response when assessing extubation parameters. If the response of the adductor pollicis is strong, then it is assumed that the respiratory muscles are likely to have recovered to a greater degree; thus, monitoring a peripheral muscle provides a larger margin of safety with extubation criteria.

Electrode Placement for Peripheral Nerve Stimulation

- The positive electrode, either red or white, should be placed in proximal position to the heart. The black lead is the negative lead.
- Electrode placement should be along the expected nerve path; avoid direct stimulation of the muscle.
- Nerve stimulation on a paralyzed limb will show an exaggerated evoked response because of the increased extrajunctional receptors that are resistant to muscle relaxants.

Ulnar Nerve—most commonly used for neuromuscular monitoring

Muscle: adductor pollicis ulnar nerve

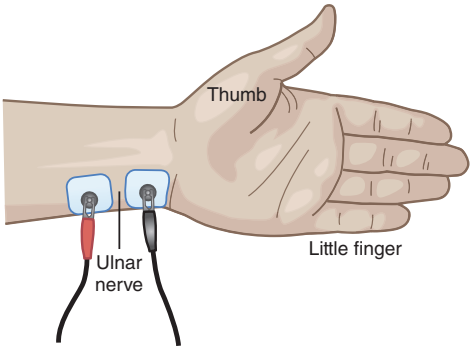


Figure 3-20 TOF Ulnar Nerve

Monitor: thumb adduction

Black (distal lead): 1–2 cm proximal to wrist flexor crease

White/Red: 2–5 cm proximal to black lead

Notes: extend the arm, palm up in a relaxed state. The 2 electrodes are placed over the path of the ulnar nerve. Fingers can twitch if the electrodes are placed directly over the muscle.

Facial nerve

Muscle: orbicularis oculi and corrugator supercilii

Monitor: orbicularis oculi (contraction of eyebrow), corrugator supercilii (pulling the eyebrow toward the nose)

Black: just anterior to tragus of ear

White/Red: lateral to outer canthus of eye or on the forehead.

Notes: the facial nerve comes from the brainstem and emerges behind the stylomastoid foramen moving forward to the lateral border of the orbit.

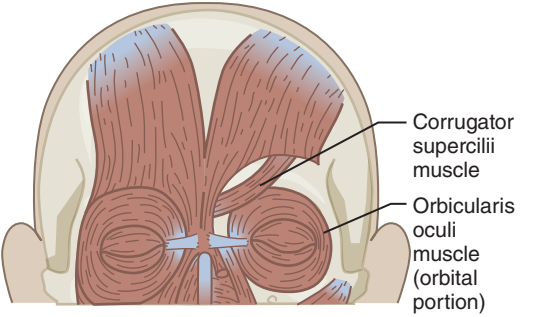


Figure 3-21 TOF Orbicularis Oculi and Corrugator Supercilii

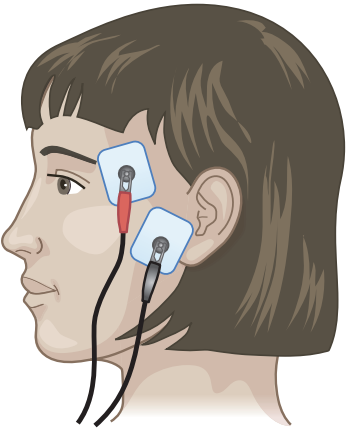


Figure 3-22 TOF Orbicularis Oculi

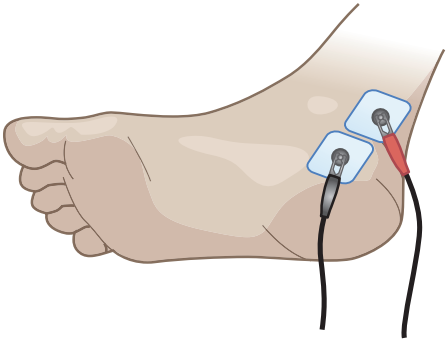


Figure 3-23 TOF Posterior Tibial Nerve

Posterior Tibial Nerve

Muscle: Flexor hallucis brevis

Monitor: Plantar flexion of great toe

Black: 2 cm posterior to medial malleolus

White/Red: 2–3 cm proximal to black

Notes: posterior tibial nerve is a *branch of sciatic nerve*.

Residual Neuromuscular Blockade

Residual neuromuscular blockade can be defined by inadequate neuromuscular recovery as measured by objective neuromuscular monitoring. This can lead to many physiological changes with impaired muscle tone and coordination:

Upper airway pharyngeal and esophageal muscles

Increased risk of aspiration

Increased risk of airway obstruction

Laryngeal muscles

Increased risk of aspiration

Impaired phonation

Impaired cough

Respiratory muscles

Impaired ventilation and oxygenation

3.13 Neurologic: EEG and BIS

Electroencephalography (**EEG**) is a monitor to record electrical activity of the brain. Usually done with electrodes placed along the scalp, although invasive electrodes are sometimes used.

- Detects areas of cerebral ischemia (carotid endarterectomy)
- Monitors isoelectricity for cerebral protection during hypothermic arrest

BIS (Bispectral Index)

BIS assesses central nervous system depression during general anesthesia to assess for awareness under anesthesia.

- BIS measures electrical activity in the brain. It is based on the surface electroencephalogram (EEG), which predictably changes in amplitude and frequency as the depth of anesthesia increases (hypnosis). It uses less electrodes and gives a global assessment of EEG, calculated over 15–30 seconds. Newer technology allows current-time waveform assessment.
- Raw EEG information is obtained via a sensor placed on the patient's forehead.
- The BIS system processes the EEG information and calculates a number between 0 and 100 that provides a direct measure of the patient's level of consciousness.

- 70–100: moderate sedation to full awareness with intact memory
- 60–70: light hypnosis. Patient is more likely to have awareness.
- 40–60: ideal anesthetic state
- 0–40: deep hypnosis
- 0: indicates the absence of brain activity (cortical silence)

Research has shown that BIS-monitored patients wake up faster, are extubated sooner, and are more oriented upon arrival to the PACU.

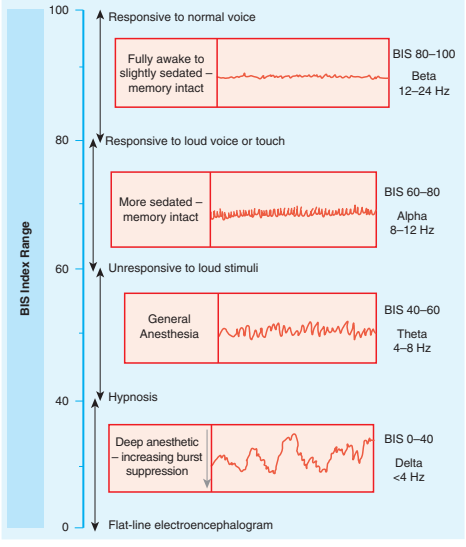


Figure 3-24 BIS Waveforms

BIS readings are affected by:
- Electrocautery
- Pacer spikes
- EKG
- Patient movement

Monitor Measurements along with BIS
- **SR**—suppression ratio: the amount of the EEG that is in burst suppression.
- **SQI**—quality index: how accurate the BIS number is.

BIS waveforms:
- **beta**—tiny flat; low amplitude—very awake
- **alpha**—quiet awake
- **theta**—first stages of sleep
- **delta**—wider loopy—unconsciousness

3.14 Neurophysiology Monitoring

Neurophysiological monitoring is valuable in the identification of any pathologic condition that may alter neurologic function or in the prevention of neurological injury during surgical manipulations; it is also used to guide the surgeon in localizing anatomical structures during dissection. The goal of this monitoring is to identify changes in the brain, spinal cord, or peripheral nerve function prior to irreversible damage.

Evoked Potentials (EP): the electrophysiologic responses of the central nervous system in response to external sensory or motor stimulation of specific nerve pathways—done to monitor the functional integrity of "at risk" neural structures during surgery.

All EPs are described in terms of amplitude, shape, and latencies of the responses that are monitored:
- Latency describes the time between the stimulus and the generated potential.
- Amplitude is a measure of the peak of the waveform from baseline.

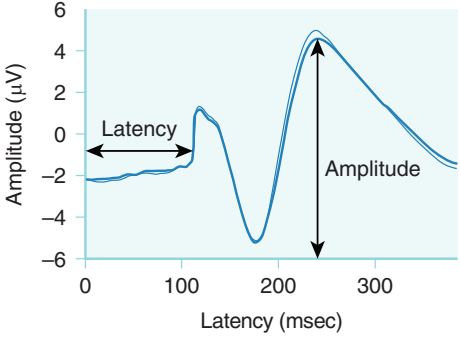


Figure 3-25 Latency and Amplitude

When used in intraoperative monitoring, changes in latency and amplitude from baseline responses are the most important indicators of neurological dysfunction.
Alarm criteria

- 50% decrease in amplitude (amplitude changes have been implicated as the most sensitive measure of cerebral ischemia)
- 10% increase in latency

I Sensory Evoked Potentials

1. **Somatosensory evoked potentials (SSEP):** done to assess the functional integrity of afferent nerve pathways; the speed of a stimulus in the ascending dorsal (posterior) and lateral sensory pathways of spinal cord is measured. SSEP is unable to test the motor pathway, a significant limitation of this test.

   Technique: SSEP is produced by stimulation of a peripheral nerve wherein a response in the cortex, spinal cord, or nerve can be measured. Electrical stimulus ascends the spinal cord through the dorsal roots, ascends to the third-order fibers to the

thalamus, then on to the contralateral sensory cortex where cortical response is measured.

Change due to surgical maneuvers (e.g., spinal distraction) or ischemia (e.g., after placement of an artery clamp) can be abrupt and localized and may affect only one side of the body.

Common sites of stimulation:

- Upper extremity—the median nerve at the wrist (most common), can also be done at ulnar or radial nerve.
- Lower extremity—the posterior tibial nerve at the ankle or peroneal nerve at the knee.

Clinical Uses: surgery to the major peripheral nerves, spinal surgery: with nerve roots, discectomies, laminectomies, corpectomies, anterior and posterior fusions, and pedicle screw procedures; posterior fossa; supratentorial intracranial; *cranial/vascular surgery,* including endarterectomy, and anterior and posterior cerebral aneurysms; thoracoabdominal aortic aneurysm, cardiac bypass procedures, and aortic cross-clamping

Motor Homunculus: The choice of electrode placement for recording SSEP from the scalp will be dictated by the site of stimulation. For example, note that the lower extremities are located more medially, while the hand and face are located more laterally in the motor homunculus. The SSEP recording electrode is placed midline to record tibial nerve evoked potentials (note "leg" in homunculus is midline).

2. **Brainstem auditory evoked potentials (BAEP):** other terms for this technique include brainstem auditory evoked responses (BAER), auditory evoked potentials (AEP), and auditory brainstem responses (ABR).

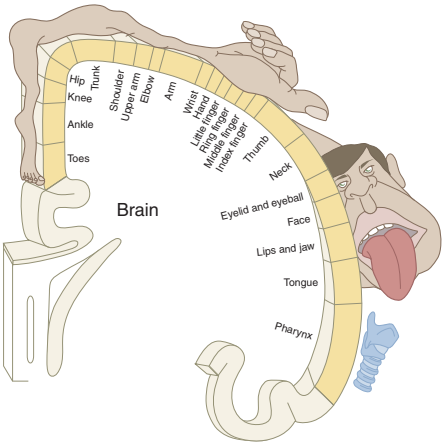


Figure 3-26 Motor Homunculus

Technique: BAEP measures brainstem activity for cranial nerve VIII. Auditory evoked potential can be used to trace the signal generated by a sound (auditory clicks in the ear) through the ascending auditory pathway. The evoked potential is generated in the cochlea and goes through the cochlear nerve, to the midbrain, and finally to the cortex where recordings are obtained by standard EEG scalp electrodes (best results obtained near the ears). This recording provides an index of disturbance secondary to ischemia of the cochlear nerve and the divisions of the brainstem.

Clinical uses: Good for posterior fossa or where brainstem is at risk, vestibular nerve, cerebellopontine angle surgery, acoustic neuroma, posterior

fossa, brainstem lesions, or for microvascular decompression for tic douloureux or hemifacial spasm.

3. **Visual evoked potentials (VEP)**: monitoring of visual pathways has potential utility in surgery performed in proximity to the visual apparatus.

   Technique: visual stimulation is done by flashing light-emitting diodes (LED) or strobe lights. *Light entering the eye is converted into electrical energy at the retina and travels through the optic nerve to the visual cortex of the brain. VEP measures the strength of the signal reaching the visual cortex and how fast it gets there.* Potentials are recorded with electrodes over occipital cortex.

   Clinical uses: useful for surgery near optic pathways, especially in the parasellar region as tumors that arise in this area carry significant risk of visual impairment—they include craniopharyngiomas, pituitary adenomas, and suprasellar meningiomas.

4. **Dermatomal Evoked Potentials (DEPs)**: stimulation of a single dermatome to depolarize a single nerve root to ascertain ascending conduction of a signal via the dorsal column pathways to the primary somatosensory cortex. Much like SSEP, though SSEPs do not provide information on the integrity of individual nerve roots.

   Anatomical pathways and alarm criteria with regards to amplitude or latency are the same as with SSEPs.

   Technique: electrodes are placed several centimeters apart from one another in a single dermatome. Problems can arise because overlap between dermatomes exists.

   Clinical uses: spina bifida/tethered cord releases, lesions of specific nerve roots, brachial plexus surgery.

II Motor Evoked Potentials (MEP)

MEPs monitor efferent corticospinal (motor) tracts of the ventral (anterior) spinal cord. Tongue biting is the most common complication, and placing bite blocks must be done to prevent serious damage.

Technique: electrical stimulation is performed transcranially over the motor cortex. The stimulus is propagated via motor pathways that descend to the anterior spinal cord via the corticospinal tract. It then travels via a peripheral nerve to produce a muscle response.

Significant motor deficits have been seen in patients undergoing spinal surgery despite normal SSEPs. MEPs were developed to better monitor the motor neurophysiological pathways, and their use in combination with SSEP appears to improve the accuracy of monitoring spinal cord function.

Clinical Uses: MEPs are used in procedures with anterior approaches to spinal cord, abdominal aortic aneurysms, scoliosis, spinal cord tumors, decompression and fusion spinal vertebrae.

The use of MEP is contraindicated in patients with epilepsy, increased intracranial pressure, or cochlear implants.

Anesthesia and MEP: muscle relaxants can depress myoneural transmission; check with surgeon before giving.

III Spontaneous Potentials

1. **Electromyography (EMG):** EMG is monitored for spontaneous (can also be induced) electrical activity. Practically any muscle can be monitored, including face, tongue, and sphincter musculature.

   Technique: multiple recording EMG needles are placed directly into the muscles of interest. Alarm criteria in monitoring is the "presence of EMG";

normal is the absence of spontaneous muscle activity.

Clinical Uses: facial nerve/other cranial nerve monitoring, spinal exploration and fusions; dorsal rhizotomy, tethered spinal cord release, pedicle screw placement

Anesthesia with EMG: muscle relaxation cannot be used.

2. **Electroencephalogram (EEG):** electrodes placed along the scalp to read spontaneous electrical activity of the cortex. Most commonly used during carotid endarterectomy but also during cerebral aneurysm repair and during hypothermic circulatory arrest. The most important requirement for intraoperative EEG recording is knowledge of expected changes with deepening levels of anesthesia. Any changes that cannot be explained by anesthetic technique may reflect changes in systemic blood pressure and/or cerebral blood flow.
3. **Electrocorticography (ECoG):** electrodes are placed directly on the exposed brain surface to read electrical activity of the cortex; it is used to help determine resection margins for epilepsy surgery and to monitor for seizures during electrical stimulation of the brain.

Intraoperative EP Changes May Result from

- Surgical injury or ischemia
- Nonspecific physiologic factors
  - Hypothermia/hyperthermia
  - Hypotension
  - $PaCO_2$ due to changes in spinal cord and cerebral blood flow
  - PaO_2 due to ischemia of cerebral tissues
  - Electrical interference
  - Hypoxia
  - Hemodilution/decreased hematocrit

Effects of Anesthetics on Evoked Potentials and EEG

Evoked potentials are affected by almost all anesthetic agents, some quite significantly. Anesthetics exert their effects on the brain by depressing cerebral metabolism.

Electrophysiologist will instruct what agents may or may not be used and their dose limits.

- **Volatile anesthetics**
  - The volatile agents, which include the halogenated anesthetics and nitrous oxide, have the most potentially deleterious effect of all anesthetics on EPs. All cause similar depression of amplitude and increase in latencies. MAC of volatile (including nitrous oxide) should be minimized to 0.5 or less; however, successful monitoring has been done up to 1.0.
  - They affect cortically evoked responses more than subcortical, spinal, or peripherally evoked responses. At high concentrations, most also can suppress epileptiform discharges.
- **Propofol**
  - Increases latency and decreases amplitude of cortical evoked potentials.
- **Etomidate**
  - In low doses, etomidate can increase evoked potential amplitude but prolong latencies. At induction doses, amplitude may be reduced.
- **Ketamine**
  - Ketamine either does not affect or may increase evoked potential amplitude. Do not use in patients with intracranial pathology.
- **Narcotics**
  - Narcotics cause mild reduction in amplitude of evoked potentials but usually allow consistent monitoring.

- **Dexmedetomidine and clonidine**
  - Have minimal effect on cortical EPs and are considered safe to use during EP monitoring.
- **Benzodiazepines**
  - Benzodiazepines usually result in decreased amplitude with little effect on latencies.
- **Neuromuscular blockers**
  - These agents have no significant effect on evoked potentials. Muscle relaxation reduces artifactual signals from spontaneous muscle activity and, if complete, suppresses evoked muscular responses as well.
- **Barbiturates**
  - These may decrease evoked potential amplitude and increase latency, but typically recordings can be obtained despite high doses. They also increase beta frequency activity. Fast-acting barbiturates (e.g., methohexital) sometimes can increase epileptiform spikes.

Sensitivity to Anesthesia—Least to Most Sensitive

BAEP–EEG–SSEP–VEP

3.15 Temperature Monitoring

Basic monitoring standards dictate that during all anesthetics, among other monitoring devices, the temperature should be continually evaluated.

During Anesthesia

- Infants and small children are prone to thermal lability due to their high surface area to volume ratio.
- Adults subjected to large evaporative losses or low ambient temperatures (as occur with exposed body

cavity, large volume transfusion of unwarmed fluids, or burns) are prone to hypothermia.
- Malignant hyperthermia is always a possible complication (when volatile anesthetics or succinylcholine are used), and temperature monitoring is one step in its diagnosis.
- Anesthesia inhibits thermoregulation. Causes redistribution hypothermia; heat loss > heat production.
- Heat transfer to cold operating room (most to least) radiation > convection > evaporation > conduction.

Benefits of Hypothermia
- Tissue metabolic rate decreases ~8%/1°C decrease in temperature.
- Protects heart due to decreased myocardial oxygen and metabolic demand.
- CNS protection; improved neurologic outcomes after cardiac arrest.

Consequences of Hypothermia
- Coagulopathy (esp. platelets), increased blood loss
- Increased infection rate; delayed wound healing; jeopardizes graft success
- Increased drug duration of action and delayed emergence
- Left shift of the oxyhemoglobin dissociation curve (tissue hypoxia)
- Increased postoperative shivering and increased O_2 consumption (400% increase)

Prevent Hypothermia
- Warmed air (Bair hugger)
- Radiant heat lamps
- Fluid warmer
- Circuit heat and humidification moisture exchanger
- Warm OR temperature

Blood Temperature Measurements May Be Obtained with

- Thermistor of a pulmonary artery catheter (gold standard)—"core" temperature
- Esophagus: correlates with core temp; probe should be located at the lower third of the esophagus
- Nasopharynx: correlates with core temperature
- Tympanic membrane: correlates with core temp but not practical in the operating room environment
- Oropharynx: good estimate of core temperature
- Bladder: correlates with core if urinary output is high
- Rectum: does not correlate with core temp; affected by extremity venous return and stool
- Axillary and skin: inaccurate