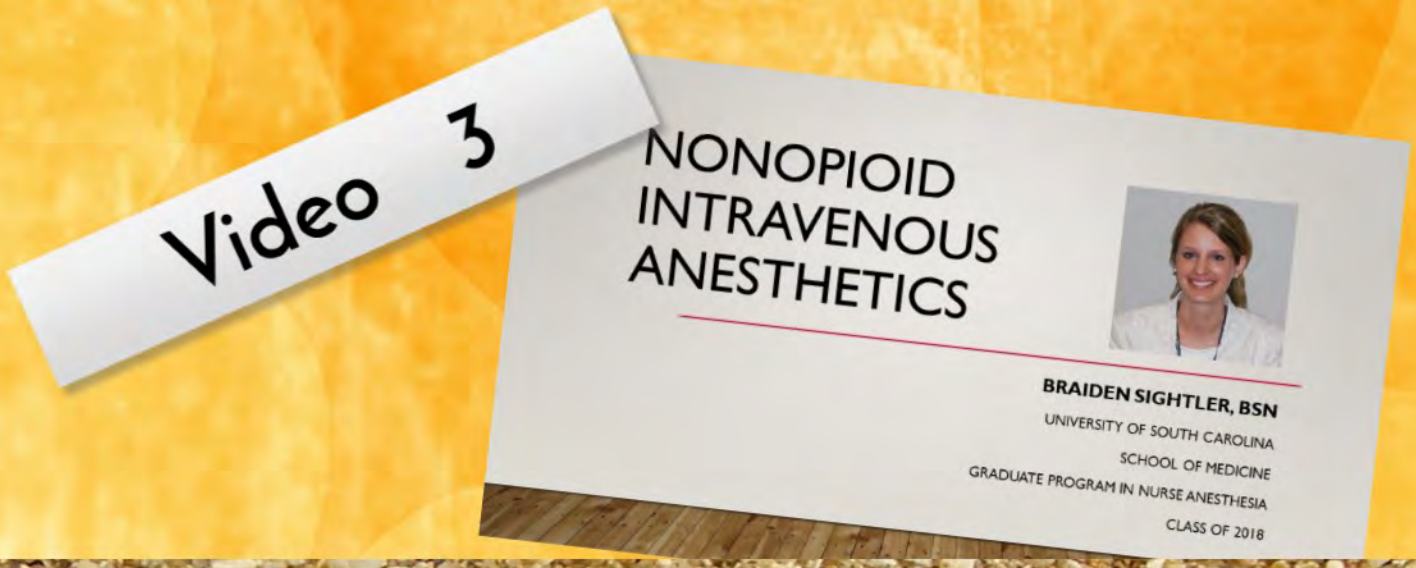
Anesthesia for Post Anesthesia Care Nurses



Coleman, Harman, Koke, Sightler, Squires, Storm

STORM ANESTHESIA

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Introduction

Welcome to Storm Anesthesia's Anesthesia for Post Anesthesia Care nurses. This is a seven-video series on the basics on pharmacology and anesthesia techniques for the perianesthesia care nurse.

We, a group of five senior students from the University of South Carolina School of Medicine Nurse Anesthesia Program and one CRNA, have created this series in the hopes it will help the transition into the perianesthesia world. The series attempts to shine a bit of light on the techniques anesthesia uses during surgery, as well as explain the basics of the pharmacology behind our drug uses. This is by no means a series that will explain everything that happens during anesthesia, but our hope is that you, the perianesthesia nurse, will find our report a little less intimidating and a little more informative. After all, the better you understand the report, the better you can take care of the patient, and ultimately, this will increase the safety and satisfaction for both your patients and yourself.

The group consists of Alexandra Harman, BSN, RN; Braiden Sightler, BSN, RN; Jordan Coleman, BSN, RN, CCRN; Kelsey Squires, BSN, RN, CCRN; Victoria Koke, BSN, RN; and Michael Storm, DNAP, CRNA, CCRN.

The videos can be watched separately, but there are some references among the videos and the basics of the pharmacology along the way. Therefore, it may be beneficial to watch the series in order. Either way, have fun and don't forget to download the accompanying handouts. These handouts are the complete transcripts of the narrations and include all relevant pictures from the videos.

This video-series is sponsored by Storm Anesthesia and Palmetto Health Richland Anesthesia Department.

Enjoy and let's get started.

Michael Storm, DNAP, CRNA, CCRN
Editor
November 2017

Nonopioid Intravenous Anesthetics

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Class of 2018

Hi, I'm Braiden Sightler, a registered nurse and current senior student in the University of South Carolina Nurse Anesthesia program. Today, we are going to discuss Nonopioid Intravenous Anesthetics.

Objectives

Our objectives for this presentation include: review keywords and definitions, understand GABA and its role in several of the IV anesthetic drugs mechanism of action, discuss clinical uses and key features for each of the IV anesthetics, and review common side effects and contraindications for each anesthetic.

Keywords and Definitions

Let us review some common keywords and definitions crucial to understanding of these drugs.

Analgesia: the inability to feel pain

Amnesia: partial or total loss of memory

Anterograde amnesia: the inability to recall events that occur after the onset of amnesia

Dissociative anesthesia: analgesia and amnesia, but with no loss of respiratory drive

Sedatives: substances that have a calming effect

Anxiolytic: substances that reduce anxiety

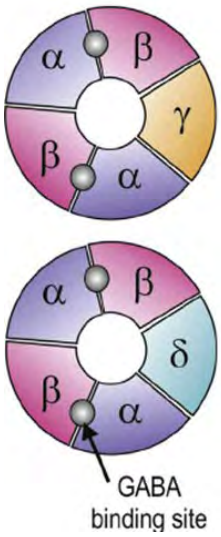
Hypnotic: sleep inducing

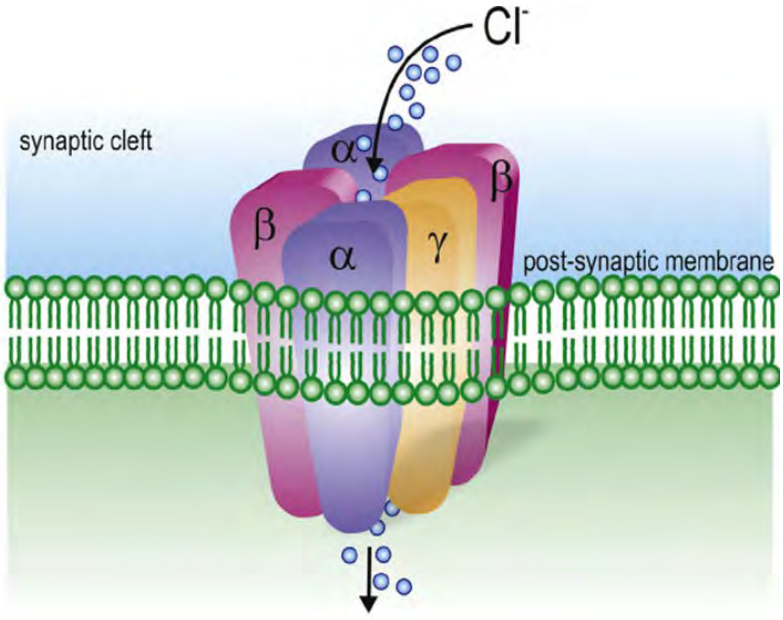
Key Concept

A key concept with several of the IV anesthetic agents is understanding GABA or GAMMA AMINOBUTYRIC ACID and its receptor which we will discuss in the following slide. GABA is an amino acid that functions as an inhibitory neurotransmitter in the brain and spinal cord. The role of GABA is to inhibit or reduce the activity of neurons. GABA helps to control fear and anxiety when neurons become overexcited.

$GABA_A$ Receptor

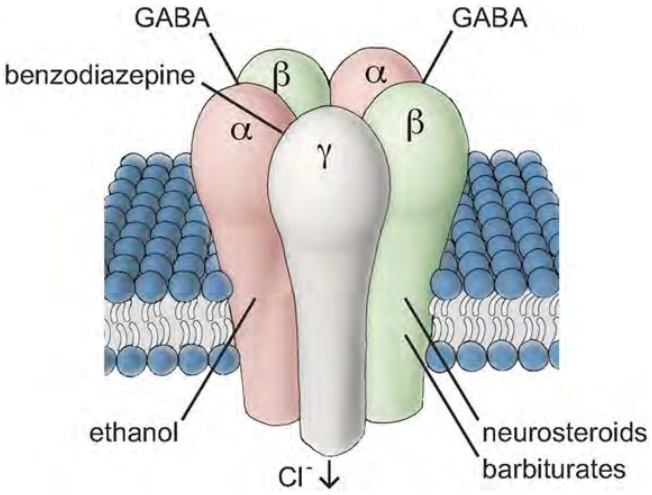
Now let us talk about where GABA binds…. In the brain, there are specific binding sites for GABA and the main one we will discuss is the $GABA_A$ receptor... GABA has a specific binding site on this receptor as you can see in the picture. This receptor has a pentameric structure and each receptor contains two alpha subunits, two beta subunits, and one gamma subunit. It has a structural and functional similarity with ligand gated ion channels - meaning they open to allow ions such as Na^+, K^+, Ca^{2+}, and/or Cl^- (which in our case is the most important) to pass through the membrane in response to the binding of a chemical messenger, in our case GABA.





So How Does It Work?

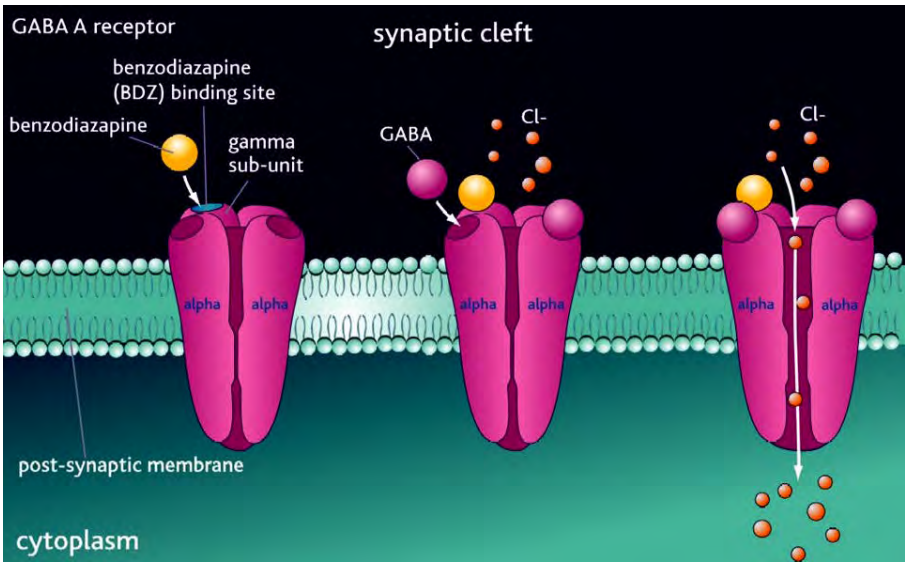
GABA binds at its specific binding site on the $GABA_A$ receptor. This opens the chloride channel and causes hyperpolarization of the neuron, which causes an inhibitory effect on neurotransmission by diminishing the chance of a successful action potential occurring, thus, depression of the central nervous system.



Several of the nonopioid IV anesthetic drugs we will discuss, have some interaction with GABA. As you can see in the illustration, barbiturates, benzodiazepines, and even ethanol all work at the $GABA_A$ receptor through specific binding sites.

Benzodiazepines

The first class of drugs we will discuss are the benzodiazepines, which is a fairly large class of drugs. Overall, these drugs potentiate GABA, therefore, they increase the effect of the GABA receptor. Now, GABA neurotransmitter binds at its two sites on the $GABA_A$ receptor and the benzodiazepine binds on its own site on the GABA receptor as well. This combination of binding, the usual GABA and the additional benzodiazepine, causes an increase in the frequency of chloride channel opening and, thereby, an increase in the GABA created CNS effect. The CNS effects that result are: sedation, hypnosis, and anxiolysis. Moderate muscle relaxation also occurs.



It is important to note that opiates enhance the hypnotic action of benzos.

Summary of Clinical Uses for Benzodiazepines

As you can see from this chart, there are countless uses for benzodiazepines. They are used for anxiety disorders, insomnia, convulsive or spastic disorders, involuntary movement disorders, and before surgery as an amnestic.

Anxiety disorders
- Acute anxiety
- Generalized anxiety disorder
- Panic disorder
- Phobias (social, simple)
- Post-traumatic stress disorder
- Obsessive-compulsive disorder

Insomnia

Anxiety associated with medical illness

Convulsive disorders
- Acute *status epilepticus*
- Neonatal seizures or febrile convulsions
- Preeclampsia
- Tetanus
- Adjunct to other anticonvulsants

Spastic disorders; acute muscle spasm
- Cerebral palsy
- Multiple sclerosis
- Paraplegia secondary to spinal trauma

Amnestic (before surgery or procedure)

Involuntary movement disorders
- Restless leg syndrome
- Akathisia associated with neuroleptic use
- Choreiform disorders
- Myoclonus

Detoxification from alcohol and other substances

Agitation/ anxiety due to psychiatric conditions
- Acute mania
- Psychotic illness
- Anxiety associated with depression
- Impulse control disorders
- Catatonia or mutism

Other adjunctive uses
- Surgery
- Dentistry
- Diagnostic studies, such as computed tomography, magnetic resonance imaging and endoscopy
- Cardioversion
- Chemotherapy

Biotransformation and Excretion

The benzodiazepines are metabolized by cytochrome P450 enzymes in the liver. They are excreted via the urine. Some metabolites retain activity and may accumulate with chronic dosing.

In patients with liver disease, they can cause enhanced sedation due to slower elimination via cytochrome P450 enzymes, therefore caution needs to be taken in these patients.

Patient Considerations to Clinical Use of Benzodiazepines

As with all drugs, we must examine the patient and their health history to safely administer these medications. Caution needs to be taken in:

- Older age (greater than 65)
  - can cause mental confusion, amnesia, falls and fractures, partly due to accumulation of long-acting, active metabolites
  - this explains why some geriatric patients aren't given benzodiazepines before surgery.
- Chronic respiratory disease (and sleep apnea)
  - may cause respiratory depression
- Liver disease
  - may cause enhanced sedation due to slower elimination
- Pregnancy category D
  - can cause neonatal withdrawal

Midazolam

Uses: premedication for surgery, cardioversion, endoscopic procedures and sedation

Provides: profound anterograde amnesia, which explains why most patients pre-medicated before surgery don't remember going back to the operating room. Also, provides some anticonvulsant properties.

- Usual dose: 1-4 mg IV
- Rapid onset of action: 1-5 minutes
- Peak action: 2-5 minutes
- Duration of action: 15-90 minutes

PACU Concerns

There are specific PACU concerns for this drug, particularly if a patient is given additional midazolam in the PACU. Respiratory depression can occur due to midazolam causing a dose-dependent respiratory depression and also potentiation with other drugs from surgery. Oxygen and resuscitative equipment must be available and the patient age should be considered with elderly patients having a reduction in dosage by 25-30%.

Diazepam

The next benzodiazepine we will discuss is diazepam. It has several uses: antianxiety, sedation, CNS-mediated muscle relaxation, alcohol withdrawal, and status epilepticus.

Anterograde amnesia has been noted as long as 48 hours after surgery. When combined with an opiate, respiratory depression is greatly increased. Lastly, it has strong anticonvulsant activity and can stop generalized seizure activity.

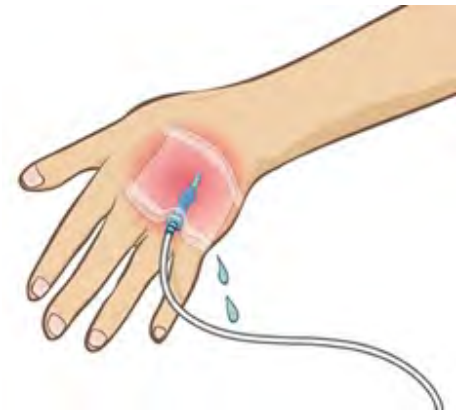
Dosing

As far as dosing:

- Sedation: 2-10 mg IV
- Status Epilepticus: 5-10 mg IV/IM every 5-10 minutes not to exceed 30 mg

It has a rapid onset of action at 1-5 minutes with a peak action of 15-30 minutes. The duration of action is around an hour.

Administration concern



There is an administration concern with diazepam when given intravenously, thrombophlebitis can occur therefore, it should be given:

- in a large vein
- slowly
- not mixed with any other drugs
- not diluted

Adverse reactions to diazepam

Adverse reactions to diazepam include:

- hiccups
- nausea
- phlebitis at the site of injection
- occasional acute hyper-excited states

Lorazepam

The last benzodiazepine we will discuss is lorazepam. The drug promotes anxiolysis and amnesia and is the drug of choice for status epilepticus. It has a long duration of action, but no active metabolite, therefore there is no concern in patients with liver disease. However, it does have a high abuse potential.

Dosing

As far as dosing:

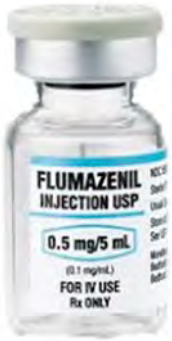
- 1-2 mg IV over 2 minutes
- IV infusions: 1-5 mg/hr
- Status epilepticus: 4 mg/dose slow IV at 2 mg/min. If seizure persists after 5-10 min, administer 4 mg IV again

The onset of action is between 1-5 minutes with a peak action time between 20-40 minutes and duration of 4-6 hours.

Benzodiazepine Antagonist - Flumazenil

To reverse benzodiazepines, flumazenil competitively antagonizes activity at the benzodiazepine recognition site on the $GABA_A$ receptor and reverses the CNS effects of benzodiazepines. However, it is not as effective in the treatment of hypoventilation or respiratory failure since benzodiazepines are very unlikely to cause respiratory depression. The most frequent effect is slightly reduced respiratory rate, but the RN must consider other drugs or if the patient has had any opioids and consider reversal due to potentiation.

Dosing



- Usual reversal dose for flumazenil is 0.4 mg administered IV in 0.1 mg increments
- Should be administered slowly to avoid the consequences of abrupt withdrawal
- Maximum dose is 1 mg at one time and no more than 3 mg within a 1-hour period

Onset of action is within 5 minutes and the duration of action is 1-2 hours.

Considerations

Considerations for flumazenil include: it does not reverse effects of barbiturates, opiates, or ethanol. Great caution should be taken in patients with epilepsy as reversal can result in seizures.

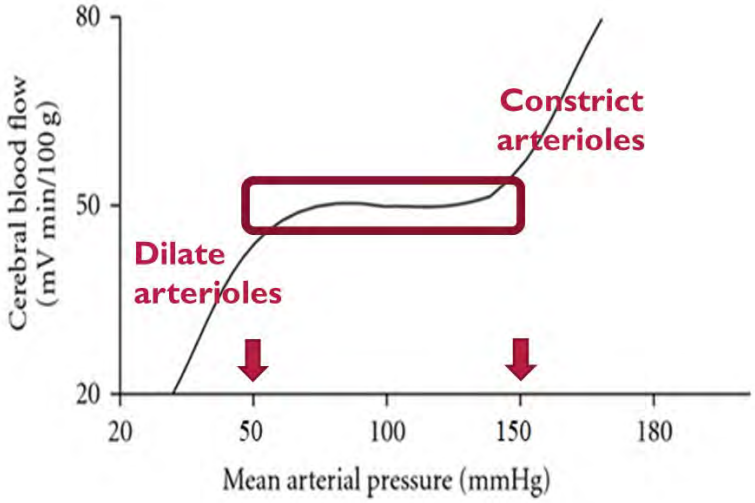
Incidence rate of post-operative nausea and vomiting is increased after administration. And lastly, this drug has a shorter duration of action than most of the benzodiazepines, meaning re-sedation can occur and subsequent doses may be needed.

Thiopental

Because of its historical significance, we will briefly discuss thiopental, an ultra-short acting barbiturate now rarely used and not readily available in the US due to its controversy in capital punishment. This drug also works by regulating $GABA_A$ specifically increasing the opening time allowing more influx of chloride and nerve hyperpolarization. It was previously used for induction of anesthesia and control of elevated intraocular pressure with an onset of action in 15-30 seconds and a duration of action of 5-10 minutes. The usual dose is 3-5 mg/kg.

Neuroprotection of Thiopental

Thiopental is often associated with neuroprotection, utilizing a concept called autoregulation. Cerebral blood flow (CBF) remains constant in the face of changing mean arterial pressures (MAP). Between mean arterial pressures of 50-150 mmHg autoregulation keeps CBF relatively constant. Above and below a MAP of 50-150 mmHg, however, cerebral



arterioles must dilate or constrict (autoregulate) to maintain constant flow.

Thiopental is considered neuroprotective, since it causes a decrease in cerebral metabolic rate (CMR). This decrease in turn causes a decreased need for blood flow to maintain metabolism, which again will result in a decrease in intracranial pressure (less blood volume to create pressure).

This dynamic of decreased CMR, CBF, and ICP is critical in surgical patients with space occupying lesions or trauma since they have decreased intracranial compliance (i.e., ability to compensate from surges in volume and pressure) and, thereby, may be extremely sensitive to ICP changes.

Other Considerations for Thiopental

For other considerations, side effects include: bradycardia, hypotension, and respiratory depression. Severe tissue injury with extravasation can occur and at low concentrations anti-analgesic effects can occur.

Methohexital

Methohexital is an ultra-short acting barbiturate, three times more potent than thiopental that is currently used in the US for electroconvulsive therapy and identification of seizure foci during ablative surgery

Its mechanism of action is regulation of $GABA_A$, increasing opening time. Recovery time from anesthesia is extremely rapid at 4-7 minutes with an onset in 30 seconds. The usual dose is 1-2.5 mg/kg.

Propofol

Continuing with drugs that potentiate the $GABA_A$ receptor, we will now discuss propofol. Propofol is one of the most popular IV anesthetics and has varied uses including induction of general anesthesia, IV sedation for minor surgeries and regional anesthesia, and sedation in the ICU especially with patients on the ventilator. Its mechanism of action is at the $GABA_A$ receptor as an agonist, increasing Cl^- conductance and causing hyperpolarization.
The formulation is interesting, with it being a 1% solution in an emulsion of egg lecithin, soybean oil, and glycerol.

Propofol Dosing

- Induction of general anesthesia: 2-2.5 mg/kg IV
- Infusion: 25-200 mcg/kg/min

It has a rapid onset of action in 30-60 seconds and a duration of action of 5-10 minutes.

Propofol Effects

HR	MAP	Contractility	CBF	ICP	IOP	MV	Ventilatory Drive
↔ or ↓	↓↓↓	↓	↓↓↓	↓↓↓	↓↓↓	↓↓	↓↓↓

Propofol's effects on the body can best be explained in this graph. As far as heart rate, there is no change or a small decrease if seen. Mean arterial pressure is significantly decreased. There is a small decrease in contractility. Cerebral blood flow, intracranial pressure, and intraocular pressure are all significantly decreased. Minute ventilation is decreased and ventilatory drive is significantly decreased.

Propofol Considerations

Considerations for propofol include reducing the dose for elderly, critically ill, and hypovolemic patients. The unique lipid formulation supports bacterial growth linked to sepsis, therefore you should observe aseptic technique and use within 12 hours of opening. Propofol can change the color of the urine specifically to a green color from the phenol excretion.

Additional considerations include pain or burning on injection, which can be minimized by: Injecting in a larger, more proximal vein and giving lidocaine or an opioid prior to injection. Propofol also has antiemetic properties, although the exact mechanism is unknown, but has no analgesic properties. In the past, there was concern with using propofol in those with egg allergies. As we stated earlier, propofol is made from the lecithin, which is the egg yolk. By far, most people allergic to eggs are allergic to the protein in eggs or the egg whites, therefore there is actually very little risk of allergy.

Propofol Infusion Syndrome

One additional consideration for propofol is propofol infusion syndrome, which typically occurs with long infusions greater than 48 hours and a dose greater than 67mcg/kg/min. It is more common in children than adults. And additional risk factors include patient with inadequate oxygen delivery, sepsis, or significant cerebral injury. Signs and symptoms include: acute refractory bradycardia, metabolic acidosis with a base deficit greater than 10, hyperlipidemia, lipemia, rhabdomyolysis, enlarged or fatty liver, and renal failure.

Etomidate

The next drug we are going to discuss is etomidate. This is a short acting IV hypnotic used for general anesthesia. It works as a $GABA_A$ agonist and is favored for induction in hemodynamically unstable patient's due to minimal myocardial depression.

- Induction dose ranges from 0.2-0.4 mg/kg
- Onset of action in 30-60 seconds
- Duration of action 5-15 minutes

HR	MAP	Contractility	CBF	ICP	IOP	MV	Ventilatory Drive
↔	↔ or ↓	↔	↓↓	↓↓	↓	↔	↓

Etomidate Effects

The cardiovascular effects of etomidate are minimal; when the drug is injected in therapeutic doses, HR usually remains unaffected and mean arterial blood pressure is unaffected or only a small decrease is seen. Contractility is unaffected. This drug is cardiac stable for patients with coronary artery disease. Cerebral blood flow is decreased as well as intracranial and intraocular pressure, therefore it is considered safe for patients with intracranial pathologic conditions. Minute ventilation is unaffected, but ventilatory drive is slightly decreased, but much less than propofol.

Etomidate Considerations

Considerations for etomidate include cough and hiccups that can occur during injection. Spontaneous involuntary movements (or myoclonus) and tremor have also been noted after injection

Post-operative nausea and vomiting is a common side effect and etomidate is often referred to as E-vom-idate. And lastly Etomidate should be avoided in patients with a history of acute intermittent porphyria and although, controversial, etomidate can elicit acute intermittent porphyria in rats.

Another consideration for etomidate is the suppression of adrenocortical function. Cortisol and aldosterone synthesis is dependent on the enzyme 11-beta-hydroxylase, etomidate is a known inhibitor of this enzyme.

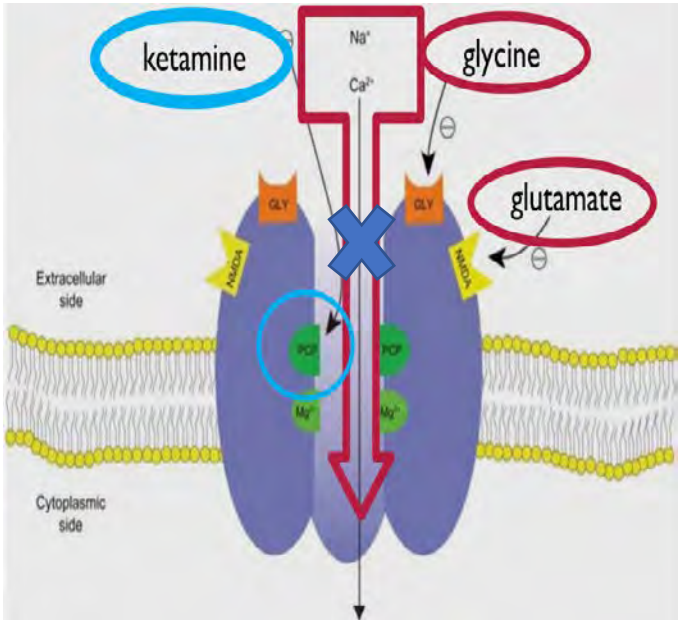
A single dose suppresses adrenocortical function for 5-8 hours. Because of this etomidate is CONTRAINDICATED in: sepsis, Addisonian crisis, and adrenocortical insufficiency.

Ketamine

The next drug we will discuss is Ketamine. It produces analgesia, unique dissociative hypnosis, and amnesia. It is used for induction of general anesthesia, as an adjunct to sedation cases, and for pain management

The mechanism of action is non-competitive N-Methyl-D-Aspartate (NMDA) receptor antagonism by acting on the cortex and limbic systems. It dissociates the thalamic (sensory) from the limbic system (awareness). Also exerts effects on: opioid receptors, muscarinic receptors, and GABA receptors (weak stimulation).

N-Methyl-D-Aspartate Receptor



The NMDA receptor is an ionotropic glutamate receptor for controlling synaptic plasticity and memory function. Glutamate, is the major excitatory neurotransmitter. Activation of NMDA receptor by binding of both glutamate and glycine results in the opening of the channel. This allows voltage-dependent flow of Na^+ and small amounts of Ca^{2+} ions into the cell and K^+ out of the cell. Ketamine has a specific binding site and blocks this conduction.

Ketamine Dosing

- Induction dose: 1-2 mg/kg
- Sedation dose: 0.25-0.5 mg/kg
- Adjunct to opioid analgesia: 5-10 mg per dose
- IM dose: 6-13 mg/kg

Intramuscular dosing or IM injection of ketamine is sometimes needed for children, those with learning disabilities, or those who won't tolerate an IV being placed in pre-op. Onset of action is 1-2 minutes with a peak effect in 3-5 minutes and duration of action of 5-15 minutes. It is important to note that ketamine is metabolized by hepatic cytochrome P450 enzymes forming norketamine metabolite, which is 1/5 to 1/3 as potent as ketamine.

Ketamine Effects

HR	MAP	Contractility	CBF	ICP	IOP	MV	Ventilatory Drive
↑↑	↑↑	↓	↑	↔ or ↑	↑	↓	↔

The effects of ketamine include an increase in heart rate and mean arterial pressure. A slight decrease in contractility is seen. A slight increase in cerebral blood flow is seen, a slight increase in intracranial pressure or no change at all, intraocular pressure is slightly increased. Minute ventilation is slightly decreased and ventilatory drive is unaffected.

Ketamine Considerations

Ketamine increases sympathetic tone, which is useful if a patient is hemodynamically unstable, but harmful in coronary artery disease patients. It causes bronchodilation and maintains pharyngeal and laryngeal reflexes, but also increases salivation. Lastly, it can cause unpleasant hallucinations and emergence delirium may occur. Although this is rarely seen with a dose less than 0.5 mg/kg and benzodiazepines will help alleviate this.

Dexmedetomidine

The next drug we will discuss is dexmedetomidine which has sedative, amnestic, and analgesic effects. It is used in procedural sedation in the OR and short-term sedation in the ICU, as well as for fiberoptic intubations.

The mechanism of action is a selective alpha 2 adrenergic agonist, which means it inhibits adenylyl cyclase activity and reduces brainstem vasomotor center-mediated CNS activation; which explains its sedative effects. It is desirable for sedation with very minimal respiratory depression and maintenance of arousability. Patients are actively able to cooperate with various procedures.

Dexmedetomidine Dosing

- Bolus: 1 mcg/kg x 10 minutes before maintenance drip
- Infusion and sedation dose range: 0.2-1.0 mcg/kg/hr

The onset of action is less than 5 minutes with a peak of 15 minutes and a duration of action of 2-3 hours for a single injection.

Dexmedetomidine Effects

HR	MAP	Contractility	CBF	ICP	IOP	MV	Ventilatory Drive
↓	↑ Bolus ↓ Infusion	↔	↓	↔	↔	↔	↔

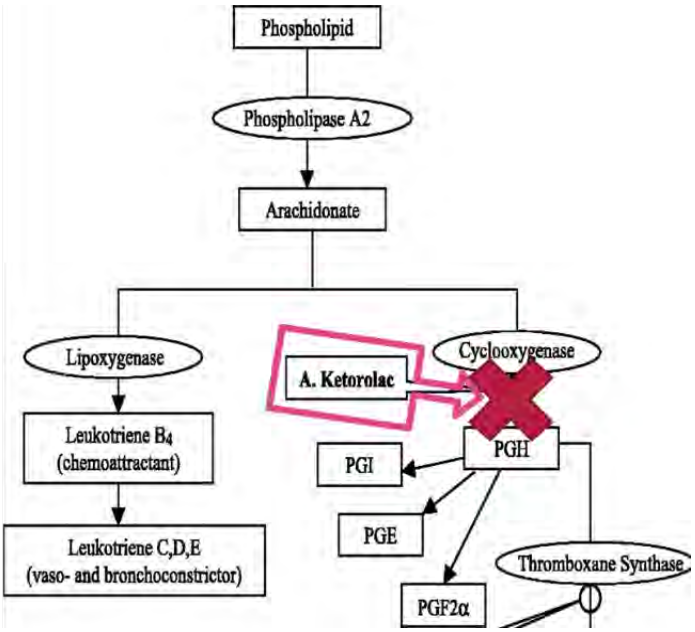
Dexmedetomidine has a biphasic effect on blood pressure with decreased blood pressure observed at lower concentrations and elevated blood pressure at higher concentrations. Rapid IV administration or bolus has been associated with bradycardia and low blood pressure due to peripheral α2-receptor stimulation. Contractility is unaffected. Cerebral blood flow is slightly decreased with intracranial and intraocular pressure both unaffected. Minute ventilation and ventilatory drive is also unaffected.

Caution in patients with 2nd or 3rd degree heart block, hypovolemia, or hypotension. Bradycardia and hypotension occur in up to 42% of patients and might cause profound left ventricular dysfunction and refractory shock. Usually, these temporary effects can be successfully counteracted with atropine, ephedrine, and volume supplementation. However, clinicians need to be well informed about the potential of dexmedetomidine to cause bradycardia, which may progress to pulseless electrical activity, particularly in patients older than 50 years and patients with cardiac abnormalities like 2nd or 3rd degree heart block.

Ketorolac

The next drug we will discuss is ketorolac or Toradol, which is a nonsteroidal anti-inflammatory drug (NSAID) used for its analgesic, anti-inflammatory, and antipyretic actions.

In response to injury or certain diseases, such as arthritis, cyclooxygenase (COX), as seen in the picture, is involved in producing prostaglandins. These prostaglandins cause pain, swelling and inflammation. The mechanism of action for ketorolac is inhibition of prostaglandin synthesis by blocking cyclooxygenase. When ketorolac blocks



the production of these prostaglandins it, thereby, is effective at relieving pain and inflammation.

- Usual dose: 15-30 mg
- Onset of action: 10 minutes
- Duration of action: 6 hours

Ketorolac Considerations

Ketorolac is contraindicated in patients with GI bleeding as it inhibits platelet function and can enhance bleeding. It is also contraindicated in renal failure as it can cause significant volume depletion and further damage. Lastly, it should not be given to those with asthma as it has been associated with increased bronchospasm risk. The dose should be adjusted in those older than 65, less than 110 pounds, or those with an increased serum creatinine, usually 15 mg is given in these situations.

Ofirmev

The last drug we will discuss is Ofirmev, which is an IV formulation of acetaminophen, used for mild to moderate pain and reduction of fever. It can be used in adults and children age 2 years and older. The dose is 15 mg/kg in pediatric patients or small adults. In those greater than 50 kg, 1,000 mg is usually given over 15-minute infusion. Onset of action is within 5-10 minutes with a peak of action at 1 hour and a duration of 4-6 hours. IV acetaminophen therefore has a benefit to PO with its faster onset of action and can provide significant pain relief within 15 minutes after initiating infusion. Although, it is more expensive.

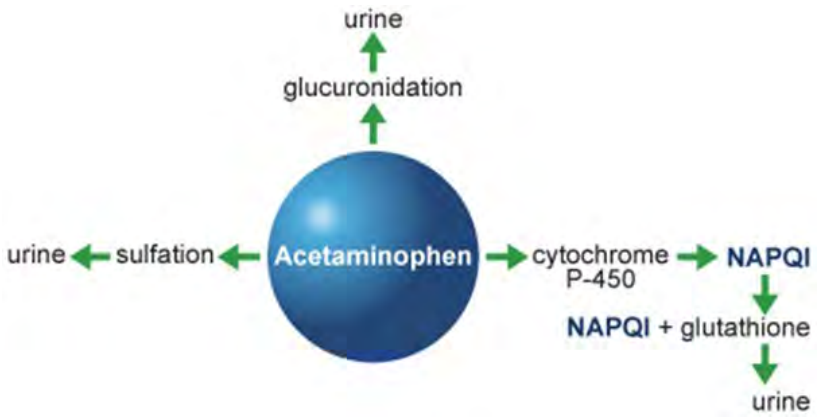
Ofirmev Considerations

Ofirmev has not been shown to affect platelet function or increase surgical bleeding. However, caution should be taken in those with alcoholism as alcohol interferes with metabolism. This drug is contraindicated in those with liver impairment or disease due to the metabolism of this drug and potential hepatotoxicity, which we will explain on the next slide. It is also contraindicated in those with a hypersensitivity to acetaminophen.

Hepatotoxicity - Acetaminophen

IV administration decreases exposure to the liver by nearly half, because PO, after intestinal absorption, goes to the liver first. This is called first-pass metabolism. IV acetaminophen bypasses this first-pass metabolism.

However, hepatic necrosis can occur and is potentially fatal with excessive doses.



Acetaminophen is metabolized by sulfation, glucuronidation, and cytochrome P-450 enzymes. N-acetyl-p-benzoquinone or NAPQI if formed and combine with glutathione, to be excreted by urine. Acetaminophen overdose will deplete glutathione, leaving the hepatotoxic NAPQI in the system, which leads to hepatotoxicity.

Summary

In summary, we have reviewed keywords and definitions in the understanding of IV anesthetics. We discussed the importance of GABA in relation to IV anesthetics and its structure and function. We have reviewed dosages, clinical uses, and key features for each of these drugs. And lastly, we have discussed risks, common side effects, and contraindications for each anesthetic.

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