


GENERAL ANESTHETICS


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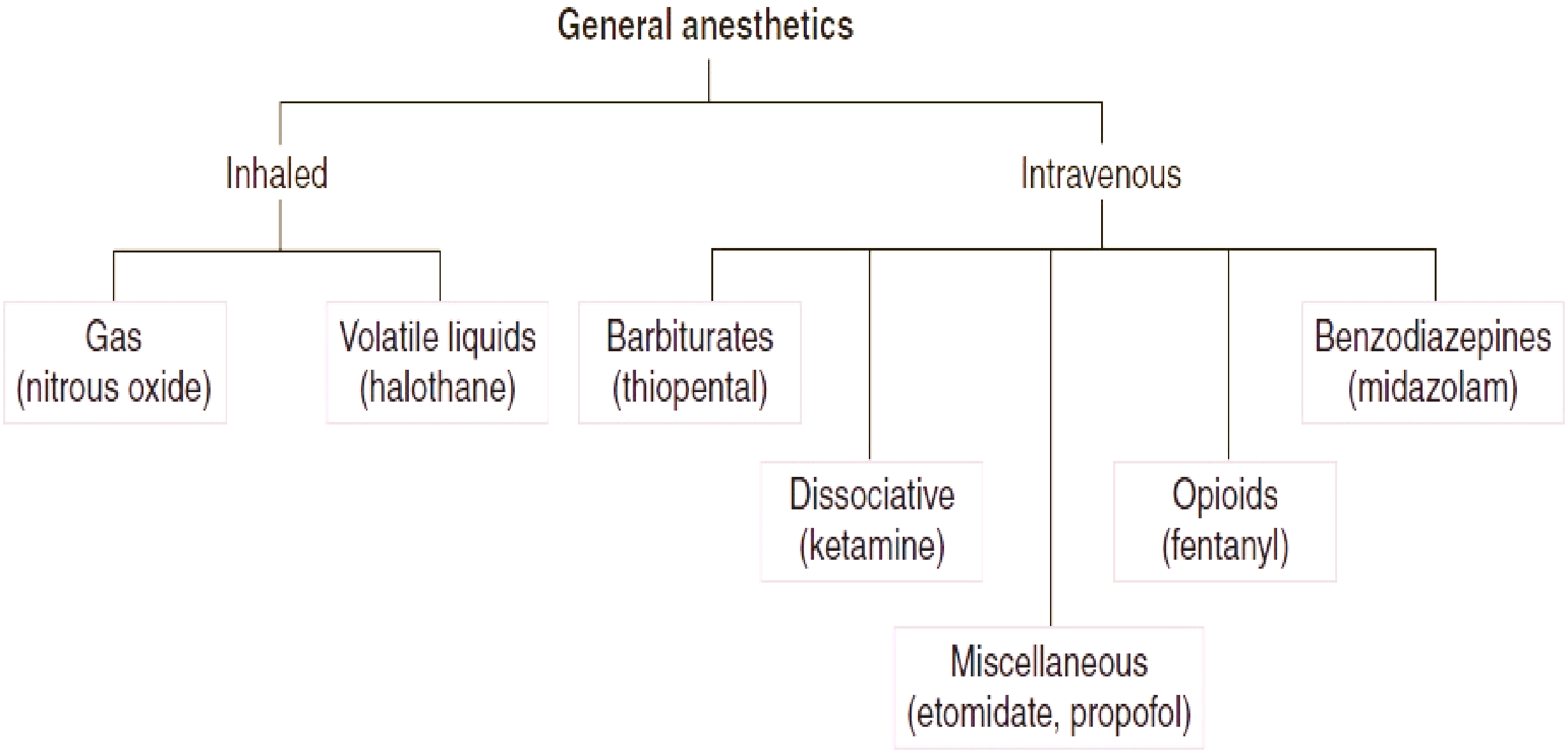
- General anesthesia is a state characterized by unconsciousness, analgesia, amnesia, skeletal muscle relaxation, and loss of reflexes.
- Drugs used as general anesthetics are CNS depressants with actions that can be induced and terminated more rapidly than those of conventional sedative-hypnotics.


General anesthetics
Inhaled
Intravenous
Gas (nitrous oxide)
Volatile liquids (halothane)
Barbiturates (thiopental)
Dissociative (ketamine)
Miscellaneous (etomidate, propofol)
Opioids (fentanyl)
Benzodiazepines (midazolam)

STAGES OF ANESTHESIA

Modern anesthetics act very rapidly and achieve deep anesthesia quickly. With older and more slowly acting anesthetics, the progressively greater depth of central depression associated with increasing dose or time of exposure is traditionally described as **stages of anesthesia.**

❑A. Stage 1: Analgesia

The patient has decreased awareness of pain, sometimes with amnesia. Consciousness may be impaired but is not lost.

❑B. Stage 2: Disinhibition

The patient appears to be delirious and excited. Amnesia occurs, reflexes are enhanced, and respiration is typically irregular.

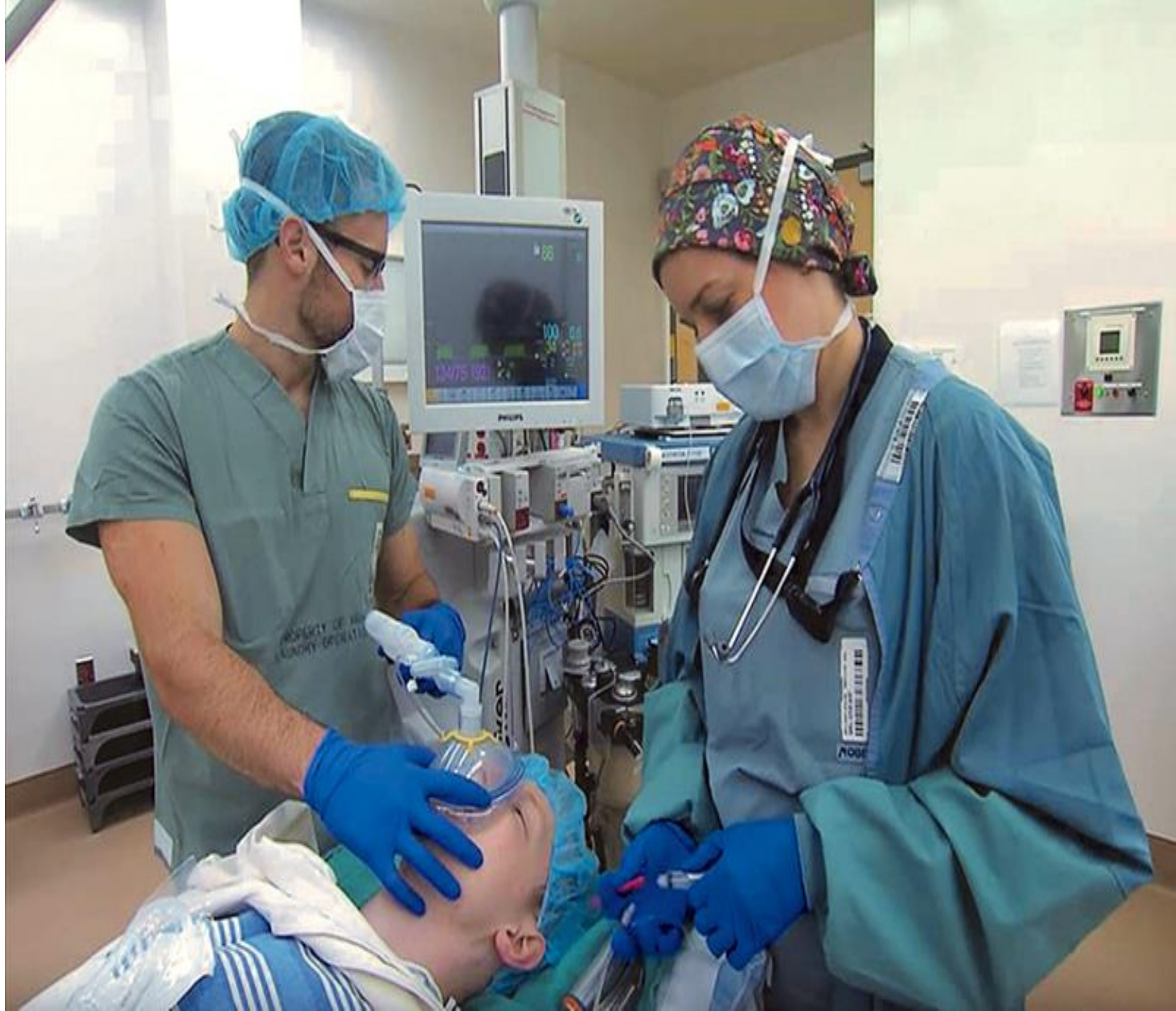
❑C. Stage 3: Surgical Anesthesia

In stage 3, the patient is unconscious and has no pain reflexes; respiration is very regular, and blood pressure is maintained.

❑D. Stage 4: Medullary Depression

In stage 4, the patient develops severe respiratory and cardiovascular depression that requires mechanical and pharmacologic support.

Current Levels of Anesthesia



➢Induction

This level encompasses the administration of preoperative medications, adjunctive drugs to anesthesia, and the anesthetics required for surgery.

➢Maintenance

This level begins when the patient has achieved a depth of anesthesia sufficient to allow the surgery to begin and ends upon the completion of the surgical procedure.

➢Recovery

The recovery phase begins with the termination of the surgical procedure and continues throughout the postoperative recovery period until the patient is fully responsive to his or her environment.

INHALED ANESTHETICS

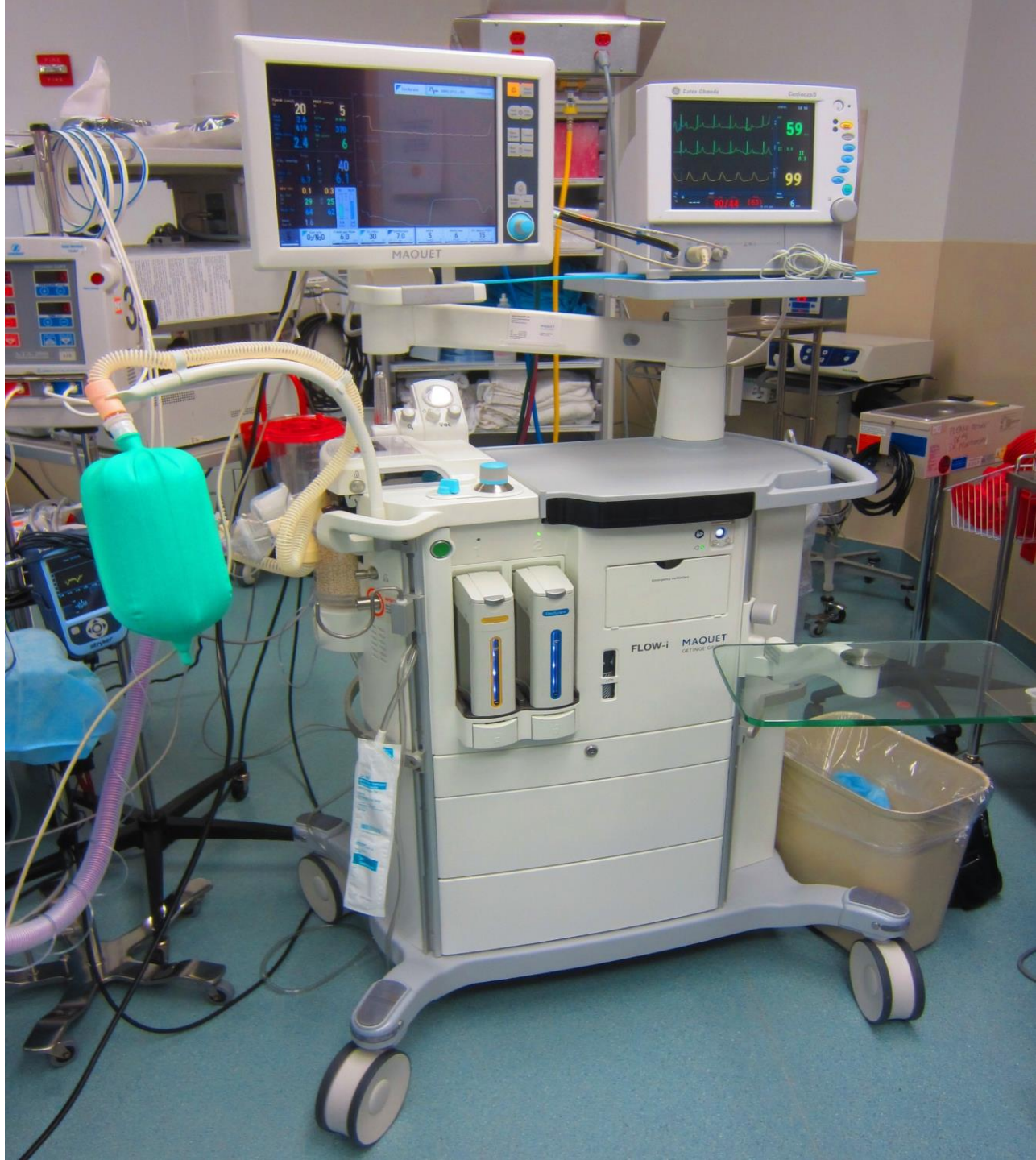
A. Classification and Pharmacokinetics

The agents currently used in inhalation anesthesia are nitrous oxide (a gas) and several easily vaporized liquid halogenated hydrocarbons, including halothane, desflurane, enflurane, isoflurane, sevoflurane, and methoxyflurane.

Factor Effect The Speed Of Induction Of Inhaled Anesthetic

1. Solubility
2. Inspired gas partial pressure
3. Ventilation rate:
4. Pulmonary blood flow
5. Arteriovenous concentration gradient

Effects of Inhaled Anesthetics



1. CNS effects

- Inhaled anesthetics decrease brain metabolic rate.
- They reduce vascular resistance and thus increase cerebral blood flow, this may lead to an increase in intracranial pressure.

2. Cardiovascular effects

- Most inhaled anesthetics decrease arterial blood pressure moderately.
- Enflurane and halothane are myocardial depressants that decrease cardiac output.
- Isoflurane, desflurane, and sevoflurane cause peripheral vasodilation. Nitrous oxide is less likely to lower blood pressure than are other inhaled anesthetics.

3. Respiratory effects

- All inhaled anesthetics cause a dose-dependent decrease in tidal volume and minute ventilation, leading to an increase in arterial CO2 tension.
- Inhaled anesthetics decrease ventilatory response to hypoxia even at subanesthetic concentrations.
- Nitrous oxide has the smallest effect on respiration.
- Most inhaled anesthetics are bronchodilators, but desflurane is a pulmonary irritant and may cause bronchospasm.

Intravenous Anesthetics

A. Barbiturates

- **Thiopental** and **methohexital** have high lipid solubility, which promotes rapid entry into the brain and results in surgical anesthesia in one circulation time (<1 min).
- These drugs are used for induction of anesthesia and for short surgical procedures.
- Barbiturates are respiratory and circulatory depressants; because they depress cerebral blood flow, they can also decrease intracranial pressure.

B. Benzodiazepines

- **Midazolam** is widely used adjunctively with inhaled anesthetics and intravenous opioids. The onset of its CNS effects is slower than that of thiopental, and it has a longer duration of action.
- Cases of severe postoperative respiratory depression have occurred.
- The benzodiazepine receptor antagonist, flumazenil, accelerates recovery from midazolam and other benzodiazepines.

C. Ketamine

- This drug produces a state of "dissociative anesthesia" in which the patient remains conscious but has marked catatonia, analgesia, and amnesia.
- The drug is a cardiovascular stimulant, and this action may lead to an increase in intracranial pressure.
- Emergence reactions, including disorientation, excitation, and hallucinations, which occur during recovery from ketamine anesthesia.

D. Opioids

- **Morphine** and **fentanyl** are used with other CNS depressants (nitrous oxide, benzodiazepines) in anesthesia regimens.
- Intravenous opioids may cause chest wall rigidity & respiratory depression with these drugs may be reversed postoperatively with naloxone.
- Alfentanil and remifentanil have been used for induction of anesthesia.

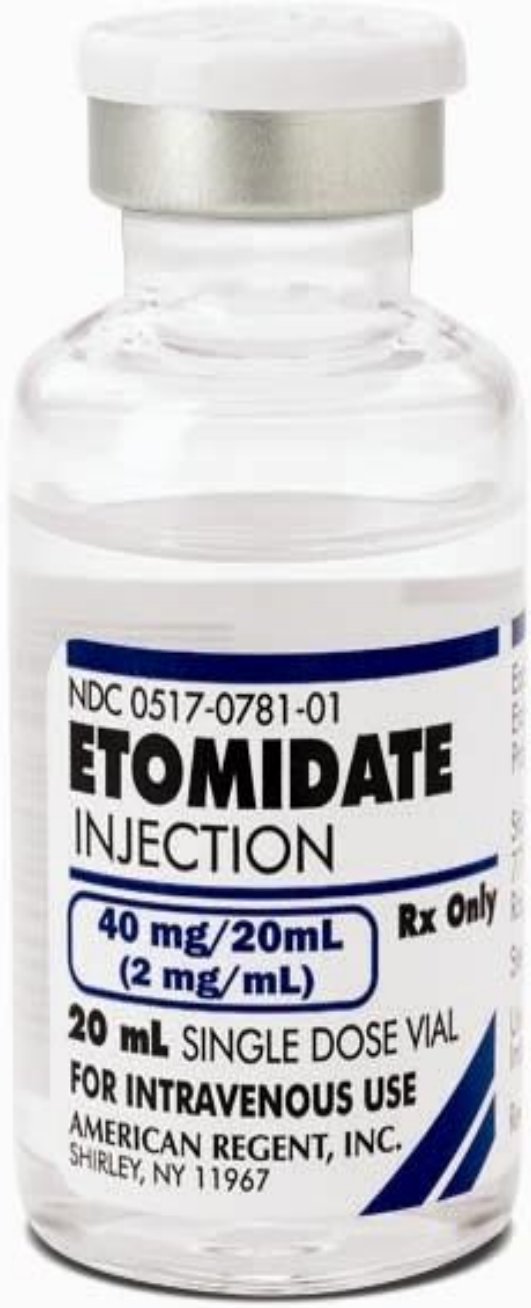
Propofol


20 mL
4463021
Latex Free
Propofol
200 mg/20 mL
10 mg/mL
Sterile / Stérile

- Propofol produces anesthesia as rapidly as the intravenous barbiturates, and recovery is more rapid.
- Propofol is also effective in producing prolonged sedation in patients in critical care settings.
- Propofol may cause marked hypotension during induction of anesthesia, primarily through decreased peripheral resistance.
- Fospropofol, a water-soluble prodrug form, is broken down in the body by alkaline phosphatase to form propofol.
- Onset and recovery are both slower than with propofol.
- Fospropofol appears to cause less pain at injection sites than the standard form of the drug, many patients experience paresthesias.

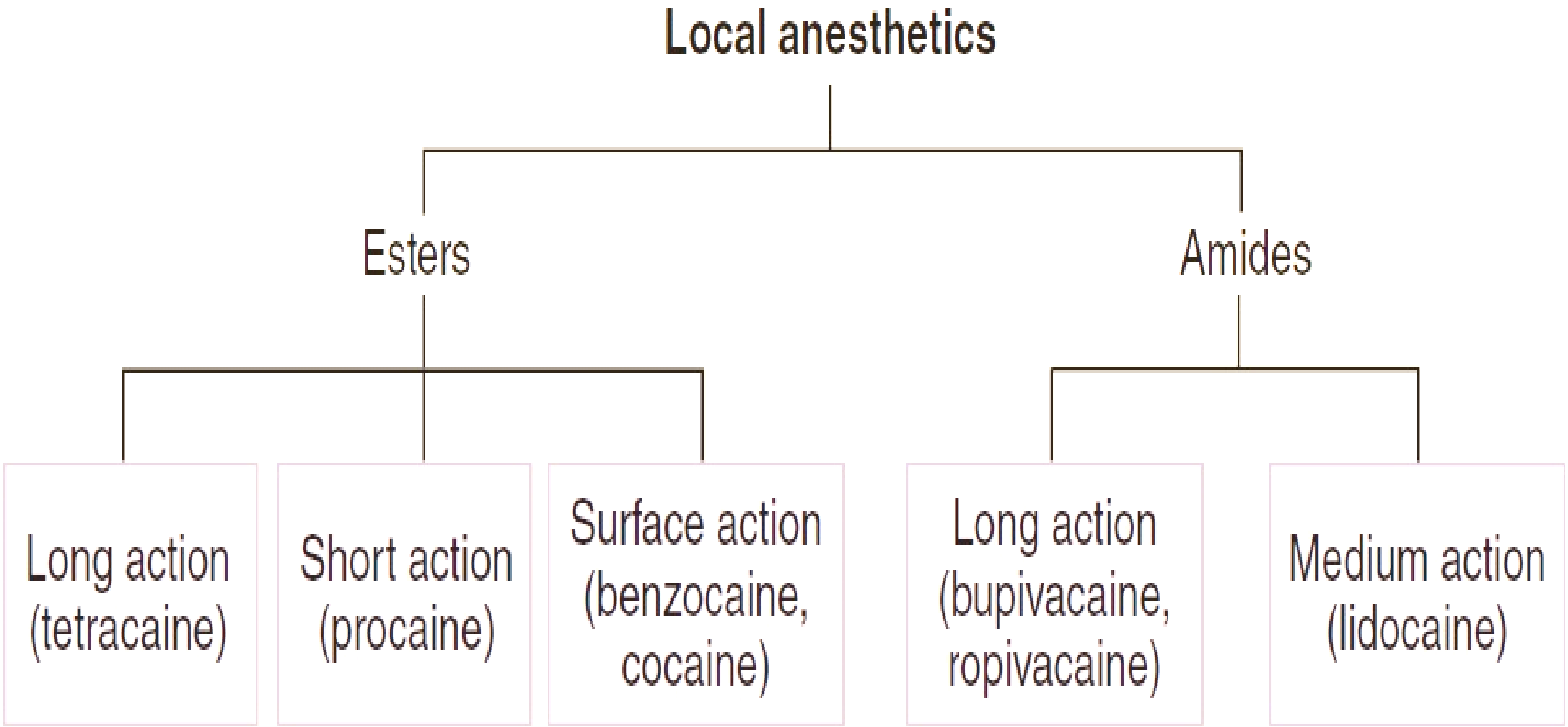
F. Etomidate

- This imidazole derivative affords rapid induction with minimal change in cardiac function or respiratory rate and has a short duration of action.
- The drug is not analgesic, and its primary advantage is in anesthesia for patients with limited cardiac or respiratory reserve.
- Etomidate may cause pain and myoclonus on injection and nausea postoperatively.
- Prolonged administration may cause adrenal suppression.



Local Anesthetics

- Local anesthesia is the condition that results when sensory transmission from a local area of the body to the CNS is blocked.
- The local anesthetics constitute a group of chemically similar agents (esters and amides) that block the sodium channels of excitable membranes.
- Because these drugs can be administered by injection in the target area, or by topical application.
- When given intravenously, local anesthetics have effects on other tissues.

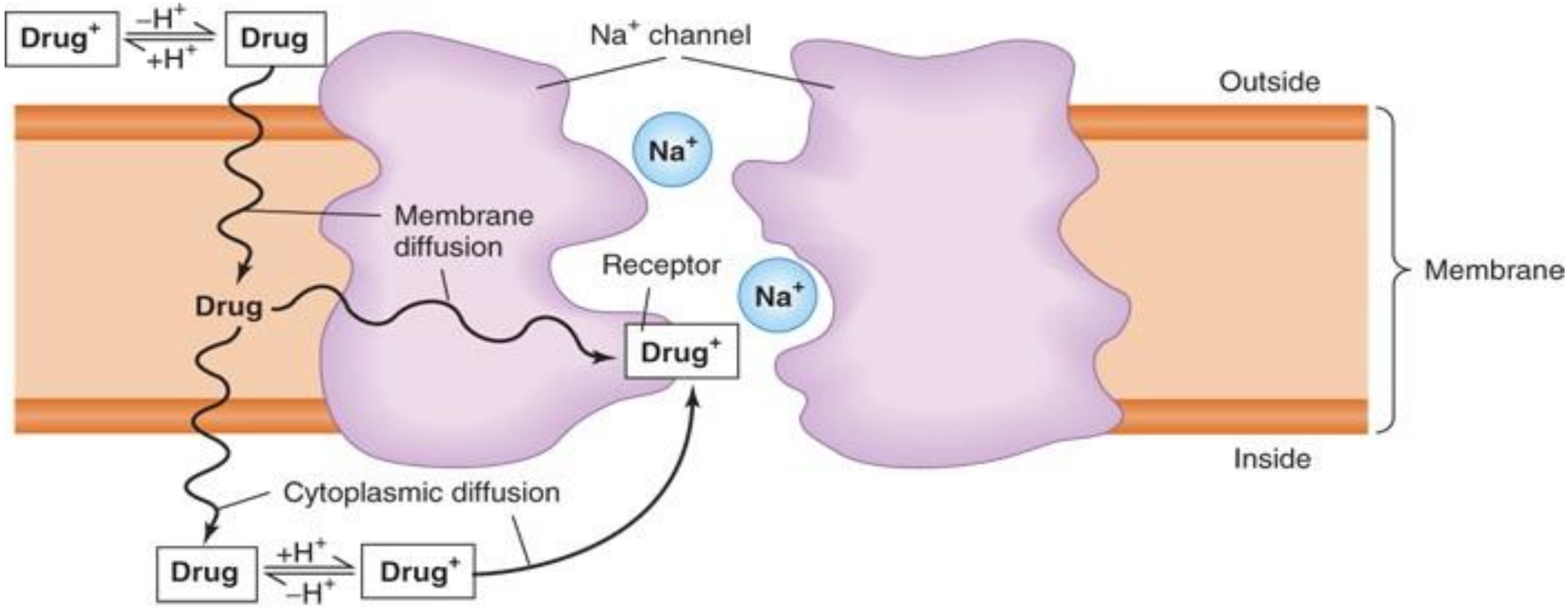

Local anesthetics
Esters
Amides
Long action (tetracaine)
Short action (procaine)
Surface action (benzocaine, cocaine)
Long action (bupivacaine, ropivacaine)
Medium action (lidocaine)

PHARMACOKINETICS

- Many shorter-acting local anesthetics are readily absorbed into the blood from the injection site after administration. The duration of local action is therefore limited unless blood flow to the area is reduced. This can be accomplished by administration of a vasoconstrictor.
- The longer-acting agents (eg, bupivicaine, ropivicaine, tetracain) are also less dependent on the coadministration of vasoconstrictors.
- Surface activity is a property of certain local anesthetics, especially cocaine and benzocaine, lidocaine, and tetracaine.

- Metabolism of ester local anesthetics is carried out by plasma cholinesterases (pseudocholinesterases) and is very rapid for procaine (half-life, 1–2 min), slower for cocaine, and very slow for tetracaine).
- The amides are metabolized in the liver, in part by cytochrome P450 isozymes. The half-lives of lidocaine and prilocaine are approximately 1.5 h.
- Bupivacaine and ropivacaine are the longest-acting amide local anesthetics with half-lives of 3.5 and 4.2 h, respectively.
- Liver dysfunction may increase the elimination half-life of amide local anesthetics

MECHANISM OF ACTION



PHARMACOLOGIC EFFECTS

- Differential sensitivity of various types of nerve fibers to local anesthetics depends on fiber diameter, myelination, physiologic firing rate, and anatomic location.
- In general, smaller fibers are blocked more easily than larger fibers, and myelinated fibers are blocked more easily than unmyelinated fibers.
- Fibers located in the periphery of a thick nerve bundle are blocked sooner than those in the core because they are exposed earlier to higher concentrations of the anesthetic.

CLINICAL USE

- Local anesthetics are also used in spinal anesthesia and to produce autonomic blockade in ischemic conditions.
- Slow epidural infusion at low concentrations has been used successfully for postoperative analgesia.
- Intravenous local anesthetics may be used for reducing pain in the perioperative period.
- Oral and parenteral forms of local anesthetics are sometimes used adjunctively in neuropathic pain states.

TOXICITY

- **CNS_**All local anesthetics are capable of producing a spectrum of central effects, including light-headedness or sedation, restlessness, nystagmus, and tonic-clonic convulsions. Severe convulsions may be followed by coma with respiratory and cardiovascular depression.
- **Cardiovascular_**Patients with preexisting cardiovascular disease may develop heart block and other disturbances of cardiac electrical function at high plasma levels of local anesthetics.

✓Bupivacaine may produce severe cardiovascular toxicity, including arrhythmias and hypotension.

✓levobupivicaine, is less cardiotoxic.

✓Cardiotoxicity has also been reported for ropivicaine when used for peripheral nerve block.

Treatment of Toxicity

- Severe toxicity is treated symptomatically; there are no antidotes.
- Convulsions are usually managed with intravenous diazepam or a short-acting barbiturate such as thiopental.
- Hyperventilation with oxygen is helpful.
- Occasionally, a neuromuscular blocking drug may be used to control violent convulsive activity.
- The cardiovascular toxicity of bupivacaine overdose is difficult to treat and has caused fatalities in healthy young adults.

Dental Concerns

- Vasoconstrictors increase the length of anesthetic effect which increases the chance of accidental patient self-mutilation.
- In small quantities, epinephrine acts as a vasodilator thus having the potential to increase post-op bleeding.
- Patients with cardiovascular disease, who can receive a vasoconstrictor, should receive the lowest dose possible by means of the best injection technique.
- Adverse Reactions
  - Ischemia
  - Prolonged pain, numbness or paresthesia
  - Feelings of nervousness or fast heart rate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