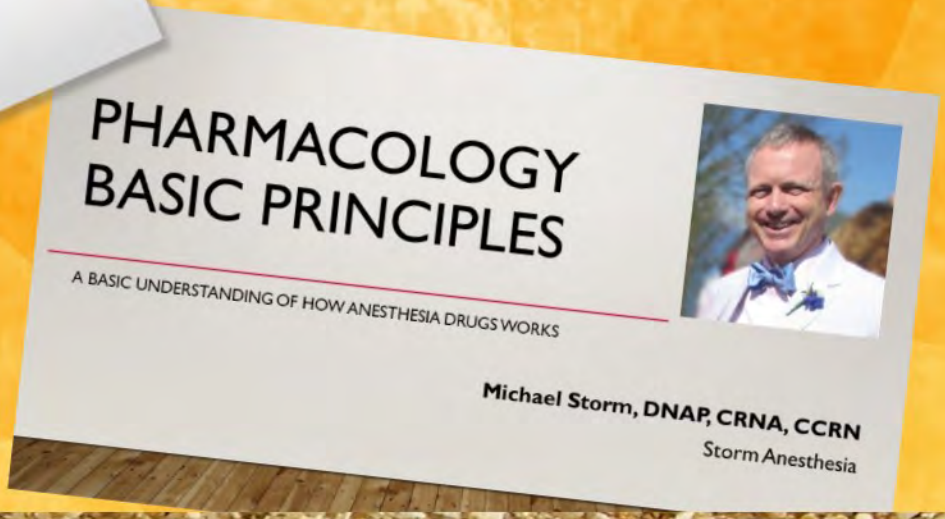
Anesthesia for Post Anesthesia Care Nurses

Video 1



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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
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Pharmacology Basic Principles

A Basic Understanding of How Anesthesia Drugs Work

Michael Storm, DNAP, CRNA, CCRN
Storm Anesthesia

This first lecture is presented by Michael Storm, DNAP, CRNA and will cover the basic principles of pharmacology.

Objectives

The objectives for this lecture are to

- review keywords and definitions
- review basic pharmacokinetics
- review basic pharmacodynamics

as they relate to anesthesia drugs.

At the end of the lecture you should have an enhanced understanding of

- routes of drug administration
- the concept of compartment modeling
- the concepts of distribution and redistribution of drugs
- the concepts of elimination and metabolism of drugs
- the basic concepts of how drugs influence the body
- drug dosing principles and why
- drug toxicities and therapeutic index
- receptor types found in the body where drugs act
- how drugs will interact with other drugs in the body, including herbal supplements
- and finally, I will cover basic sedation of patients in the PACU

Keywords and Definitions

First let's cover some basic keywords and definitions.

Additive Effect: Occurs when a second drug with properties similar to the first is added to produce an effect equal to the algebraic sum of the effect of the two individual drugs. The shorthand used to represent this is 1 + 1 = 2.

Adverse effect: Any noxious, unintended, and undesired effect of drug, when administered in therapeutic dose to humans.

Agonist: Drugs such as dopamine that attach to and activate specific receptors.

Antagonist: Drugs such as naloxone (Narcan) that attach to a specific receptor and do not activate it but instead prevent an agonist or body chemical such as a neurotransmitter from stimulating the receptor.

Bioavailability: The amount of drug expressed as a percent that enters the blood in an unchanged form after administration. Will vary depending on the route of administration.

Competitive Antagonist: When the concentration of the antagonist is higher than the agonist concentration, resulting in reversal or antagonism of the agonist. Examples include naloxone (Narcan) reversing fentanyl or flumazenil (Romazicon) reversing midazolam (Versed). The shorthand often used to represent this is 1 + 1 = 0.

Cross Tolerance: Tolerance to a drug because of an existing tolerance to a similar drug. An example of cross tolerance is a patient who has developed a tolerance to morphine due to repeated administration will also require higher doses of all other opioids as well.

Drug: Chemical agent used in treatment

Efficacy of a drug: Refers to the maximum effect that can be produced by a drug.

Half-life: Generally, refers to the elimination half-life, which is the time it takes the blood concentration to fall by one half. It takes four to five half-lives to eliminate a drug (98-99%).

Hyperreactivity: An abnormal reaction to an unusually low dose of a drug. For example, patients with Addison disease, myxedema, or dystrophia myotonica have hyperreactivity to unusually low doses of barbiturates.

Hypersensitivity (Anaphylaxis): A drug-induced antigen-antibody reaction. The particular hypersensitivity reaction can be either an immediate (anaphylactic) or a delayed reaction. Hypersensitivity reactions can occur with succinylcholine, antibiotics, and many other drugs administered in the PACU.

Hyporeactivity: An indication that a person needs excessively large doses of a drug to obtain a therapeutic or desired effect

Iatrogenic illness: Illness induced by treatment

Idiosyncrasy: An adverse drug reaction that occurs in a small number of persons and has no correlation to dosage or type of therapy. Postoperative liver dysfunction after halothane administration is an example.

Mechanism of Action: How a drug exerts its effect on cells or tissues. Usually acting via a receptor.

Pharmacodynamics: The study of the mechanisms of drug action and other biochemical and physiologic effects on the body.

Pharmacokinetics: The study of the movements of drugs throughout the body including the processes of absorption, distribution, biotransformation or metabolism, and excretion.

Placebo effect: Effect of the act of giving drug

Potency of a drug: The dose necessary of a particular drug to produce a specific effect designated as the effective dose (ED). When that effect is achieved in a particular percentage of patients, it is quantified as ED_{50} for 50% of the patients and ED_{95} for 95% of the patients who show an effect to the drug. The dose that will result in death to the patients is called the lethal dose (LD) and is quantified as LD_{50} and LD_{95} for 50% and 95% death respectively. Therapeutic index (TI) is LD_{50}/ED_{50} and higher is safer.

Potentiation: The enhancement of the action of one drug by a second drug that has no detectable action of its own. The shorthand often used to represent this 1 + 0 = 3.

Receptors: The portion on or in a cell, usually a protein complex, at which attachment of drugs leads to a physiologic response. The receptors are selective in that they recognize and bind only to specific pharmacologic or physiologic agents.

Redistribution: The movement of a drug from one tissue to another as equilibrium shifts in the body. Redistribution is the reason some of the highly lipophilic anesthetic drugs exit the brain quickly. Leads to short duration of action.

Side effect: Response to drug other than intended

Synergistic Effect: Addition of a second drug to a drug with properties like the first that results in an effect greater than the algebraic sum of the effect of the two individual drugs. The shorthand often used to represent this is 1 + 1 = 3.

Tachyphylaxis: An acute drug tolerance, for example, succinylcholine administered by intravenous drip. Over time, a higher drip rate is needed to achieve the necessary response.

Tolerance: A type of hyporeactivity acquired during chronic exposure to a drug in which unusually large doses are needed to reach a desired effect. A prime example is a person who has become dependent on opioids and needs larger than normal doses to elicit the desired therapeutic response.

Pharmacology

Pharmacology is the relationship between drug and body and pharmacists differentiate between pharmacokinetics ie. what the body will do to the drug and pharmacodynamics, ie. what a drug can do to the body. Pharmacokinetics are how does the drug make it into and out of the body, think: the pathway a drug will have to take before and after it exerts its effect.

Pharmacodynamics are what effects drugs have on the body.

To remember the difference between pharmacokinetics and pharmacodynamics you can use the mnemonic that dynamics has a d in the word which should remind you that: drug before body.

Pharmacokinetics

The movements of the drug through the body

Let us start with the pharmacokinetics, how the drugs arrive into and move through the body.

Administration

Drugs are administered in many ways. Each has its benefit and can be appropriate for a given situation. Certain drugs do not lend themselves to be administered via just any route, be that because of high first pass in the liver or because the drug is not absorbable via the intestines.

So how does a drug make it into the body? Several different routes are available to the manufacturer.

- Topical – applied to skin
- PO – by mouth
- SL – sublingual
- SQ – subcutaneous
- PR – per rectum
- IM – intramuscular
- IV – intravenous
- Inhalational – through the lungs
- Intrathecal – with the CSF (cerebral spinal fluid) in the spine

Most anesthesia drugs are administered using the IM, IV, Inhalational, or Intrathecal route. Due to NPO requirement prior to surgery for our patients, these routes are most beneficial to anesthesia.

Distribution

After the drug has been administered, it will be absorbed into the body. Pharmacologically we look at the body as single or multiple compartments; this allows us to calculate how much drug

is necessary to reach a certain concentration, which will give a specified and desirable effect. This is called mathematical modelling.

The simplest way of looking at this distribution is to see the body as a single, large compartment. This model considers the given drug fully distributed to all corners of the body instantly after the drug is given. Some IV drugs are considered single compartment drugs, eg. aminoglycosides (gentamycin, tobramycin, streptomycin). Such a drug has a fast distribution (it is considered fully equalized immediately), but also the elimination will be relatively fast and predictable, since it follows a straight-line algorithm.

By far the most drugs are not single compartment drugs, but multiple compartment.

A two-compartment drug consider the first compartment the central space, where the drug is primarily distributed. The second compartment is the peripheral space, where the drug will go next and where the drug may be stored for a while. Many drugs are seen this way. These drugs have a slower distribution and equalization, since they go to the areas with the highest perfusion (called the vessel rich group) first and then, secondarily, distributes throughout the body in the second, peripheral, compartment (muscle, skin, poor vessel group).

The distribution of drugs is dependent on the flow of blood throughout the body. The cardiac output will distribute its flow with most of it going to the vessel rich group, VRG, which consists of the lungs, liver, kidneys, brain, and heart. These organs are getting 75% of the cardiac output and at a much higher flow of blood, approximately 70-100 mL/minute/100 g tissue, than any other organ or tissue.

The muscle group consists of muscle and skin. This group receives 19% of the cardiac output, but at a much slower flowrate of 3 mL/minute/100 g tissue.

The fat group is fat and bone marrow and for this group the blood distribution has fallen once again to 6% of the cardiac output and at approximately 2 mL/minute/100 g tissue flow.

Vessel Rich Group		Muscle Group		Fat Group		Vessel Poor Group	
Lungs Liver Kidneys Brain Heart	75% 70-100 mL/min/ 100 g	Muscles Skin	19% 3 mL/min/ 100 g	Fat Bone marrow	6% 2 mL/min/ 100 g	Bones Ligaments Tendons Cartilage	~0% 0

As can be seen the flow is falling dramatically through the groups. The VRG organs received much more blood flow than any other compartment in the body.

Distribution - Redistribution

Distribution – redistribution is responsible for fast onset and fast offset of a drug. Given a bolus IV injection of a lipophilic drug, eg. propofol, this drug will rapidly go to the vessel rich group,

which includes the brain. Here it exerts it effect and the patient go to sleep, induction. Propofol will quickly move away from the vessel rich group organs and start distributing to the other compartments, ie. muscle group and fat group. This rapid movement away from the VRG organs towards the other compartments is called redistribution.

This redistribution is the reason some anesthetic drugs have a very short effect. They move away from the organs where they exert their effect, eg. propofol is a hypnotic causing severe slowdown in the brain. When the drug redistributes to the other groups, where there is no effect, eg. no sedative effect on the muscle or fat, the effect of propofol stops very quickly.

Drugs like these require a continuous infusion for their effect to be maintained.

Elimination

Immediately after distribution of a drug the metabolism and elimination begin.

Most drugs are metabolized by the liver, hepatic metabolism, using a process called cytochrome P450 reactions. A single enzyme, cytochrome P450 3A4 (abbreviated CYP3A4) is responsible for almost half of all drugs being metabolized.

These P450 reactions are divided into a Phase I and a Phase II reaction.

A phase I reaction will change the chemical structure of a drug and, for the most part, will render a drug less toxic (potent) than the original form. This removes most of the effect this drug is exerting on the body. Most drugs also become more water-soluble and, therefore, can be excreted by the kidneys.

For the drugs that could not be excreted after a phase I reaction, the liver will start a phase II reaction. Now these metabolites, created in the phase I reaction, will combine with some chemical substance and produce a water-soluble compound, which can be excreted by the kidneys.

For a drug to be eliminated from the body it must, for the most part, be water-soluble. Lipid-soluble drugs will have a much slower elimination, since these drugs very easily become reabsorbed back into the body.

Most drugs are eliminated, after some form of metabolism by the liver or by the kidneys. There is also a general rule that if all else fails, drugs will eventually be excreted by the kidneys. But, this can be a slow process.

There are other avenues for metabolism and elimination. Some drugs are eliminated via the lungs or through the skin. Other drugs are metabolized outside the liver, eg. lungs, skin, or intestines. For the most part, these avenues are of lesser concern than the liver and kidney.

All drugs are eventually eliminated or excreted from the body. This can happen in either a metabolized form, eg. changed by the liver, or as an intact molecule. After being metabolized by the liver, most drugs are eliminated by the kidneys in the urine, although, some are

eliminated via the liver produced bile. Then again, some are eliminated through the lungs or skin.

The elimination half-life of a drug is a valuable parameter that gives an accurate estimate of the length of time a drug will exert its effect on the body. This is useful when we decide how much and how often to dose and redose a given drug. Although, in anesthesia we often see cessation of effect by redistribution and not half-life.

Elimination half-life is a time value, which represents when 50% of a drug is removed from the plasma. In other words, the first half-life is when the body has excreted half of the initial dose of drug. The next half-life is when the body has excreted half of the remaining concentration of drug.

In anesthesia, this is not necessarily a very useful parameter, since many drugs stop their effect by redistribution, eg. propofol. When a drug redistributes away from its primary target the effect of the drug diminishes, but it is still in the system.

A drug is considered "gone" when 4-5 half-lives have gone. As can be seen in this chart 4-5 half-lives equals 94-98 % elimination.

Elimination of drugs by half-life

Half-life	Drug removed this half-life time	Drug removed total	Remaining drug in body
0	0%	0%	100%
1	50%	50%	50%
2	25%	75%	25%
3	12.5%	87.5%	12.5%
4	6.25%	93.75%	6.25%
5	3.125%	98.875%	3.125%

When the function of the kidneys or the liver deteriorates the metabolism, and/or the elimination of drugs will suffer.

Decreased renal function results in decreased glomerular filtration rate, which leads to decreased elimination of drugs

The same happens when the liver has decreased flow or function. Phase I and phase II reactions will diminish, and metabolism of drugs will decrease.

When cardiac function decrease, the cardiac output will often go down, which leads to less perfusion, fluid build-up (overload), and, therefore, a change in drug distribution. Poor cardiac function requires careful and vigilant monitoring of the patient. Especially after anesthesia.

Pharmacodynamics

The dynamic effect the drug has on the body

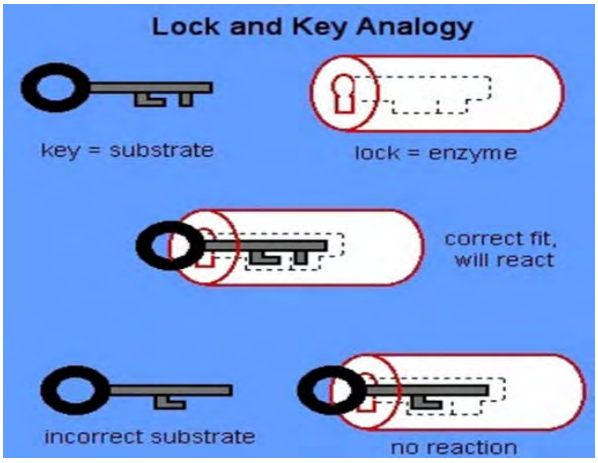
Now we have covered the basics of pharmacokinetics and I will continue onto what the drug does to the body, pharmacodynamics.

Most pharmacodynamics will be covered in other lectures that are specific for individual drugs. This lecture will cover the basic pharmacodynamic principles.

Receptor Theory

We believe that drugs work by attaching themselves to a specific receptor at specific places in the body. The same receptor will often give a different reaction when the receptor is in a different location in the body.

A receptor is a naturally occurring protein or protein complex that will have some form of endogenous activity. An endogenous activity is when a receptor reacts to input from the body and not from some outside actor, ie. the heart beats faster when we get scared due to release of endogenous catecholamines.



Pharmaceuticals are drugs that can attach themselves to a specific receptor. After being absorbed and distributed, they search for this specific receptor and then attach to this site.

We consider it a lock and key principle, where only a correctly sized key (the drug) fits into a specific key hole (the receptor) in the lock.

If the key is smaller, larger, has incorrectly positioned teeth, or is incorrectly shaped it will not fit into the lock. Therefore, there will not be an effect of that drug on this receptor.

Only when the drug finds the correct receptor will the key fit the keyhole and unlock the lock, which causes an effect.

Efficacy of Drug

The efficacy of a drug is determined by a receptors willingness to accept the drug to bind to it. This is called affinity.

How strong the effect will be after a drug attaches itself to a receptor is called the intrinsic activity. A stronger activity means an increased intrinsic activity.

When a drug binds to a receptor it does not, for the most part, remain bound indefinitely to this receptor site. It will quickly be knocked off the receptor, but then another close by drug will attach to the receptor instead. The drug effect is determined by having an adequate drug concentration around the receptor, so whenever the first drug molecule is knocked off another is readily available to attach to the receptor. To reach this level of drug concentration we must give an adequate dose of the drug and often must redose at specific intervals to maintain the effect we desire.

Dosing of Drugs

Once adequate amounts of drug binds to a receptor it will trigger the action, we call this the therapeutic effect.

Mostly, increasing the amount of drug we give, will increase the effect.

Now, we cannot just give unlimited amounts of drugs. All drugs are inherently unsafe.

Basically, they are all poisons. It is the amount, the dose, of the drug we give that will determine if we primarily create a desirable effect or undesirable effects, called adverse reactions or side effects. But it is important to realize that all drugs are potentially poisons.

There is no magic bullet. All drugs have multiple effects, unfortunately, not just the desired effect. So, we must determine how many and which side effects we can tolerate.

Increasing the dose will increase the concentration in the body of the drug. This may cause some undesirable effects.

If this undesirable effect is minor, we call it a side effect, eg. nausea after giving narcotics or dry mouth with scopolamine.

If the undesirable effect is worse, we consider this a toxic effect. This is never what we are after. We can live with side effects, but not toxic effect. We will consider respiratory depression after narcotics or cardiac dysrhythmias, confusion or psychosis after digitalis, toxicities.

Drug Safety

The effective dose of a drug is the amount of drug that produces a specific, desirable effect. When 50% of individuals receiving this specific dose have the desirable effect we call this dose the ED50 or effective dose for 50%. Respectively, when 95% of the individuals receiving a specific dose have the desired effect we call that the ED95.

Given a higher dose, eventually it will be a poison and possible have a lethal effect. Following the same principle as with effective dose, when 50% of individuals receiving a specific dose dies we call this LD50 or lethal dose 50%. Again, respectively, when 95% of individuals receiving a specific dose dies we call that LD95.

The relationship between lethal dose 50% and effective dose 50% is called the therapeutic index or TI. The higher this ration is the safer the drug, since it will require a relatively higher amount of drug to cause death.

Receptor Types

Now let's turn our attention to what happens when a drug attaches to a receptor.

An agonist drug will enhance or activate a receptor and trigger the receptors endogenous effect, eg. isoproterenol (Isuprel) attaches to a beta receptor and we see an increase in the beta receptors activity, like increased heart rate.

An antagonistic drug that binds to a receptor will block the receptors endogenous effect, eg. metoprolol (Lopressor) will block the endogenous effect of a beta receptor, like slowing the heart rate.

A partial agonist drug will bind to a receptor and activate the receptor, but not to the full extend as seen with an agonist. The result is a smaller effect.

Finally, an agonist/antagonist drug will, if given alone, partially activate the endogenous effect of the receptor, but if given along an agonist drug the agonist/antagonist drug will reverse some of the endogenous effect caused by the agonist drug. An example could be the newer treatment for opioid addiction, buprenorphine (Subutex). This is an opioid agonist, but much weaker than regular opioids, eg. heroine, so when given alone buprenorphine has some opioid effect. When given after the patients has taken an opioid it acts as an antagonist to the opioid the patient took. The benefit is that the agonistic effect buprenorphine exert will allow for suppression of withdrawal symptoms and cravings that arises when buprenorphine takes away the opioid effect from the drug the patient took.

Regulation of Receptors

Up- and Down-regulation

Although there is a baseline amount and efficacy of receptors this can change.

If the body become injured, eg. a traumatic spinal cord lesion, although there is not a loss of receptors, when these receptors are activated they do not send their activated signal to the brain, it is cut off at the lesion site. Therefore, the brain treats the lack of response as if there were no receptors available and tries to increase the number of available receptors. This is called up-regulation of receptors. Also, chronic administration of an antagonist drug will cause the body to sprout more receptors to compensate for the diminished response of the blocked receptors. Therefore, an increased dose is now needed to keep blocking the receptors.

Similarly, if we overstimulate the body with an agonist drug the body “get tired” of this enhanced response it must produce and responds with a down-regulation, removal from service, of receptors. Also, some agonist drugs create this down-regulation and we call it tolerance, which requires an increased dose to create the expected response.

Drug-Drug Interactions

It is to be expected that patients are prescribed other medications than the ones we use during the surgery. These medications may, possibly, interact with the drugs we give during our anesthetic. These interactions may cause unexpected reactions, some of which may be noted in the PACU. A common drug prescribed for hypertension is an angiotensin-converting-enzyme inhibitor ~ or ACEI. If not stopped at least 24 hours prior to anesthesia these drugs are notorious for causing hypotension during anesthesia. Often this hypotension is difficult to treat with our usual treatment options, eg. ephedrine or phenylephrine, and may require vasopressin bolus and/or infusion.

There may be changes in the absorption, distribution, metabolism, or excretion, which are all pharmacokinetic changes. This can result in an altered pharmacologic response than the one intended for the drug given.

The changes may be of pharmacodynamic nature, where giving one drug will change the effect of another drug we give during anesthesia.

Pharmacokinetic Interactions

Examples include adding a vasoconstrictor to a local anesthetic. This will slow down the absorption of the local anesthetic, because the vasoconstrictor reduces the blood flow in the area where the local anesthetic is injected. This will ultimately prolong the effect of the local anesthetic because it takes longer to remove it from the tissue.

A patient who takes Maalox or Mylanta, which are aluminum containing antacids commonly taken over the counter, will reduce the absorption of drugs like tetracycline, digoxin, phenytoin, and chlorpromazine. This could be cause for diminished effect of those drugs. At the same time Maalox and Mylanta will enhance absorption of pseudoephedrine and levodopa, which will cause an increased effect of these drugs.

Grapefruit juice significantly inhibits CYP3A4 reactions, remember this enzyme is responsible for almost 50% of all metabolism of drugs by the liver. This will increase the concentration of those drugs, eg. antifungal drugs, TB drugs, SSRIs, and some antibiotics.

Other drugs, Paxil, Prozac, and quinidine, inhibit another enzyme, CYP2D6. This enzyme is necessary to convert codeine to morphine. Therefore, when CYP2D6 is inhibited codeine, oxycodone, and hydrocodone will not have their intended effects.

Certain antibiotics, eg. the aminoglycosides tobramycin, gentamycin, and streptomycin as well as the polymyxins, Cortisporin and triple antibiotic ointment, inhibits the release of acetylcholine from the presynaptic membrane. This will intensify the neuromuscular blockade caused by a non-depolarizing neuromuscular blocker like rocuronium and vecuronium. This will make the reversal of these drugs difficult.

A feared reaction among opioids is the response seen when a patient taking a mono amine oxidase inhibitor, an antidepressant, and we then give the patient meperidine, Demerol.

What happens is that MAOIs increase the amount of two catecholamines, norepinephrine and serotonin, in the nerve synapses by blocking the breakdown of these catecholamines. This will increase the amount of available norepinephrine and serotonin in the synapse. Serotonin is a monoamine neurotransmitter in the GI and CNS synapses. The normal cycle is for serotonin to move back into the cell after its use in the synapse; this is called reuptake. Meperidine is a, albeit weak, serotonin reuptake inhibitor. Therefore, when we have created an increased amount of serotonin in the synapse with the mono amine oxidase inhibitor and then meperidine starts to block some of the reuptake, catastrophe is set to happen. We call this a serotonin crisis or serotonin syndrome. This can be mild or very severe. We differentiate this reaction in two types, type 1, an excitatory phase with headache, agitation, muscle rigidity, and fever, and a type 2, which is more of a depressive character with hypotension, respiratory depression and coma.

Mono amine oxidase inhibitors are very slow to be eliminated from the body and can stay around for up to three weeks.

Patients on chronic steroid treatment, which for this purpose is defined as more than 5 mg/day for more than 3 weeks, has an inadequate ability to increase their own production of steroid as needed. This lack of response is not a drug-drug interaction, but it could be the cause of hypotension, respiratory depression, and delayed recovery, which may be noted in the PACU.

The goal in modern day medicine is to continue the patients normal dose regime, but if the patient has stopped taking their normal dose it is recommended to treat with hydrocortisone pre- and post-operatively. Only if the surgery is long should it be considered to give an intra-operative dose as well.

Herbal Medicines or Supplements

Many patients take herbal supplements. It has been estimated that between 20-35% of the general population takes one or more herbal supplements. None of these are FDA approved or regulated.

Many of these patients, upwards 70%, do not report that they take herbal medications. Most do not consider these to be medicine, so they don't feel a need to inform their healthcare practitioners about these drugs. Many have not told their surgeon about these supplements either.

These herbal supplements can interact with the drugs we use during anesthesia and in PACU.

It is generally recommended to refrain from these drugs for 1-2 weeks prior to surgery. There are a few exceptions, eg. valerian, where an abrupt discontinuation could cause withdrawal

Herbal Medications or Supplements and Their Untoward Effects

Herb	Uses	Untoward Effects	Discontinue
Ephedra	Weight loss, congestion, bronchospasm	Dysrhythmias, ↑BP, bronchodilation, diuresis, tachycardia	24 hrs
Feverfew	Headache, fever, migraine	Inhibits platelets, insomnia, anxiety	1 week
Garlic	Hyperlipidemia, atherosclerosis	Increase bleeding times, increases fibrinolysis	1 week
Ginger	Nausea, vomiting, motion sickness	Increase bleeding times	1 week
Ginkgo biloba	Memory, dementia, peripheral vascular disease	Increase bleeding times, inhibits platelets	36 hours
Ginseng	Improve concentration, hypoglycemia, stress	Increase bleeding times, insomnia, irritability, mania, interact with digoxin, warfarin, lithium	1 week
Kava	Anxiety, stress, insomnia	Potentiate barbiturates, inhibits platelets, potentiates anesthetics	24 hours
St. John's wort	Antidepressant, sedative	Inhibits CYP3A4	5 days
Valerian	Sedative, insomnia, muscle relaxant	Potentiates sedatives	Taper off over 2 weeks

syndrome, very much like benzodiazepine withdrawal. This drug is recommended to be tapered off slowly.

These herbal supplements are giving us the most complications. This list is just a condensed version and these herbal supplements could have many more uses and complications than listed here.

- Ephedra is used for weight loss, congestion, and bronchospasm. The main issue is it can cause dysrhythmias, hypertension, bronchodilation (which is mostly a good thing), diuresis, and tachycardia. Recommended to stop for a minimum of 24 hours.
- Feverfew is used against headache, fever, and migraine. It inhibits platelets (bleeding issue), causes insomnia and anxiety. Recommended to stop for at least 1 week.
- Garlic is used for hyperlipidemia and atherosclerosis. Can cause increased bleeding times and increased fibrinolysis. Recommended to stop for at least 1 week.
- Ginger is often used against nausea, vomiting, and motion sickness. It can increase bleeding times and should be stopped at least 1 week prior to surgery.
- Ginkgo biloba is advertised to improve memory, help with dementia and peripheral vascular disease. It also increased bleeding times and may inhibit platelets. Recommended to stop for at least 36 hours.
- Ginseng is often used to improve concentration, help with hypoglycemia, and stress. It can increase bleeding times, cause insomnia, irritability, mania, and interact with digoxin, warfarin, and lithium. Recommended to stop for at least 1 week.
- Kava is for anxiety, stress, and insomnia. It may potentiate barbiturates, inhibits platelets, and generally potentiates anesthetics. Recommended to stop for at least 24 hours.
- St. John's wort is used as an antidepressant and a sedative. It inhibits CYP3A4, which is an essential enzyme to metabolize drugs in the liver. Recommended to stop for at least 5 days.
- Valerian is used as a sedative, against insomnia, and as a muscle relaxant. This will potentiate sedatives used for anesthesia. Should be tapered off over 2 weeks.

Sedation in PACU

It is not uncommon for PACU patients to require sedation to avert the stress. The noise in the PACU, be that from alarms, agitation of other patients, loss of control, confusion, or pain will all increase the patients stress level and may warrant sedation. Stress can cause increased oxygen consumption due to increased heart rate, blood pressure and exacerbated hyperglycemia. Remember, stress from surgery has already increased cortisol levels, which causes hyperglycemia.

When deciding to sedate a PACU patient it is paramount to consider what was given during anesthesia. Many of the anesthetic drugs or gasses will still have effect in PACU; well, that is the

reason the patient goes to PACU in the first place. Therefore, it should be obvious that any sedation of the patients should be done gently and with a vigilant eye towards adverse effects of the sedation.

It is strongly recommended, although not a standard, to use some form of a sedation scale. There are several useful scales available, eg. RAMSAY Sedation Scale (RSS) is the among the oldest going back to the 70s, MOAASS or Modified Observer's Assessment of Alertness/Sedation Scale, the Sedation Visual Analogue Scale, or POSS, the Pasero Opioid-induced Sedation Scale. Either scale could be beneficial to the PACU nurse.

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