# Central Nervous System Pharmacology Practice Test (Sample)

## Study Guide


**Everything you need from our exam experts!**

# Questions

**1. What is a common side effect of taking SSRIs in terms of sexual health?**

**A. Increased libido**

**B. Delayed ejaculation**

**C. Heightened arousal**

**D. Premature ejaculation**

**2. What type of muscle relaxant is characterized as a nicotinic antagonist with reversible effects?**

**A. Depolarizing muscle relaxant**

**B. Non-depolarizing muscle relaxant**

**C. Muscle relaxant with CNS effects**

**D. Heart-affecting muscle relaxant**

**3. What dangerous interaction can occur with GABAa drugs?**

**A. Hypertension when combined with diuretics**

**B. Life-threatening respiratory depression with CNS depressants**

**C. Increased heart rate with stimulants**

**D. Decreased effectiveness of anticoagulants**

**4. In what form is Atomoxetine typically administered?**

**A. Inhalation**

**B. Intravenous**

**C. Oral tablets**

**D. Topical patch**

**5. What is a common pharmacokinetic property of phenytoin at high doses?**

**A. First-order kinetics**

**B. Zero-order kinetics**

**C. Linear absorption**

**D. Rapid metabolism**

**6. What does a lower MAC indicate about an anesthetic agent?**

- **A. Higher potency**
- **B. Faster induction time**
- **C. Increased side effects**
- **D. Reduced effectiveness**

**7. Which benzodiazepine is used for status epilepticus?**

- **A. Temazepam**
- **B. Lorazepam**
- **C. Oxazepam**
- **D. Alprazolam**

**8. Which drug class does bupropion belong to?**

- **A. SSRI**
- **B. TCA**
- **C. NE and DA reuptake inhibitor**
- **D. MAOI**

**9. What is the primary mechanism of action of atypical antipsychotics?**

- **A. Inhibition of dopamine and 5HT2 receptors**
- **B. Boosting serotonin levels exclusively**
- **C. Selective blockade of adrenergic receptors**
- **D. Enhancing GABA activity**

**10. What is a primary use of diazepam?**

- **A. Anxiety**
- **B. Sleep disorders**
- **C. Preoperative sedation**
- **D. All of the above**

# Answers

**1. B**
**2. B**
**3. B**
**4. C**
**5. B**
**6. A**
**7. B**
**8. C**
**9. A**
**10. D**

# Explanations

## 1. What is a common side effect of taking SSRIs in terms of sexual health?

**A. Increased libido**

**B. Delayed ejaculation**

**C. Heightened arousal**

**D. Premature ejaculation**

**Selective serotonin reuptake inhibitors (SSRIs) are commonly prescribed for conditions such as depression and anxiety, but they have several side effects, particularly concerning sexual health. One of the noteworthy effects is delayed ejaculation. SSRIs increase serotonin levels in the brain, which can lead to various changes in sexual function. The increased serotonergic activity tends to inhibit the ejaculatory reflex, causing a significant delay in ejaculation for many individuals taking these medications. This can be frustrating and distressing for patients, and it is an important consideration when discussing treatment options for those who may also be concerned about their sexual health. Increased libido, heightened arousal, and premature ejaculation are not typically associated with SSRIs. In fact, they can contribute to reduced sexual desire and arousal in some people. Therefore, delayed ejaculation stands out as a common and clinically relevant side effect associated with the use of SSRIs.**

## 2. What type of muscle relaxant is characterized as a nicotinic antagonist with reversible effects?

**A. Depolarizing muscle relaxant**

**B. Non-depolarizing muscle relaxant**

**C. Muscle relaxant with CNS effects**

**D. Heart-affecting muscle relaxant**

**The type of muscle relaxant characterized as a nicotinic antagonist with reversible effects is indeed a non-depolarizing muscle relaxant. Non-depolarizing muscle relaxants work by competitively blocking the neuromuscular junction, specifically the nicotinic acetylcholine receptors on the motor end plate. By preventing acetylcholine from binding to these receptors, they inhibit the transmission of impulses from nerve to muscle, leading to muscle relaxation. One of the key features of non-depolarizing agents is their reversibility; they can be reversed by administering agents that increase the availability of acetylcholine at the neuromuscular junction, such as anticholinesterases. This is in contrast to depolarizing muscle relaxants, which initially stimulate the receptor before causing paralysis and are not easily reversible. Muscle relaxants with central nervous system effects typically refer to those that act on the CNS to produce muscle relaxation without necessarily blocking the neuromuscular junction, which is a different mechanism. Heart-affecting muscle relaxants would not accurately describe either class, as their primary function is not to influence heart activity specifically, but rather to induce muscle relaxation in skeletal muscles.**

## 3. What dangerous interaction can occur with GABAa drugs?

**A. Hypertension when combined with diuretics**

**B. Life-threatening respiratory depression with CNS depressants**

**C. Increased heart rate with stimulants**

**D. Decreased effectiveness of anticoagulants**

**The interaction between GABAa drugs and other central nervous system (CNS) depressants is critical to understand due to the potential for life-threatening respiratory depression. GABAa receptor agonists, such as benzodiazepines, barbiturates, and certain anesthetics, enhance the inhibitory action of gamma-aminobutyric acid (GABA) in the brain, promoting sedation, muscle relaxation, and anxiolysis. When these drugs are used in combination with other CNS depressants—such as alcohol, opioids, or additional sedatives—the combined effects can significantly amplify sedation and respiratory suppression. As both types of drugs lower the respiratory function by diminishing the responsiveness of the brain's respiratory centers, this can lead to dangerously low levels of oxygen in the bloodstream and potentially result in respiratory failure. This interaction is particularly alarming because the effects can be unpredictable and vary among individuals, making monitoring and caution essential during combined therapy. Other options mention interactions that do not typically involve GABAa drugs. For instance, hypertension from diuretics, increased heart rate due to stimulants, and decreased effectiveness of anticoagulants are not associated with the pharmacological mechanisms of GABAa agonists, emphasizing the importance of recognizing the specific risks related to**

## 4. In what form is Atomoxetine typically administered?

**A. Inhalation**

**B. Intravenous**

**C. Oral tablets**

**D. Topical patch**

**Atomoxetine is primarily administered in the form of oral tablets. This method of delivery allows for convenient dosing and consistent absorption in the gastrointestinal tract, which is particularly important for medications used in the treatment of attention-deficit/hyperactivity disorder (ADHD). Oral administration also enables patients to take the medication at home without the need for medical supervision, making it more accessible for long-term use. Inhalation, intravenous administration, and topical patches are not common routes for Atomoxetine. Inhalation is often associated with respiratory medications, intravenous routes are typically reserved for medications requiring rapid effects or used in hospital settings, and topical patches are more often related to pain management or hormone delivery. Therefore, oral tablets are the preferred and standard formulation for Atomoxetine, aligning with its intended use and pharmacokinetic profile.**

## 5. What is a common pharmacokinetic property of phenytoin at high doses?

**A. First-order kinetics**

**B. Zero-order kinetics**

**C. Linear absorption**

**D. Rapid metabolism**

**Phenytoin demonstrates zero-order kinetics at high doses due to its metabolism being saturable. In the context of pharmacokinetics, zero-order kinetics means that the rate of drug elimination is constant, regardless of the drug concentration in the bloodstream. This occurs because the enzymes responsible for metabolizing phenytoin become saturated when high doses are administered, leading to a situation where the body cannot process the drug any faster, causing the plasma concentration to increase disproportionately with each additional dose. In contrast, at normal therapeutic doses, phenytoin may exhibit first-order kinetics, where the elimination rate is proportional to the drug concentration. However, as the dose rises to levels that exceed the metabolic capacity of the liver enzymes, the zero-order kinetics becomes predominant. This is a critical concept in understanding how dose adjustment and monitoring are vital for medications with saturable metabolism like phenytoin, especially to avoid toxicity. Other options like linear absorption and rapid metabolism do not apply correctly to this scenario. Linear absorption suggests that the absorption rate remains constant and is independent of concentration, which does not occur with phenytoin in the case of its saturable metabolism. Rapid metabolism would imply that the drug is processed quickly, which contradicts the zero-order**

## 6. What does a lower MAC indicate about an anesthetic agent?

**A. Higher potency**

**B. Faster induction time**

**C. Increased side effects**

**D. Reduced effectiveness**

**A lower MAC (Minimum Alveolar Concentration) indicates that an anesthetic agent is more potent. MAC is a standard measurement that reflects the concentration of an inhaled anesthetic required to prevent movement in 50% of patients in response to a surgical stimulus. A lower MAC value signifies that only a small concentration of the anesthetic is needed to achieve the desired level of anesthesia, thereby indicating a higher potency. This characteristic is important in clinical settings, as it allows for effective anesthesia with potentially lower doses, which can lead to a reduced risk of side effects and complications associated with higher doses. In contrast, a higher MAC value would suggest that higher concentrations are necessary for effective anesthetic action, pointing to lower potency. Understanding MAC helps anesthesiologists select appropriate agents tailored to individual patient needs during surgical procedures.**

## 7. Which benzodiazepine is used for status epilepticus?

**A. Temazepam**

**B. Lorazepam**

**C. Oxazepam**

**D. Alprazolam**

**The benzodiazepine most commonly used for status epilepticus is lorazepam. This medication acts as a potent anticonvulsant and is effective in rapidly terminating seizure activity. Lorazepam works by enhancing the effects of the neurotransmitter gamma-aminobutyric acid (GABA) at the GABA-A receptor, which leads to increased inhibitory signals in the brain. This property is particularly beneficial in a critical situation like status epilepticus, where immediate seizure control is necessary. In clinical practice, lorazepam is preferred due to its relatively long duration of action, which allows for a sustained effect and prevents the recurrence of seizures. Unlike certain other benzodiazepines, lorazepam can also be administered intravenously, making it suitable for emergency situations where rapid action is required. In summary, lorazepam's efficacy as an anticonvulsant, along with its pharmacokinetic properties, makes it the go-to choice for managing status epilepticus in clinical settings.**

## 8. Which drug class does bupropion belong to?

**A. SSRI**

**B. TCA**

**C. NE and DA reuptake inhibitor**

**D. MAOI**

**Bupropion is classified as a norepinephrine and dopamine reuptake inhibitor (NDRI). This mechanism of action primarily involves the inhibition of the reuptake of norepinephrine and dopamine neurotransmitters in the brain, which helps to enhance mood and alleviate depressive symptoms. Bupropion is commonly used as an antidepressant and is also effective for smoking cessation. Understanding why bupropion does not belong to the other classes is essential. Selective serotonin reuptake inhibitors (SSRIs) focus mainly on serotonin reuptake inhibition, addressing mood disorders with a different mechanism. Tricyclic antidepressants (TCAs) have a broader spectrum of action, affecting multiple neurotransmitter systems, but their side effect profile is often less favorable. Monoamine oxidase inhibitors (MAOIs) primarily inhibit the enzyme that breaks down neurotransmitters, leading to increased levels of norepinephrine, serotonin, and dopamine, but they require dietary restrictions and have significant interactions with other medications. This distinct action of bupropion as a norepinephrine and dopamine reuptake inhibitor is what makes it a unique choice in the treatment of depression and other conditions, differentiating it from the other mentioned classes.**

## 9. What is the primary mechanism of action of atypical antipsychotics?

**A. Inhibition of dopamine and 5HT2 receptors**

**B. Boosting serotonin levels exclusively**

**C. Selective blockade of adrenergic receptors**

**D. Enhancing GABA activity**

**Atypical antipsychotics are primarily characterized by their mechanism of action, which involves the inhibition of both dopamine and serotonin (5-HT2) receptors in the brain. This dual action is key to their effectiveness in treating symptoms of schizophrenia and other mood disorders. The blockade of dopamine receptors, particularly the D2 subtype, helps to alleviate positive symptoms such as hallucinations and delusions. Meanwhile, the inhibition of serotonin receptors, specifically 5-HT2A, enhances the regulation of dopaminergic activity and plays a crucial role in improving negative symptoms and cognitive deficits often associated with these disorders. The balance in this receptor activity is what differentiates atypical antipsychotics from traditional antipsychotics, which primarily target dopamine receptors. The other options do not accurately reflect the primary mechanism. While boosting serotonin levels may seem relevant, atypical antipsychotics do not act exclusively in this manner. Selective blockade of adrenergic receptors and enhancing GABA activity are not significant or primary mechanisms of action for atypical antipsychotics either, as these actions are not central to their therapeutic effects.**

## 10. What is a primary use of diazepam?

**A. Anxiety**

**B. Sleep disorders**

**C. Preoperative sedation**

**D. All of the above**

**Diazepam is a versatile medication that belongs to the benzodiazepine class and is widely used for various medical conditions. Its primary uses include managing anxiety, providing preoperative sedation, and treating sleep disorders. As an anxiolytic, diazepam is effective in reducing symptoms of anxiety by enhancing the effect of the neurotransmitter gamma-aminobutyric acid (GABA) in the brain, leading to a calming effect. This makes it a common choice in treating anxiety disorders. In addition, diazepam is often administered as a preoperative sedative. It helps to alleviate pre-anesthetic anxiety in patients before surgical procedures, allowing them to relax and feel more comfortable prior to the surgery. Furthermore, diazepam can also be used to manage certain sleep disorders due to its sedative properties. It can help initiate and maintain sleep, especially in cases where anxiety contributes to insomnia. Given these varied applications, it is accurate to conclude that diazepam has multiple primary uses, which encompasses anxiety, sleep disorders, and preoperative sedation, affirming that "all of the above" is the correct response.**