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**FREQUENTLY  
MISSED  
CONCEPTS**

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# TRANSURETHRAL VS INTRACAVERNOUSAL ADMINISTRATION OF ALPROSTADIL.

Transurethral administration has fewer side effects than intracavernous administration of alprostadil.



Both intracavernous and transurethral administration lead to increased arterial blood flow to the penis.



The dosage of transurethral alprostadil ranges from 125 to 1000 mcg, which is higher than the dose required for intracavernous administration (5 to 40 mcg).



Erection develops 5 to 10 minutes after drug insertion and lasts 30 to 60 minutes.

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# DOXAZOSIN

Hypotension, dizziness, and nasal congestion are all adverse effects of doxazosin, an  $\alpha$ 1-adrenergic antagonist.

**Hypotension would be the most concerning because it can lead to inadequate peripheral tissue perfusion.**

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# ORAL CONTRACEPTIVES WITH CARBAMAZEPINE

Carbamazepine induces hepatic cytochrome P450 enzymes and thus accelerates the metabolism of oral contraceptives. Spotting is a sign of reduced estrogen blood levels

Patients experiencing this effect may need an increased estrogen dose rather than changing the carbamazepine.

If the dose of OC is not changed, the woman may use condoms along with the OC.

Reducing or increasing the dose of carbamazepine may lead to subtherapeutic or toxic doses.

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# TESTOSTERONE TREATMENT

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Patients receiving testosterone may experience edema secondary to sodium and water retention.

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Treatment involves discontinuing the drug and giving diuretics if needed.

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Growth of pubic hair is an expected effect.

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A rash at the site of transdermal Apply is a common effect.

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Acne is an expected effect.

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# INTRAVAGINAL ADMINISTRATION OF ESTROGENS

Estrogens for intravaginal administration are used for local effects

Primarily to treat vulval and vaginal atrophy, so there is a lower risk of systemic effects.

PRAMIPEXOL  
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A few patients taking pramipexole have experienced sleep attacks, or an overwhelming and irresistible sleepiness that comes on without warning.



Dizziness, hallucinations, and dyskinesias are listed as side effects of all dopamine agonists.

## VENLAFAXIN E

Venlafaxine can cause a variety of adverse effects.

The most common is nausea (37% to 58%), followed by headache, anorexia, nervousness, sweating, somnolence, and drowsiness. Significant weight loss may occur secondary to anorexia.

Venlafaxine can also cause dose-related sustained diastolic hypertension; blood pressure should be monitored.



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# SEVERE DEPRESSION

Patients with severe depression benefit more from a combination of drug therapy and psychotherapy than from either component alone.

Nondrug therapies should be explored.

Once a drug has been selected for treatment, it must be used for 4 to 8 weeks before its efficacy can be assessed.

Until a drug has been used at least 1 month without success, it should not be considered a failure.

Adding a second medication, changing to a different medication, and increasing the dose of this medication should all be reserved until the current drug is deemed to have failed after at least 4 weeks.

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# B AGONIST / ANTAGONISTS

While  $\beta$  agonists such as albuterol help asthma symptoms, they can worsen anxiety related to making presentations.

While  $\beta$  antagonists such as propranolol will decrease anxiety, they may worsen asthma.

Benzodiazepines (e.g., clonazepam [Klonopin], alprazolam [Xanax]) are an option for some patients. These drugs are well tolerated, and their benefits are immediate, unlike those of the SSRIs.

As a result, benzodiazepines can provide rapid relief and can be used PRN.

Accordingly, these drugs are well suited for people whose fear is limited to performance situations and who must face those situations only occasionally.

The usual dosage is 1 to 3 mg/day for clonazepam and 1 to 4 mg/day for alprazolam.

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# FIRST GENERATION ANTIPSYCHOTI CS (FGAS)

Anticholinergic effects such as dry mouth and constipation are common with first generation antipsychotics (FGAs).

Neuroendocrine effects, orthostatic hypertension, and sedation can occur with FGAs.

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# ST. JOHNS WORTS

St. John's wort has the potential to interact with many drugs through three different mechanisms.

One mechanism, induction of P450 enzymes, accelerates the metabolism of many drugs, causing loss of therapeutic effects.

St. John's wort reduces the effects of digoxin, because P-glycoprotein transports drugs out of tubular cells of the kidney and into the urine, greatly reducing digoxin levels.

St. John's wort does not counteract the beneficial effects of CNS depressants; it can actually intensify the effects of serotonin. St. John's wort is not known to increase the risk of bleeding.