

Alpha blockers

Alpha-adrenoceptor antagonists that lower vascular tone and relax prostatic and bladder-neck smooth muscle, defined clinically by first-dose hypotension, by the selective and non-selective split, and by intraoperative floppy iris syndrome.

Online guide: <https://www.medguideglobal.com/pharmacology/cardiovascular/alpha-blockers>

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Quick revision

One Liner: Alpha blockers modulate the sympathetic nervous system at alpha-adrenergic receptors: alpha-1 blockade vasodilates and relaxes prostatic smooth muscle, alpha-2 blockade removes the brake on norepinephrine release, and the first dose is the one that most often causes trouble.

1. The class falls into three categories: non-selective agents, selective alpha-1 blockers, and selective alpha-2 blockers. [1]
2. Alpha-1 receptors sit largely on vascular smooth muscle and, when activated by catecholamines, cause vasoconstriction and raise systemic arterial pressure and peripheral resistance. [1]
3. Alpha-2 receptors sit on peripheral nerve endings and inhibit norepinephrine release when activated, forming a feedback loop. [1]
4. Selective alpha-1 blockers can be recognised by the suffix -osin: alfuzosin, doxazosin, prazosin, silodosin, tamsulosin and terazosin. [1]
5. First-dose hypotension, syncope, dizziness and headache are characteristic of the selective alpha-1 agents, and reflex tachycardia may follow a sudden fall in pressure. [1]
6. Doxazosin, prazosin and terazosin are used for essential hypertension but are second-line because of orthostatic hypotension. [1]
7. Alfuzosin, tamsulosin and silodosin have relatively minimal orthostatic effects and are the first-line alpha blockers for symptomatic benign prostatic hyperplasia. [1]
8. Non-selective agents phenoxybenzamine and phentolamine are approved for phaeochromocytoma; phenoxybenzamine is irreversible and phentolamine reversible. [1]
9. Blocking alpha-2 alongside alpha-1 increases norepinephrine release, which produces the tachycardia and tremulousness seen with non-selective agents. [1]
10. Intraoperative floppy iris syndrome complicates cataract surgery and is most commonly associated with tamsulosin. [1]
11. Alpha blockade raises the spontaneous expulsion rate of ureteric stones and is now first-line for that indication, silodosin appearing the most effective. [1]
12. There is no specific antidote; management of toxicity is supportive, with positioning, fluids and vasopressors as a last resort. [1]

Overview

1. Alpha blockers act on the sympathetic nervous system through alpha-adrenergic receptors, which govern vascular tone and the release of norepinephrine. They are used in essential hypertension, benign prostatic hyperplasia and phaeochromocytoma, and the class divides into three categories: non-selective agents, selective alpha-1 blockers and selective alpha-2 blockers. [1]
2. Most alpha-1 adrenergic receptors sit on vascular smooth muscle in the skin, gastrointestinal sphincters, kidneys and brain. Activation by epinephrine or norepinephrine causes vasoconstriction, raising systemic arterial pressure and peripheral resistance; norepinephrine has the higher affinity of the two. Alpha-2 receptors sit instead on peripheral

nerve endings and inhibit norepinephrine release when activated, forming a self-limiting feedback loop. [1]

3. Selective alpha-1 antagonists therefore lower blood pressure by preventing norepinephrine from acting at the vascular receptor, and they relax smooth muscle in the prostate and bladder neck to relieve obstructive urinary symptoms. They work quickly enough that symptomatic relief in prostatic disease is often noticed within days, which is why they are the preferred initial treatment there - while for hypertension they remain second-line because of orthostatic hypotension. [1]

4. The non-selective agents behave differently because blocking alpha-2 raises norepinephrine release, partly counteracting the vasodilation and driving beta-mediated tachycardia and tremulousness. That profile makes them most useful where circulating catecholamines are already high, as in phaeochromocytoma, and unsuitable for long-term use. [1]

Classification and drug examples

Intro: Three categories, and the useful mental split is which receptor is blocked and whether the agent is intended for the vasculature or for the lower urinary tract. Within the selective alpha-1 group, orthostatic burden is what separates the hypertension-associated members from those preferred for prostatic symptoms.

1. Name: Selective alpha-1 blockers used for blood pressure

1. Note: Effective antihypertensives but second-line, because orthostatic hypotension is the dominant adverse effect. All are recognisable by the -osin suffix.

1. Name: Doxazosin

1. Route: Oral

1. Note: The immediate-release formulation can serve as a second-line antihypertensive where prostatic symptoms coexist; the extended-release formulation is approved for prostatic symptoms but not for hypertension. [3]

2. Name: Prazosin

2. Route: Oral

2. Note: Approved for hypertension but not a first-line agent; it was the first alpha antagonist used for prostatic hyperplasia and has recognised off-label roles including nightmares in post-traumatic stress disorder. [2]

3. Name: Terazosin

3. Route: Oral

3. Note: Approved first for hypertension and later for lower urinary tract symptoms of prostatic hyperplasia. First-dose syncope is rare and may be mitigated by administration at bedtime. [4] [1]

2. Name: Selective alpha-1 blockers preferred for prostatic symptoms

2. Note: Recommended first-line for symptomatic benign prostatic hyperplasia because their orthostatic adverse effects are relatively minimal.

1. Name: Tamsulosin

1. Route: Oral

1. Note: Relaxes muscle in the prostate and bladder so urine can flow more easily; the agent most commonly associated with intraoperative floppy iris syndrome. [5][1]

2. Name: Silodosin

2. Route: Oral

2. Note: Also appears the most effective member for medical expulsive therapy of ureteric calculi. [1]

3. Name: Alfuzosin

3. Route: Oral

3. Note: A further first-line option for prostatic symptoms, taken immediately after a meal. [1] [1]

3. Name: Non-selective alpha blockers

3. Note: Block alpha-1 and alpha-2 together, approved for the management of phaeochromocytoma and intended for short-term use only.

1. Name: Phenoxybenzamine

1. Route: Oral

1. Note: An irreversible alpha blocker, started well before phaeochromocytoma excision to control hypertensive crises around surgery. [1]

2. Name: Phentolamine

2. Route: IM/IV

2. Note: A reversible alpha blocker used intraoperatively, in cocaine-induced cardiovascular complications, and as a component of intracavernosal injection therapy. [1] [1]

4. Name: Selective alpha-2 blockers

4. Note: Included for completeness rather than for cardiovascular practice: they inhibit the negative feedback on norepinephrine and so stimulate the sympathetic nervous system, but the clinical significance of that in human medicine is limited.

1. Name: Yohimbine

1. Route: Oral

1. Note: Historically used for erectile dysfunction; effectiveness was never conclusively established and it is not approved for any indication. [1]

2. Name: Idazoxan

2. Route: Research use

2. Note: Used primarily in research settings, with no established clinical role. [1] [1]

Mechanism of action

Summary: Alpha blockers antagonise alpha-adrenergic receptors. Alpha-1 blockade vasodilates and relaxes prostatic and bladder-neck smooth muscle; additional alpha-2 blockade removes the feedback inhibition on norepinephrine release and so partly opposes the vasodilation.

Target: Alpha-1 adrenergic receptors on vascular, prostatic and bladder-neck smooth muscle; alpha-2 autoreceptors on peripheral nerve endings

Cidal Static: receptor antagonist

Kill Kinetics: reversible blockade, except phenoxybenzamine which is irreversible

1. Step: 1

1. Title: Catecholamines constrict vessels through alpha-1

1. Detail: Epinephrine and norepinephrine activate alpha-1 receptors on vascular smooth muscle, causing vasoconstriction that raises systemic arterial blood pressure and peripheral resistance, with norepinephrine the higher-affinity ligand. [1]

2. Step: 2

2. Title: Blocking alpha-1 produces vasodilation

2. Detail: A selective alpha-1 antagonist prevents norepinephrine from activating the receptor, so vascular smooth muscle relaxes and blood pressure falls. [1]

3. Step: 3

3. Title: The same blockade relaxes lower urinary tract smooth muscle

3. Detail: Alpha-1 blockade relaxes smooth muscle in the prostate and bladder neck, which facilitates urine flow in obstructive uropathy from prostatic enlargement. [1]

4. Step: 4

4. Title: An abrupt fall in pressure triggers the first-dose effect

4. Detail: Because the vasodilation appears rapidly, the first exposure or a reinitiation after a break can produce hypotension, syncope and dizziness, sometimes with reflex tachycardia. [1][3]

5. Step: 5

5. Title: Alpha-2 blockade adds an opposing action

5. Detail: Alpha-2 receptors on nerve endings normally inhibit norepinephrine release. Blocking them increases

release, which counteracts the alpha-1-mediated vasodilation and drives beta-mediated tachycardia and tremulousness. [1]

6. Step: 6

6. Title: That combination suits a catecholamine excess

6. Detail: Non-selective agents are most effective when sympathetic activity is high, such as with the circulating catecholamines of pheochromocytoma, which is where their approved role sits. [1]

Major clinical uses

1. Drug: Doxazosin

1. Organism: Not applicable

1. Indication: Signs and symptoms of benign prostatic hyperplasia and lower urinary tract symptoms; immediate-release formulation as second-line therapy in hypertension

1. Line: second-line

1. Note: Where combination therapy is needed for blood pressure, pairing with a diuretic is recommended for optimal effectiveness. [3]

2. Drug: Prazosin

2. Organism: Not applicable

2. Indication: Hypertension, alone or with other antihypertensive agents

2. Line: second-line

2. Note: Not a first-line agent under current evidence-based guidelines; off-label roles include nightmares associated with post-traumatic stress disorder and Raynaud phenomenon. [2]

3. Drug: Terazosin

3. Organism: Not applicable

3. Indication: Hypertension, and lower urinary tract symptoms associated with benign prostatic hyperplasia

3. Line: second-line

3. Note: Approved for blood pressure first and for prostatic symptoms several years later. [4]

4. Drug: Tamsulosin

4. Organism: Not applicable

4. Indication: Benign prostatic hyperplasia

4. Line: first-line

4. Note: Works by relaxing the muscles in the prostate and bladder so that urine can flow more easily. [5]

5. Drug: Silodosin

5. Organism: Not applicable

5. Indication: Benign prostatic hyperplasia, and medical expulsive therapy for distal ureteric stones

5. Line: first-line

5. Note: Appears the most effective member of the class for expulsive therapy; a limited course is suggested before the attempt is considered a failure. [1]

6. Drug: Phenoxybenzamine

6. Organism: Not applicable

6. Indication: Pheochromocytoma, to control hypertensive crises before and during surgery

6. Line: targeted

6. Note: Irreversible blockade, begun in advance of excision rather than at the time of operation. [1]

7. Drug: Phentolamine

7. Organism: Not applicable

7. Indication: Pheochromocytoma intraoperatively; cocaine-induced cardiovascular complications unresponsive to first-line treatment

7. Line: targeted

7. Note: Preferred over beta blockade in cocaine-associated presentations because of the risk of unopposed alpha-mediated coronary vasoconstriction. [1]

Pharmacokinetics

1. Drug: Doxazosin

1. Route: Oral

1. Absorption: Immediate-release and an extended-release form using a gastrointestinal therapeutic system

1. Csf Penetration: Not a defining feature of this agent

1. Metabolism: See a current prescribing reference

1. Elimination: See a current prescribing reference

1. Half Life: Long enough for once-daily use in the extended-release form

1. Dose Adjust: Multistep titration is used to avoid syncope, including on reinitiation [3]

2. Drug: Prazosin

2. Route: Oral

2. Absorption: Oral

2. Csf Penetration: Not a defining feature of this agent

2. Metabolism: See a current prescribing reference

2. Elimination: See a current prescribing reference

2. Half Life: Short enough that it was traditionally given several times a day

2. Dose Adjust: Initial exposure timed to night-time because of first-dose hypotension [2]

3. Drug: Terazosin

3. Route: Oral

3. Absorption: Oral

3. Csf Penetration: Not a defining feature of this agent

3. Metabolism: See a current prescribing reference

3. Elimination: See a current prescribing reference

3. Half Life: Long enough for once-daily administration

3. Dose Adjust: Bedtime administration mitigates the rare first-dose syncope [4]

4. Drug: Tamsulosin

4. Route: Oral

4. Absorption: Oral

4. Csf Penetration: Not a defining feature of this agent

4. Metabolism: See a current prescribing reference

4. Elimination: See a current prescribing reference

4. Half Life: See a current prescribing reference

4. Dose Adjust: See a current prescribing reference [5]

1. Selective alpha-1 blockers are oral medicines usually taken at night to reduce the risk of orthostatic hypotension, an effect most pronounced with doxazosin, prazosin and terazosin. [1]

2. Symptomatic relief in prostatic disease often appears within a few days of starting therapy, which is part of why these agents are preferred for initial treatment there. [1]

3. Extended-release formulations of doxazosin give more stable concentrations across the day and a lower incidence of orthostatic hypotension and syncope than the immediate-release form. [3]

4. This page gives no dose regimens by design. Doses depend on the indication, the formulation, age, comorbidity and concurrent antihypertensive therapy, and belong in a prescribing reference used by the treating clinician.

Adverse effects

1. Effect: Dizziness, fatigue, headache and weakness

1. Detail: The everyday tolerability problems reported with selective alpha-1 blockade. [3]
2. Effect: Orthostatic hypotension
2. Detail: Commoner in older patients, in whom it raises the risk of falls; it is also the reason these agents are second-line for hypertension. [1]
3. Effect: Reflex tachycardia
3. Detail: May follow a sudden drop in blood pressure after alpha-1 blockade. [1]
1. Effect: First-dose hypotension and syncope
1. Detail: Characteristic of the selective alpha-1 agents and most pronounced after the initial exposure or on reinitiation of therapy; risk rises when combined with another antihypertensive, a nitrate or a phosphodiesterase type 5 inhibitor.
1. Action: Night-time administration and a multistep titration are the standard mitigations. [1][3]
2. Effect: Intraoperative floppy iris syndrome
2. Detail: A cataract surgery complication with iris billowing, prolapse through surgical incisions and progressive pupillary constriction despite mydriatic therapy; long-term use may cause permanent iris dilator muscle damage.
2. Action: Alpha blocker exposure, current or past, should be known to the ophthalmic surgeon before cataract surgery. [1]
3. Effect: Severe hypotension in toxicity
3. Detail: The commonest toxic effect, which when severe can cause ischaemic damage to major organs and increase the risk of falls.
3. Action: Supportive management only; no specific antidote exists. [1]
1. Drug: Non-selective agents (phenoxybenzamine, phentolamine)
1. Effect: Hypotension, weakness, tachycardia and tremulousness, the last two arising from norepinephrine spillover after alpha-2 blockade. [1]
2. Drug: Tamsulosin
2. Effect: The alpha blocker most commonly implicated in intraoperative floppy iris syndrome. [1]
3. Drug: Alpha blockers used for premature ejaculation
3. Effect: May decrease semen volume and inhibit seminal emission. [1]

Contraindications, precautions and interactions

1. Known hypersensitivity to the agent or any component of its formulation. [1]
2. Phenoxybenzamine and phentolamine in a breastfeeding mother. [1]
1. Existing hypotension or a history of orthostatic hypotension, where further vasodilation is poorly tolerated. [1]
2. Older patients, in whom orthostatic effects and the risk of complications during cataract surgery are both greater. [1]
3. For the non-selective agents, marked renal impairment, cerebrovascular disease, coronary artery disease or a current respiratory infection. [1]
4. Residence in a care setting, where the fall risk from orthostatic hypotension warrants specific precautions. [1]
1. With: Other antihypertensive agents
1. Effect: Additive blood pressure lowering, increasing the risk of orthostatic hypotension and syncope. [3]
2. With: Nitrates
2. Effect: Additive vasodilation and a greater risk of orthostatic hypotension or syncope. [3]
3. With: Phosphodiesterase type 5 inhibitors
3. Effect: Additive hypotensive effect, particularly around initiation of the alpha blocker. [3]
4. With: 5-aminolevulinic acid in photodynamic-enhanced cystoscopy
4. Effect: An increased risk of significant hypotension in bladder cancer patients. [1]

Comparison tables

Title: Selectivity, principal role and orthostatic burden

Caption: The practical question is which receptor is blocked and how much orthostatic hypotension the agent brings.

Actual therapy is governed by a current prescribing reference.

1. Key: drug

1. Label: Drug

1. Is Row Header: true

2. Key: selectivity

2. Label: Selectivity

3. Key: role

3. Label: Principal role

4. Key: orthostatic

4. Label: Orthostatic burden

1. Drug: Doxazosin

1. Selectivity: Selective alpha-1

1. Role: Prostatic symptoms; second-line for blood pressure

1. Orthostatic: Pronounced [1][3]

2. Drug: Prazosin

2. Selectivity: Selective alpha-1

2. Role: Hypertension, not first-line; off-label roles including PTSD nightmares

2. Orthostatic: Pronounced [1][2]

3. Drug: Terazosin

3. Selectivity: Selective alpha-1

3. Role: Hypertension and prostatic symptoms

3. Orthostatic: Pronounced [1][4]

4. Drug: Tamsulosin

4. Selectivity: Selective alpha-1

4. Role: Benign prostatic hyperplasia, first-line

4. Orthostatic: Relatively minimal [1][5]

5. Drug: Silodosin

5. Selectivity: Selective alpha-1

5. Role: Prostatic symptoms and stone expulsion

5. Orthostatic: Relatively minimal [1]

6. Drug: Alfuzosin

6. Selectivity: Selective alpha-1

6. Role: Benign prostatic hyperplasia, first-line

6. Orthostatic: Relatively minimal [1]

7. Drug: Phenoxybenzamine

7. Selectivity: Non-selective, irreversible

7. Role: Pheochromocytoma, before and during surgery

7. Orthostatic: Hypotension plus tachycardia and tremulousness [1]

8. Drug: Phentolamine

8. Selectivity: Non-selective, reversible

8. Role: Pheochromocytoma intraoperatively; cocaine-associated presentations

8. Orthostatic: Hypotension plus tachycardia and tremulousness [1]

High-yield exam pearls

1. Pearl: The first dose is the dangerous one.

1. Why: Selective alpha-1 blockers cause first-dose hypotension, syncope, dizziness and headache through vasodilation and vascular smooth muscle relaxation, which is why administration at night is recommended and why

titration schedules exist. [1][3]

2. Pearl: Alpha-2 blockade explains why non-selective agents cause tachycardia.

2. Why: Antagonising the alpha-2 autoreceptor removes the negative feedback on norepinephrine release; the resulting norepinephrine spillover stimulates beta receptors, producing tremulousness and tachycardia that selective alpha-1 agents largely avoid. [1]

3. Pearl: The same receptor serves two very different indications.

3. Why: Alpha-1 blockade relaxes vascular smooth muscle to lower blood pressure and relaxes prostatic and bladder-neck smooth muscle to improve urine flow, which is why one class covers both hypertension and benign prostatic hyperplasia. [1]

4. Pearl: Not all -osin drugs are used the same way.

4. Why: Doxazosin, prazosin and terazosin are the hypertension-associated members, while alfuzosin, tamsulosin and silodosin are preferred for prostatic symptoms because their orthostatic effects are relatively minimal. [1]

5. Pearl: Phenoxybenzamine is irreversible, phentolamine is not.

5. Why: That difference is why phenoxybenzamine is started well before phaeochromocytoma surgery while phentolamine is used intraoperatively and in short-lived catecholamine crises. [1]

6. Pearl: Ask about cataract surgery before starting an alpha blocker.

6. Why: Long-term use can cause dysfunction or atrophy of the iris dilator muscle, producing intraoperative floppy iris syndrome with iris billowing, prolapse through incisions and progressive pupillary constriction despite mydriatics. [1]

7. Pearl: Beta blockade alone is the wrong answer in a catecholamine surge.

7. Why: In cocaine-associated presentations, beta blockade is less favourable because of unopposed alpha-mediated coronary vasoconstriction and hypertension, and phentolamine is the agent recommended when first-line treatments fail. [1]

Common exam traps

1. Trap: "Alpha blockers are first-line antihypertensives."

1. Correction: Doxazosin, prazosin and terazosin are considered second-line for essential hypertension because of adverse effects, particularly orthostatic hypotension. [1]

2. Trap: "Any alpha blocker will do for benign prostatic hyperplasia."

2. Correction: Alfuzosin, tamsulosin and silodosin are the recommended first-line agents for that indication; the more orthostatically active members are not preferred despite being effective. [1]

3. Trap: "Floppy iris syndrome only matters with current use."

3. Correction: Long-term administration may cause dysfunction or atrophy of the iris dilator muscle with potentially permanent damage, so a past exposure still matters to the ophthalmic surgeon. [1]

4. Trap: "Selective alpha-1 blockade avoids all cardiovascular adverse effects."

4. Correction: Systemic effects such as tachycardia and tremulousness are less common, but first-dose hypotension, syncope, dizziness, headache and reflex tachycardia remain characteristic. [1]

5. Trap: "Non-selective alpha blockers are long-term antihypertensives."

5. Correction: Phenoxybenzamine and phentolamine are intended for short-term use and are approved for the management of phaeochromocytoma rather than routine blood pressure control. [1]

6. Trap: "Prazosin's only role is blood pressure."

6. Correction: It is approved for hypertension but is not a first-line agent, and it also has recognised off-label roles including post-traumatic stress disorder-associated nightmares and Raynaud phenomenon. [2]

Self-test questions

1. Question: Why do non-selective alpha blockers cause tachycardia and tremulousness more than selective alpha-1 agents do?

1. They stimulate cardiac beta-1 receptors directly

2. They block alpha-2 autoreceptors, increasing norepinephrine release, which then stimulates beta receptors
3. They are metabolised into catecholamines in the liver
4. They prevent vagal tone from reaching the sinoatrial node
1. Explanation: Alpha-2 receptors on peripheral nerve endings normally inhibit norepinephrine release. Blocking them removes that feedback, and the resulting norepinephrine spillover stimulates beta receptors, producing tremulousness and tachycardia. [1]
2. Question: Which set of alpha blockers is recommended first-line for symptomatic benign prostatic hyperplasia?
1. Doxazosin, prazosin and terazosin
2. Phenoxybenzamine and phentolamine
3. Alfuzosin, tamsulosin and silodosin
4. Yohimbine and idazoxan
2. Explanation: Alfuzosin, tamsulosin and silodosin have relatively minimal orthostatic adverse effects and are considered first-line for this condition. Doxazosin, prazosin and terazosin are effective but are not preferred because of their adverse effect profile. [1]
3. Question: What is the characteristic adverse effect that shapes how selective alpha-1 blockers are started?
1. First-dose hypotension with syncope and dizziness
2. Immediate bronchospasm
3. Acute hyperkalaemia
4. Sudden hearing loss
3. Explanation: Selective alpha-1 blockers cause first-dose hypotension, syncope, dizziness and headache through vasodilation and vascular smooth muscle relaxation. These effects are commoner in older patients and increase the risk of falls, which is why night-time administration is recommended. [1]
4. Question: Which intraoperative complication is most strongly associated with tamsulosin?
1. Malignant hyperthermia
2. Intraoperative floppy iris syndrome
3. Postoperative ileus
4. Transient cortical blindness
4. Explanation: Intraoperative floppy iris syndrome can occur during cataract surgery and, while associated with any selective alpha blocker, is most commonly linked to tamsulosin. Features include iris billowing, prolapse through surgical incisions and progressive pupillary constriction despite mydriatic therapy. [1]
5. Question: How do phenoxybenzamine and phentolamine differ?
1. Phenoxybenzamine is a reversible blocker and phentolamine is irreversible
2. Phenoxybenzamine is irreversible and phentolamine is reversible
3. Both are selective alpha-1 blockers used only for prostatic symptoms
4. Neither has any role in pheochromocytoma
5. Explanation: Both are non-selective agents approved for managing pheochromocytoma, but phenoxybenzamine is an irreversible alpha blocker while phentolamine is reversible. Phentolamine is also used for cocaine-induced cardiovascular complications, where beta blockade risks unopposed alpha-mediated vasoconstriction. [1]
6. Question: Why does one receptor class serve both hypertension and prostatic symptoms?
1. Because prostatic tissue produces renin
2. Because alpha-1 blockade relaxes vascular smooth muscle and also relaxes smooth muscle in the prostate and bladder neck
3. Because the drugs are excreted through the prostate
4. Because blood pressure control shrinks the prostate directly
6. Explanation: Selective alpha-1 antagonists lower blood pressure by preventing norepinephrine from activating alpha-1 receptors on vascular smooth muscle, and they relieve obstructive urinary symptoms by relaxing smooth muscle in the prostate and bladder neck to facilitate urine flow. [1]

7. Question: What is the recognised approach to significant alpha-blocker toxicity?

1. Administration of a specific competitive antidote
2. Immediate haemodialysis in all cases
3. Supportive care: supine positioning, fluid resuscitation if hypotension persists, and vasopressors as a last resort
4. Rapid intravenous beta agonist infusion

7. Explanation: No specific antidote is available. The hypotensive patient is positioned supine until blood pressure and heart rate stabilise, fluid resuscitation follows if hypotension persists, and vasopressors are reserved as a last resort.

[1]

Frequently asked questions

1. Question: What is the first-dose effect?

1. Answer: It is the hypotension, syncope, dizziness and headache that selective alpha-1 blockers can cause through vasodilation, most prominently at the very start of therapy or when therapy is restarted after a gap. Night-time administration and stepwise titration are the standard ways of reducing it. [1][3]

2. Question: Why are alpha blockers not first-line for high blood pressure?

2. Answer: Doxazosin, prazosin and terazosin lower blood pressure effectively but are considered second-line agents because of adverse effects, particularly orthostatic hypotension. [1]

3. Question: Why do these drugs help prostate symptoms?

3. Answer: Alpha-1 blockade relaxes smooth muscle in the prostate and bladder neck, which facilitates urine flow where an enlarged prostate is causing obstruction. Relief is often noticed within a few days. [1]

4. Question: Which alpha blockers are used in pheochromocytoma?

4. Answer: The non-selective agents phenoxybenzamine and phentolamine. Phenoxybenzamine is irreversible and is started ahead of surgery, while phentolamine is reversible and used to control blood pressure before and during the operation. [1]

5. Question: What should an eye surgeon know before cataract surgery?

5. Answer: Whether the patient has taken an alpha blocker, because long-term use can produce dysfunction or atrophy of the iris dilator muscle and lead to intraoperative floppy iris syndrome - most commonly with tamsulosin. [1]

6. Question: Is routine monitoring needed on these drugs?

6. Answer: Blood pressure and heart rate need close monitoring when phentolamine is given intraoperatively. For the other alpha-adrenergic antagonists, routine monitoring or specific tests are not currently recommended. [1]

References

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