

Beta blockers

Beta-adrenoceptor antagonists that slow the heart and reduce contractility, separated from one another by receptor selectivity, added vasodilator or intrinsic sympathomimetic properties, and by the rebound risk that follows abrupt withdrawal.

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

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Quick revision

One Liner: Beta blockers antagonise beta-adrenoceptors to slow rate, reduce contractility and lower blood pressure - and almost every examinable distinction inside the class comes down to which receptors an individual agent blocks, what extra property it carries, and what happens when it is stopped suddenly.

1. Beta-1 receptors sit chiefly in the heart and mediate cardiac activity; beta-2 receptors are distributed across many organ systems and mediate smooth muscle relaxation. [1]
2. Blockade slows heart rate and reduces contractility, and lowers blood pressure partly through decreased renin release and reduced cardiac output. [1]
3. Non-selective agents include propranolol, carvedilol, sotalol and labetalol; beta-1 selective (cardioselective) agents include atenolol, bisoprolol, metoprolol and esmolol. [1]
4. Carvedilol and labetalol add alpha-1 receptor blockade, which gives a more pronounced effect on blood pressure than beta blockade alone. [1]
5. Nebivolol instead gains its vasodilator effect from nitric oxide synthase stimulation via beta-3 agonism rather than from alpha blockade. [5]
6. Cardioselectivity is relative, not absolute: nebivolol is beta-1 selective at lower doses and in fast metabolisers, and loses that selectivity at higher doses and in slow metabolisers. [5]
7. Pindolol is a non-selective agent that possesses intrinsic sympathomimetic activity within its therapeutic range. [6]
8. Acebutolol is cardioselective and possesses mild intrinsic sympathomimetic activity, so selectivity and intrinsic activity are two independent axes. [7]
9. Abrupt cessation can provoke a withdrawal syndrome with rebound sympathetic activation, tachycardia, hypertension, angina or myocardial infarction. [3]
10. Bradycardia and hypotension are the commonest adverse effects, with fatigue, dizziness, nausea and constipation also widely reported. [1]
11. Non-selective agents must not be used in asthma. A cardioselective agent may be used where the patient does not respond to or cannot tolerate other treatment, at the lowest dose that works and with a bronchodilator available - because beta-1 selectivity is not absolute. [1][2]
12. Sotalol additionally blocks cardiac potassium channels, prolongs the QT interval and raises the risk of torsades de pointes. [1]
13. In compensated heart failure the specifically chosen agents are bisoprolol, carvedilol and metoprolol succinate. [1]
14. Glucagon is the antidote in beta-blocker overdose, with cardiac pacing as the second-line option if glucagon fails. [1]

Overview

1. Beta blockers are a large and internally varied family of cardiovascular medicines united by one action: antagonism at beta-adrenoceptors. Catecholamines binding beta-1 receptors increase cardiac automaticity and conduction velocity and promote renin release; binding beta-2 receptors relaxes smooth muscle and drives metabolic effects such as glycogenolysis. Blocking those receptors inhibits the corresponding effects, so heart rate falls, contractility falls and

blood pressure falls through a combination of reduced cardiac output and reduced renin. [1]

2. The class holds regulatory approval across a wide span of indications including tachycardia, hypertension, myocardial infarction, congestive heart failure, cardiac arrhythmias, coronary artery disease, hyperthyroidism, essential tremor, aortic dissection, portal hypertension, glaucoma and migraine prophylaxis. Because the receptors are distributed throughout the body, that breadth of usefulness and the breadth of adverse effects come from the same source. [1]

3. What separates one agent from another is which receptors it engages and what else it does. Non-selective agents antagonise beta-1 and beta-2 alike; cardioselective agents are directed at beta-1. Some agents add alpha-1 blockade, one adds nitric-oxide-mediated vasodilation, some possess intrinsic sympathomimetic activity, and one blocks potassium channels as well. Learning the class means learning those axes rather than memorising a list. [1][5][6]

4. The final defining feature is what happens on stopping. Beta blockade upregulates the receptor population it suppresses, so sudden withdrawal releases a rebound sympathetic surge. Increased heart rate, elevated blood pressure and a heightened risk of adverse cardiovascular events follow, most dangerously where coronary artery disease or heart failure is present, and a gradual dose reduction rather than an abrupt stop is the mitigation. [3][4]

Classification and drug examples

Intro: The clinically useful division is by receptor selectivity first, then by any additional property the molecule carries - alpha blockade, nitric-oxide-mediated vasodilation, intrinsic sympathomimetic activity or potassium channel blockade. Those extra properties, not the beta blockade itself, are what usually decide between agents.

1. Name: Beta-1 selective (cardioselective) agents

1. Note: Directed at the beta-1 receptors that predominate in the heart, sparing beta-2 receptors in the lungs and smooth muscle at usual doses. This is the group recommendations now permit in asthma, where non-selective agents are not.

1. Name: Metoprolol

1. Metoprolol tartrate

2. Metoprolol succinate

1. Route: Oral/IV

1. Note: Beta-1 selective with neither membrane stabilising nor intrinsic sympathomimetic activity. The succinate salt is the form indicated in heart failure; the tartrate salt is not. [3][1]

2. Name: Atenolol

2. Route: Oral

2. Note: A beta-1 selective agent of the same group. [1]

3. Name: Bisoprolol

3. Route: Oral

3. Note: Beta-1 selective and one of the three agents specifically chosen in compensated heart failure. [1]

4. Name: Esmolol

4. Route: IV

4. Note: Short onset and short half-life make it titratable, so it is used in intensive care and cardiac inpatient settings for refractory tachycardia. [1]

5. Name: Acebutolol

5. Route: Oral

5. Note: Cardioselective and additionally possesses mild intrinsic sympathomimetic activity within its therapeutic range. [7] [1]

2. Name: Non-selective agents

2. Note: Antagonise beta-1 and beta-2 receptors together, which brings bronchospasm and metabolic effects into the risk profile. These agents should not be used in asthma.

1. Name: Propranolol

1. Route: Oral/IV

1. Note: Lipophilic and centrally penetrant; used well beyond cardiology, including for the peripheral symptoms of anxiety such as tachycardia, sweating and tension. [1][4]

2. Name: Timolol

2. Route: Ophthalmic/oral

2. Note: Used topically in glaucoma, but systemic absorption from the eye is sufficient to add to atrioventricular nodal suppression from other rate-limiting drugs. [1][8]

3. Name: Pindolol

3. Route: Oral

3. Note: A non-selective agent that possesses intrinsic sympathomimetic activity in therapeutic dosage ranges but not quinidine-like membrane stabilising activity. [6] [1]

3. Name: Agents with additional alpha-1 blockade

3. Note: Non-selective beta blockade combined with alpha-1 receptor antagonism. Blocking alpha-1 produces vasodilation and a more pronounced effect on blood pressure than beta blockade alone.

1. Name: Carvedilol

1. Route: Oral

1. Note: Combines non-selective beta blockade with alpha-1 blockade; one of the three agents chosen in compensated heart failure, and reported to increase oedema in some patients. [1]

2. Name: Labetalol

2. Route: Oral/IV

2. Note: Non-selective beta blockade with additional alpha-1 blocking activity. [1] [1]

4. Name: Vasodilating agent acting through nitric oxide

4. Note: A cardioselective agent whose vasodilator effect comes from the endothelium rather than from alpha blockade - a genuinely different route to the same haemodynamic goal.

Name: Nebivolol

Route: Oral

Note: Beta-1 antagonist that stimulates endothelial nitric oxide synthase via beta-3 agonism, reducing systemic vascular resistance. Selectivity is dose- and metaboliser-dependent. [5] [5]

5. Name: Agent with additional potassium channel blockade

5. Note: A beta blocker that is simultaneously a class III antiarrhythmic, which changes both its usefulness and its monitoring.

Name: Sotalol

Route: Oral/IV

Note: Non-selective beta blockade plus cardiac potassium channel blockade; prolongs the QT interval and raises torsades de pointes risk, so the QTc interval is monitored. [1] [1]

Mechanism of action

Summary: Beta blockers antagonise catecholamine binding at beta-adrenoceptors, reducing heart rate, contractility, renin release and myocardial oxygen demand, with the pattern of effect determined by which receptor subtypes an individual agent engages.

Target: Beta-1 and, for non-selective agents, beta-2 adrenoceptors

Cidal Static: receptor antagonist

Kill Kinetics: competitive receptor blockade

1. Step: 1

1. Title: Catecholamines normally drive the beta receptor

1. Detail: Epinephrine and norepinephrine bind beta-1 receptors to increase cardiac automaticity and conduction velocity and to induce renin release, which raises blood pressure; beta-2 binding relaxes smooth muscle and increases

metabolic activity such as glycogenolysis. [1]

2. Step: 2

2. Title: The drug occupies the receptor and inhibits those effects

2. Detail: Once bound, the antagonist prevents catecholamine signalling at that receptor, so the chronotropic and inotropic effects on the heart are inhibited and heart rate slows. [1]

3. Step: 3

3. Title: Blood pressure falls through more than one route

3. Detail: Reduced renin release and reduced cardiac output both contribute, which is why the antihypertensive effect is not simply a consequence of the slower rate. [1]

4. Step: 4

4. Title: Reduced workload relieves ischaemia

4. Detail: Negative chronotropic and inotropic effects lower myocardial oxygen demand, and that fall in demand is how angina improves. [1]

5. Step: 5

5. Title: Atrial refractory periods lengthen

5. Detail: The class prolongs atrial refractory periods and so has a potent antiarrhythmic effect in its own right. [1]

6. Step: 6

6. Title: Individual agents add a second mechanism

6. Detail: Carvedilol and labetalol add alpha-1 blockade, nebivolol stimulates endothelial nitric oxide synthase through beta-3 agonism, and sotalol blocks potassium channels - each of which changes the clinical profile without changing the beta blockade. [1][5]

7. Step: 7

7. Title: Chronic blockade sensitises the system to withdrawal

7. Detail: Sustained blockade is followed on sudden cessation by rebound sympathetic activation, with increased heart rate, elevated blood pressure and heightened cardiovascular risk - a pharmacological consequence of the mechanism rather than a separate adverse reaction. [3]

Major clinical uses

1. Drug: Beta blockers as a class

1. Organism: Not applicable

1. Indication: Hypertension, tachycardia, coronary artery disease and cardiac arrhythmias

1. Line: first-line or add-on depending on indication

1. Note: Regulatory approval spans a wide set of cardiovascular indications; the choice of agent within the class is what individualises therapy. [1]

2. Drug: Bisoprolol

2. Organism: Not applicable

2. Indication: Compensated congestive heart failure

2. Line: targeted

2. Note: One of the three agents specifically chosen for heart failure, alongside carvedilol and metoprolol succinate, and only in a compensated state. [1]

3. Drug: Metoprolol

3. Organism: Not applicable

3. Indication: Hypertension, angina, arrhythmias and, as the succinate salt, heart failure

3. Line: first-line or add-on depending on indication

3. Note: The tartrate salt is used for other conditions such as atrial fibrillation and is not indicated for heart failure. [1][3]

4. Drug: Esmolol

4. Organism: Not applicable

4. Indication: Refractory tachycardia in critical care, including atrial fibrillation and electrical storm
4. Line: targeted
4. Note: Chosen for its short onset and short half-life, which make it titratable in an inpatient setting. [1]
5. Drug: Sotalol
5. Organism: Not applicable
5. Indication: Arrhythmia management where class III antiarrhythmic action is wanted
5. Line: targeted
5. Note: QTc interval monitoring is part of using this agent because of its potassium channel blockade. [1]
6. Drug: Timolol
6. Organism: Not applicable
6. Indication: Glaucoma, by the topical ophthalmic route
6. Line: targeted
6. Note: An example of route following disease type rather than acuity; systemic absorption still occurs. [1][8]
7. Drug: Propranolol
7. Organism: Not applicable
7. Indication: Essential tremor, migraine prophylaxis, hyperthyroidism and the peripheral symptoms of anxiety
7. Line: targeted
7. Note: Not approved for anxiety disorders, though it reduces peripheral features such as tachycardia, sweating and general tension. [1]
8. Drug: Nebivolol
8. Organism: Not applicable
8. Indication: Hypertension
8. Line: targeted
8. Note: Its nitric-oxide-mediated vasodilation is the reason it is considered where endothelial function is abnormal. [5]

Pharmacokinetics

1. Drug: Metoprolol
1. Route: Oral/IV
1. Absorption: Rapid and complete from the gastrointestinal tract; bioavailability around half, from extensive first-pass metabolism
1. Csf Penetration: Crosses the blood-brain barrier
1. Metabolism: Hepatic, extensively by CYP2D6
1. Elimination: Urinary, accounting for most of an oral dose
1. Half Life: Short, and varies with formulation, enantiomer and CYP2D6 metaboliser status
1. Dose Adjust: CYP2D6 poor metabolisers accumulate higher plasma levels [3]
2. Drug: Propranolol
2. Route: Oral/IV
2. Absorption: Oral, with high plasma protein binding
2. Csf Penetration: Centrally penetrant, contributing to central nervous system effects
2. Metabolism: Hepatic, chiefly CYP2D6, CYP1A2 and CYP2C19
2. Elimination: Renal excretion of metabolites
2. Half Life: Short, so immediate-release dosing is divided through the day
2. Dose Adjust: Hepatic or renal impairment; enzyme inhibitors and inducers alter exposure [1][4]
3. Drug: Nebivolol
3. Route: Oral
3. Absorption: Peak plasma concentration within a few hours; food does not modify its pharmacokinetics
3. Csf Penetration: Not a defining feature of this agent

- 3. Metabolism: Hepatic, with metaboliser status affecting selectivity
- 3. Elimination: See a current prescribing reference
- 3. Half Life: See a current prescribing reference
- 3. Dose Adjust: Slower metabolisers lose beta-1 selectivity [5]
- 4. Drug: Esmolol
- 4. Route: IV
- 4. Absorption: Given intravenously, so absorption does not apply
- 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: Short, which is the property that makes it titratable
- 4. Dose Adjust: Titrated to effect in a monitored setting [1]
- 1. Route follows the clinical situation: parenteral administration in arrhythmias, topical administration in glaucoma, and oral administration for chronic disease. [1]
- 2. Longer-acting formulations such as metoprolol succinate support once-daily administration, while shorter-acting agents are given more frequently; extended-release formulations also delay peak toxicity in overdose. [1]
- 3. Lipophilicity matters for tolerability rather than efficacy: agents that cross the blood-brain barrier are more associated with insomnia, sleep changes and nightmares. [1]
- 4. This page gives no dose regimens by design. Doses depend on indication, formulation, comorbidity, renal and hepatic function and metaboliser status, and belong in a prescribing reference used by the treating clinician.

Adverse effects

- 1. Effect: Bradycardia and hypotension
 - 1. Detail: The two effects that follow most directly from the mechanism and the two most commonly encountered. [1]
- 2. Effect: Fatigue, dizziness, nausea and constipation
 - 2. Detail: Widely reported and often the reason a patient finds the class difficult to continue. [1]
- 3. Effect: Sexual dysfunction
 - 3. Detail: Reported by some patients, including erectile dysfunction. [1]
- 4. Effect: Insomnia, altered sleep and nightmares
 - 4. Detail: More pronounced with agents that cross the blood-brain barrier; the class also lowers melatonin secretion. [1]
- 5. Effect: Fatigue or weight gain
 - 5. Detail: Some agents are more likely than others to produce these, and management involves reconsidering the medication. [1]
- 1. Effect: Bronchospasm
 - 1. Detail: Less common overall but a defining hazard, with asthmatic patients at higher risk.
 - 1. Action: Non-selective agents are avoided in asthma; a cardioselective agent is preferred where beta blockade is clinically necessary. [1][4]
- 2. Effect: Heart block
 - 2. Detail: All beta blockers carry this risk, especially in patients with cardiac risk factors.
 - 2. Action: Heart rate and blood pressure are monitored during therapy. [1]
- 3. Effect: Withdrawal syndrome on abrupt cessation
 - 3. Detail: Rebound sympathetic activation with tachycardia and hypertension, and a risk of angina or myocardial infarction. Metoprolol carries a boxed warning to this effect.
 - 3. Action: A structured, gradual dose reduction is advocated when the drug is being stopped. [3]
- 4. Effect: Masked hypoglycaemia
 - 4. Detail: The class can induce hyperglycaemia and blunt the adrenergic signs, such as tachycardia, that normally warn of a falling glucose.

4. Action: Extra caution where insulin or oral hypoglycaemic agents are also being used. [1][4]
5. Effect: Reduced response to epinephrine in anaphylaxis
5. Detail: Patients on beta blockers may show increased reactivity to allergens and reduced responsiveness to standard epinephrine doses.
5. Action: Glucagon is endorsed for beta-blocker-associated anaphylaxis resistant to epinephrine. [4]
1. Drug: Sotalol
1. Effect: QT interval prolongation and an increased risk of torsades de pointes, from potassium channel blockade. [1]
2. Drug: Carvedilol
2. Effect: May increase oedema in some patients. [1]
3. Drug: Propranolol and other non-selective agents
3. Effect: Antagonise the bronchodilator effect of beta-2 agonists and may precipitate bronchospasm in asthma or chronic obstructive pulmonary disease. [4]
4. Drug: Metoprolol
4. Effect: Abrupt cessation can produce a withdrawal syndrome causing angina or myocardial infarction, with tachycardia and hypertension both common. [3]

Contraindications, precautions and interactions

1. Non-selective agents in asthma, because of bronchospasm risk. [1]
2. Untreated phaeochromocytoma, where unopposed alpha-adrenergic stimulation may produce severe hypertension. [3]
3. Acute decompensated heart failure or cardiogenic shock. [4]
4. Pre-existing bradycardia or atrioventricular block, for agents that further slow conduction. [4]
5. Sotalol in a patient with long QT syndrome or previous torsades de pointes. [1]
1. Acute or chronic bradycardia and hypotension are relative contraindications across the class. [1]
2. Raynaud phenomenon, which beta blockade can exacerbate. [1]
3. Diabetes treated with insulin or oral hypoglycaemic agents, because adrenergic warning signs of hypoglycaemia are masked. [4]
4. Hepatic or renal impairment, where clearance of individual agents may be reduced. [4]
5. A history of poor adherence, because unplanned interruption reproduces the withdrawal syndrome. [3]
6. Cocaine toxicity, where beta blockade without concurrent alpha blockade can leave alpha stimulation unopposed. [4]
1. With: Verapamil and diltiazem
1. Effect: Additive depression of atrioventricular nodal conduction and contractility, increasing the likelihood of bradycardia, heart block or acute decompensated heart failure. Even ophthalmic timolol can contribute through systemic absorption. [8]
2. With: Beta-2 agonists such as salbutamol
2. Effect: Non-selective agents antagonise the bronchodilator effect and may precipitate bronchospasm. [4]
3. With: CYP2D6 inhibitors such as fluoxetine, paroxetine or quinidine
3. Effect: Raise plasma concentrations of agents cleared by that enzyme, predisposing to bradycardia, hypotension or conduction disturbance. [4]
4. With: Enzyme inducers such as rifampicin or carbamazepine
4. Effect: Reduce bioavailability and may diminish therapeutic effect. [4]
5. With: Clonidine
5. Effect: Abrupt withdrawal of clonidine in a patient taking a beta blocker may precipitate rebound hypertension through unopposed sympathetic activity. [4]
6. With: Non-steroidal anti-inflammatory drugs
6. Effect: May attenuate the antihypertensive effect by promoting sodium and water retention. [4]
7. With: Digoxin and antiarrhythmic agents

7. Effect: Additive negative chronotropic and inotropic effects, including bradycardia and atrioventricular block. [4]

Comparison tables

Title: Selectivity and additional properties across the class

Caption: Selectivity and intrinsic sympathomimetic activity vary independently, and the extra property in the final column is usually what decides between agents. 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: Receptor selectivity

3. Key: isa

3. Label: Intrinsic sympathomimetic activity

4. Key: extra

4. Label: Additional property

1. Drug: Metoprolol

1. Selectivity: Beta-1 selective

1. Isa: None

1. Extra: No membrane stabilising activity either [3][1]

2. Drug: Atenolol

2. Selectivity: Beta-1 selective

2. Isa: Not a feature of this agent

2. Extra: - [1]

3. Drug: Bisoprolol

3. Selectivity: Beta-1 selective

3. Isa: Not a feature of this agent

3. Extra: One of the heart failure agents [1]

4. Drug: Esmolol

4. Selectivity: Beta-1 selective

4. Isa: Not a feature of this agent

4. Extra: Short onset and short half-life [1]

5. Drug: Acebutolol

5. Selectivity: Cardioselective

5. Isa: Mild, within the therapeutic range

5. Extra: - [7]

6. Drug: Propranolol

6. Selectivity: Non-selective

6. Isa: Not a feature of this agent

6. Extra: Lipophilic and centrally penetrant [1][4]

7. Drug: Pindolol

7. Selectivity: Non-selective

7. Isa: Present in therapeutic dosage ranges

7. Extra: No quinidine-like membrane stabilising activity [6]

8. Drug: Carvedilol

8. Selectivity: Non-selective

8. Isa: Not a feature of this agent

8. Extra: Additional alpha-1 blockade [1]

- 9. Drug: Labetalol
- 9. Selectivity: Non-selective
- 9. Isa: Not a feature of this agent
- 9. Extra: Additional alpha-1 blockade [1]
- 10. Drug: Nebivolol
- 10. Selectivity: Beta-1 selective at lower doses and in fast metabolisers
- 10. Isa: Not a feature of this agent
- 10. Extra: Nitric-oxide-mediated vasodilation via beta-3 agonism [5]
- 11. Drug: Sotalol
- 11. Selectivity: Non-selective
- 11. Isa: Not a feature of this agent
- 11. Extra: Potassium channel blockade; class III antiarrhythmic [1]

High-yield exam pearls

- 1. Pearl: Selectivity and intrinsic sympathomimetic activity are separate questions.
- 1. Why: Pindolol is non-selective and has intrinsic sympathomimetic activity, while acebutolol is cardioselective and also has mild intrinsic sympathomimetic activity - so knowing an agent is cardioselective says nothing about whether it has intrinsic activity. [6][7]
- 2. Pearl: Metoprolol is the clean comparator on both axes.
- 2. Why: It is beta-1 selective and exhibits neither membrane stabilising nor intrinsic sympathomimetic activity, which is why it is so often used as the reference agent when the class is taught. [3]
- 3. Pearl: Two different routes to vasodilation exist inside one class.
- 3. Why: Labetalol and carvedilol vasodilate through alpha-adrenergic receptor blockade, whereas nebivolol vasodilates by stimulating endothelial nitric oxide synthase through beta-3 agonism. [5]
- 4. Pearl: Cardioselectivity is dose-dependent rather than absolute.
- 4. Why: Nebivolol is beta-1 selective at lower doses and in fast metabolisers but blocks beta-1 and beta-2 at higher doses and in slower metabolisers, which is why a cardioselective label never fully removes the bronchospasm question. [5]
- 5. Pearl: Stopping the drug is itself a clinical event.
- 5. Why: Sudden withdrawal produces rebound sympathetic activation with a rise in heart rate and blood pressure and a heightened risk of adverse cardiovascular events, which is the basis of the metoprolol boxed warning. [3]
- 6. Pearl: Beta blockade masks the warning signs of hypoglycaemia.
- 6. Why: The class can induce hyperglycaemia and blunt the adrenergic features such as tachycardia that normally signal a falling glucose, which matters most in insulin-treated diabetes. [1]
- 7. Pearl: Sotalol is a beta blocker that behaves like a class III antiarrhythmic.
- 7. Why: It blocks potassium channels as well as beta receptors, so it prolongs the QT interval and brings torsades de pointes into the risk profile in a way the rest of the class does not. [1]
- 8. Pearl: Unopposed alpha stimulation is the reason for two classic prohibitions.
- 8. Why: Beta blockade in untreated pheochromocytoma or in cocaine toxicity leaves alpha-mediated vasoconstriction unopposed, which can produce severe hypertension. [3][4]

Common exam traps

- 1. Trap: "Cardioselective means beta-1 only, at any dose."
- 1. Correction: Selectivity is relative. Nebivolol is beta-1 selective at lower doses and in fast metabolisers, and blocks both beta-1 and beta-2 at higher doses and in slower metabolisers. [5]
- 2. Trap: "All beta blockers are absolutely contraindicated in asthma."
- 2. Correction: Non-selective agents are contraindicated in asthma, and a cardioselective agent may be used where no

alternative is tolerated - as a cautious fallback at the lowest workable dose, not as routine permission, since beta-1 selectivity is not absolute. [1][2]

3. Trap: "Every beta blocker is interchangeable in heart failure."

3. Correction: Bisoprolol, carvedilol and metoprolol succinate are the agents chosen for compensated heart failure, and metoprolol tartrate is not indicated for that purpose. [1]

4. Trap: "Stopping a beta blocker is harmless as long as blood pressure is controlled."

4. Correction: Sudden withdrawal causes rebound sympathetic activation and can precipitate angina or myocardial infarction, which is why a structured taper is advocated. [3]

5. Trap: "Carvedilol and nebivolol vasodilate by the same mechanism."

5. Correction: Carvedilol vasodilates through alpha-adrenergic blockade, whereas nebivolol does so through nitric oxide released by beta-3-mediated stimulation of endothelial nitric oxide synthase. [5]

6. Trap: "A beta blocker plus verapamil is a routine combination for rate control."

6. Correction: Both depress atrioventricular nodal conduction and contractility, so the combination raises the risk of bradycardia, heart block or acute decompensated heart failure and needs a clear indication and close monitoring. [8]

Self-test questions

1. Question: Which grouping of beta blockers by receptor selectivity is correct?

1. Propranolol, carvedilol, sotalol and labetalol are non-selective; atenolol, bisoprolol, metoprolol and esmolol are beta-1 selective

2. Propranolol and sotalol are beta-1 selective; atenolol and metoprolol are non-selective

3. All beta blockers are non-selective, and the term cardioselective refers only to the route of administration

4. Selectivity refers to beta-3 receptors, which are the main cardiac receptor subtype

1. Explanation: Non-selective agents bind both beta-1 and beta-2 receptors and include propranolol, carvedilol, sotalol and labetalol. Beta-1 selective, or cardioselective, agents include atenolol, bisoprolol, metoprolol and esmolol. [1]

2. Question: A cardioselective beta blocker that also possesses mild intrinsic sympathomimetic activity is best exemplified by which agent?

1. Metoprolol

2. Acebutolol

3. Sotalol

4. Esmolol

2. Explanation: Acebutolol is described as a cardioselective beta-adrenoceptor blocking agent possessing mild intrinsic sympathomimetic activity in its therapeutically effective dose range. Metoprolol, by contrast, has neither membrane stabilising nor intrinsic sympathomimetic activity. [7][3]

3. Question: Why does abrupt discontinuation of a beta blocker matter clinically?

1. It causes an immediate and permanent loss of beta receptor function

2. It produces rebound sympathetic activation with a rise in heart rate and blood pressure and a heightened risk of adverse cardiovascular events

3. It has no cardiovascular consequence provided blood pressure was controlled beforehand

4. It reliably causes hypoglycaemia within hours of the last dose

3. Explanation: Sudden withdrawal of beta blockade leads to rebound sympathetic activation, raising heart rate and blood pressure and increasing cardiovascular risk, particularly where there is coronary artery disease or heart failure. That is the reasoning behind a structured, gradual taper rather than an immediate stop. [3]

4. Question: Which statement about vasodilating beta blockers is correct?

1. Nebivolol vasodilates through alpha-1 blockade, as carvedilol does

2. Labetalol vasodilates by releasing nitric oxide from the endothelium

3. Carvedilol and labetalol vasodilate through alpha-adrenergic blockade, while nebivolol does so through nitric oxide

4. No beta blocker has any vasodilator property

4. Explanation: Carvedilol and labetalol carry additional alpha-1 receptor blocking activity alongside non-selective beta blockade. Nebivolol achieves vasodilation differently, by stimulating endothelial nitric oxide synthase through beta-3 agonism and so reducing systemic vascular resistance. [1][5]

5. Question: Which beta blocker carries a distinctive risk of torsades de pointes?

1. Atenolol
2. Bisoprolol
3. Sotalol
4. Esmolol

5. Explanation: Sotalol blocks cardiac potassium channels in addition to beta receptors and is classified as a class III antiarrhythmic. That potassium channel blockade prolongs the QT interval and increases the risk of torsades de pointes, so the QTc interval requires monitoring. [1]

6. Question: What is the recognised antidote in beta-blocker overdose?

1. Naloxone
2. Glucagon
3. Flumazenil
4. N-acetylcysteine

6. Explanation: Glucagon is the antidote for beta-blocker overdose and is particularly useful in beta-blocker-induced cardiotoxicity. Cardiac pacing, whether transcutaneous or transvenous, is the second-line option when glucagon fails. [1]

7. Question: Why is combining a beta blocker with verapamil approached with caution?

1. Verapamil inactivates beta blockers in the stomach before absorption
2. The two agents antagonise each other so completely that neither has any effect
3. Both depress atrioventricular nodal conduction and myocardial contractility, so the combination increases the likelihood of bradycardia, heart block or acute decompensated heart failure
4. The combination is only a problem in patients under forty years of age

7. Explanation: Beta blockers and verapamil act on the same two properties - atrioventricular nodal conduction and myocardial contractility - so their effects add. The combination should follow a clear indication and close monitoring, especially where conduction disease or left ventricular dysfunction is present, and even systemically absorbed ophthalmic timolol can contribute. [8]

8. Question: Which beta blocker is used specifically because its onset and half-life are short enough for titration in critical care?

1. Esmolol
2. Atenolol
3. Nebivolol
4. Bisoprolol

8. Explanation: Esmolol is typically used in intensive care or cardiac inpatient settings for refractory tachycardia such as atrial fibrillation, because its short onset of action and short half-life make it titratable. [1]

Frequently asked questions

1. Question: What does cardioselective actually mean?

1. Answer: It means the agent is directed at beta-1 receptors, which sit chiefly in the heart, rather than at beta-2 receptors distributed through the lungs and smooth muscle. The distinction is relative rather than absolute, and can be lost at higher doses or in slower metabolisers. [1][5]

2. Question: What is intrinsic sympathomimetic activity?

2. Answer: It describes agents that partially stimulate the beta receptor even while blocking it. Pindolol possesses this property in therapeutic dosage ranges and is non-selective, while acebutolol possesses it mildly and is cardioselective - which is why the two properties have to be learnt separately. [6][7]

3. Question: Why can a beta blocker not simply be stopped?

3. Answer: Sudden withdrawal produces rebound sympathetic activation, with a rise in heart rate and blood pressure and a heightened risk of adverse cardiovascular events, particularly where there is coronary artery disease or heart failure. A gradual reduction in dose is used instead. [3]

4. Question: Are beta blockers safe in asthma?

4. Answer: Non-selective agents should not be used in asthma. Where beta blockade is clinically necessary and other treatment is not tolerated, a cardioselective agent is preferred over a non-selective one, used cautiously and at the lowest workable dose, because beta-1 selectivity is not absolute and bronchospasm remains possible. [1][4]

5. Question: Which beta blockers are used in heart failure?

5. Answer: Bisoprolol, carvedilol and metoprolol succinate are the agents chosen, and only when the patient is in a compensated state. Metoprolol tartrate is not indicated for heart failure. [1]

6. Question: Why does sotalol need extra monitoring?

6. Answer: Because it blocks cardiac potassium channels as well as beta receptors, which prolongs the QT interval and increases the risk of torsades de pointes. The QTc interval is therefore monitored alongside heart rate and blood pressure. [1]

7. Question: What is done in beta-blocker overdose?

7. Answer: Glucagon is the antidote and is particularly useful for beta-blocker-induced cardiotoxicity, with cardiac pacing as the second-line option if glucagon fails. [1]

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