

# Angiotensin receptor blockers

Selective blockers of the angiotensin II type 1 receptor that share the indications of ACE inhibitors but not their bradykinin-mediated cough, while keeping the same hyperkalaemia, renal and pregnancy cautions.

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

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## Quick revision

One Liner: ARBs block angiotensin II at the AT1 receptor rather than blocking its production, so the vasoconstriction and aldosterone release stop while bradykinin metabolism is left alone - which is exactly why the cough goes away and the hyperkalaemia does not.

1. Mechanism: selective blockade of the angiotensin II type 1 receptor, producing vasodilation and reduced aldosterone secretion. [1]
2. Indications overlap with ACE inhibitors: hypertension, heart failure, and chronic kidney disease including diabetic nephropathy. [1]
3. The defining advantage is a lower incidence of cough and angio-oedema, because ARBs do not raise bradykinin concentrations. [1]
4. Hyperkalaemia, hypotension, dizziness and renal impairment remain shared class problems that require monitoring. [1]
5. Contraindicated in pregnancy under a boxed warning because of fetal renal toxicity and skeletal deformity. [1]
6. Combining an ARB with an ACE inhibitor or a direct renin inhibitor is no longer recommended and should be avoided. [1]
7. Candesartan, olmesartan and azilsartan are administered as prodrugs and need hydrolysis in the gastrointestinal tract. [1]
8. Losartan is uniquely uricosuric, lowering uric acid by inhibiting URAT1 in the renal proximal tubule. [1]
9. Olmesartan is the member associated with sprue-like enteropathy, a rare but distinctive adverse effect. [1]

## Overview

1. Angiotensin receptor blockers selectively inhibit the binding of angiotensin II to the angiotensin type 1 receptor. That single action produces vasodilation, decreases aldosterone secretion and lowers blood pressure, and it is why the class is prescribed for hypertension, congestive heart failure and chronic kidney disease including diabetic nephropathy. [1]
2. The pathway they interrupt starts in the kidney. Renin from the juxtaglomerular cells generates angiotensin I from angiotensinogen, and angiotensin-converting enzyme - along with non-ACE routes - converts it to angiotensin II. That non-ACE conversion is part of why blocking the receptor is not simply a slower version of blocking the enzyme. [1]
3. Angiotensin II acts on two receptors that oppose each other. AT1 activation contracts vascular smooth muscle, raises systemic vascular resistance, enhances sympathetic activity and promotes sodium and water retention both directly in the proximal tubule and indirectly through adrenal aldosterone. AT2 activation does the reverse: vasodilation through nitric oxide and bradykinin, renal sodium excretion, and antiproliferative and cardioprotective effects. Chronically elevated angiotensin II also drives smooth and cardiac muscle proliferation, endothelial dysfunction, inflammation and apoptosis. [1]
4. Their therapeutic role overlaps heavily with ACE inhibitors, and the practical distinction is tolerability rather than efficacy. Many patients who develop a chronic non-productive cough on an ACE inhibitor benefit from switching, because ARBs do not raise bradykinin. What does not change is the rest of the safety profile: hyperkalaemia,

hypotension, dizziness, renal impairment, the pregnancy contraindication and the need for caution in bilateral renal artery stenosis all carry over. [1]

## Classification and drug examples

Intro: Every member blocks the same receptor, so the useful division is by whether the drug arrives active or has to be unmasked first - a split that also tracks the protein binding and clearance differences worth remembering.

1. Name: Directly active angiotensin receptor blockers

1. Note: Administered in an active form, although losartan additionally generates an active metabolite that carries much of its effect.

1. Name: Losartan

1. Route: Oral

1. Note: Bioavailability around 33%; metabolised by CYP2C9 and CYP3A4 to EXP3174, an active metabolite that contributes substantially to the antihypertensive effect. Uniquely uricosuric through inhibition of urate transporter 1. [1]

2. Name: Valsartan

2. Route: Oral

2. Note: Bioavailability around 25% and a half-life of about 6 hours; roughly 30% of clearance is renal. It is also the ARB component of the combination with sacubitril. [1]

3. Name: Irbesartan

3. Route: Oral

3. Note: The best-absorbed member, with bioavailability of 60% to 80% and a half-life of 11 to 15 hours; hepatic clearance predominates and food does not affect exposure. [1]

4. Name: Telmisartan

4. Route: Oral

4. Note: Protein binding above 99%, half-life about 24 hours, cleared mainly hepatically and conjugated rather than metabolised by CYP enzymes, which limits CYP-mediated interactions. [1]

5. Name: Eprosartan

5. Route: Oral

5. Note: Half-life about 20 hours, and the one member recommended to be taken with food. [1] [1]

2. Name: Prodrug angiotensin receptor blockers

2. Note: Given as prodrugs requiring enzymatic hydrolysis in the gastrointestinal tract to release the active moiety; once converted, they undergo little further metabolism and are largely eliminated unchanged.

1. Name: Candesartan

Candesartan cilexetil

1. Route: Oral

1. Note: Protein binding above 99%, roughly 40% renal clearance, and one of the members with a heart failure indication alongside hypertension. [1]

2. Name: Olmesartan

Olmesartan medoxomil

2. Route: Oral

2. Note: Protein binding above 99% and about 40% renal clearance. It is the member associated with sprue-like enteropathy, and it performs well for nocturnal blood pressure control. [1]

3. Name: Azilsartan

Azilsartan medoxomil

3. Route: Oral

3. Note: Metabolised by CYP2C9 but only to inactive metabolites, with about 20% renal clearance. [1] [1]

## Mechanism of action

Summary: ARBs selectively inhibit the binding of angiotensin II to the AT1 receptor. Blocking that receptor produces vasodilation, reduces aldosterone secretion and lowers blood pressure, while leaving the AT2 receptor and bradykinin metabolism untouched.

Target: Angiotensin II type 1 (AT1) receptor, a G-protein-coupled receptor expressed in heart, vasculature, kidney, adrenal gland, pituitary and central nervous system

### 1. Step: 1

1. Title: Angiotensin II is generated by more than one route

1. Detail: Renin from the juxtaglomerular cells converts angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme and by non-ACE pathways. Angiotensin II is the principal vasoactive peptide of the system. [1]

### 2. Step: 2

2. Title: AT1 activation is the harmful arm

2. Detail: Angiotensin II acting at AT1 increases blood pressure by contracting vascular smooth muscle, raising systemic vascular resistance and enhancing sympathetic activity, while promoting sodium and water retention through proximal tubular reabsorption and adrenal aldosterone production. [1]

### 3. Step: 3

3. Title: The drug occupies AT1 selectively

3. Detail: An ARB binds the AT1 receptor and prevents angiotensin II from acting there, which is what produces vasodilation, reduced aldosterone secretion and a fall in blood pressure. [1]

### 4. Step: 4

4. Title: The AT2 arm is left available

4. Detail: AT2 activation opposes AT1: it causes vasodilation largely through increased nitric oxide and bradykinin production, promotes renal sodium excretion, and exerts antiproliferative and cardiovascular protective effects. Selective AT1 blockade does not interfere with it. [1]

### 5. Step: 5

5. Title: Bradykinin metabolism is untouched

5. Detail: Because the drug acts at the receptor rather than at angiotensin-converting enzyme, kinin breakdown continues normally. That is the mechanistic reason cough and angio-oedema are less frequent than with ACE inhibitors. [1]

### 6. Step: 6

6. Title: Long-term remodelling slows

6. Detail: Chronically elevated angiotensin II contributes to smooth and cardiac muscle proliferation, endothelial dysfunction, platelet aggregation, inflammation and apoptosis. Removing AT1 signalling interrupts those processes, which is why the class also reduces intraglomerular pressure and proteinuria in chronic kidney disease. [1]

## Major clinical uses

### 1. Drug: ARBs as a class

1. Indication: Hypertension, alone or with other antihypertensives

1. Line: first-line

1. Note: One of the four classes named as first-line for initiating antihypertensive therapy, alongside thiazide diuretics, calcium channel blockers and ACE inhibitors. [1]

### 2. Drug: ARBs as a class

2. Indication: Congestive heart failure

2. Line: guideline-directed

2. Note: Supported by the American Heart Association and American College of Cardiology for managing heart failure.

[1]

3. Drug: ARBs as a class

3. Indication: Chronic kidney disease with albuminuria, including diabetic nephropathy

3. Line: first-line

3. Note: KDIGO recommends an ACE inhibitor or an ARB for CKD stages G1 to G4 with severely increased albuminuria regardless of diabetes status, and where albuminuria is moderately to severely increased in diabetes. [1]

4. Drug: ARBs as a class

4. Indication: Intolerance of an ACE inhibitor because of chronic non-productive cough or angio-oedema

4. Line: alternative

4. Note: The standard substitution, and the reason most students meet this class second rather than first. [1]

5. Drug: ARBs as a class

5. Indication: Recent myocardial infarction with left ventricular ejection fraction of 40% or below, in patients intolerant of ACE inhibitors

5. Line: alternative

5. Note: Used in this setting to help prevent symptomatic heart failure and reduce mortality risk. [1]

6. Drug: Losartan

6. Indication: High blood pressure, heart failure, and kidney disease caused by type 2 diabetes; also stroke prevention in hypertension with left ventricular hypertrophy, and proteinuria

6. Line: first-line

6. Note: The left ventricular hypertrophy stroke-prevention indication is stated as excluding African-American patients. [1][4]

7. Drug: Irbesartan

7. Indication: Hypertension and diabetic nephropathy

7. Line: first-line

7. Note: One of the members with a specific diabetic nephropathy indication. [1]

8. Drug: Telmisartan

8. Indication: Hypertension, and reduction of cardiovascular mortality in adults aged 55 and over with cardiovascular risk factors who cannot tolerate ACE inhibitors

8. Line: guideline-directed

8. Note: The same dosing basis is described for stroke and myocardial infarction prophylaxis in that group. [1]

## Pharmacokinetics

1. Drug: Losartan

1. Route: Oral

1. Absorption: Absolute bioavailability around 33%

1. Metabolism: CYP2C9 and CYP3A4 to the active metabolite EXP3174

1. Elimination: About 10% renal for losartan itself and about 60% for its active metabolite

1. Half Life: About 2 hours for losartan itself, and about 6 to 9 hours for its active metabolite

1. Dose Adjust: Correct volume or salt depletion before starting, or begin at a lower dose [1][3]

2. Drug: Valsartan

2. Route: Oral

2. Absorption: Absolute bioavailability around 25%

2. Metabolism: Minimal

2. Elimination: About 30% renal

2. Half Life: Approximately 6 hours

2. Dose Adjust: Reduce the dose or stop if renal function declines significantly [1]

3. Drug: Irbesartan

- 3. Route: Oral
- 3. Absorption: Bioavailability 60% to 80%; exposure unaffected by food
- 3. Metabolism: Hepatic
- 3. Elimination: Hepatic clearance accounts for most of the total
- 3. Half Life: 11 to 15 hours
- 3. Dose Adjust: Caution where high-dose diuretics have activated the renin-angiotensin system [1]
- 4. Drug: Telmisartan
- 4. Route: Oral
- 4. Absorption: Exposure unaffected by food; protein binding above 99%
- 4. Metabolism: Conjugation, mainly glucuronidation - not a CYP substrate
- 4. Elimination: Hepatic clearance accounts for most of the total
- 4. Half Life: Approximately 24 hours
- 4. Dose Adjust: Dose reduction or discontinuation for symptomatic hypotension or uncontrolled hyperkalaemia [1]
- 5. Drug: Olmesartan
- 5. Route: Oral
- 5. Absorption: Given as a prodrug hydrolysed in the gastrointestinal tract; protein binding above 99%
- 5. Metabolism: Minimal after conversion from the prodrug
- 5. Elimination: About 40% renal, largely unchanged
- 5. Half Life: Approximately 10 to 15 hours
- 5. Dose Adjust: Lower starting dose in adults aged 65 and over; correct volume depletion first [1]
- 1. Plasma protein binding is high across the class, typically above 95% and above 99% for telmisartan, candesartan and olmesartan, predominantly to albumin. [1]
- 2. All members may be taken with or without food, with eprosartan the exception that is recommended with food. Once-daily administration is generally supported by the half-lives involved. [1]
- 3. Volume or salt depletion should be corrected before an ARB is started, or a lower starting dose used, because an activated renin-angiotensin system makes symptomatic hypotension much more likely. [1]
- 4. Blood tests and blood pressure checks are arranged while a patient is on treatment, to see how the drug is affecting blood pressure and the kidneys. Illness with vomiting, diarrhoea or fever carries a dehydration risk that is specifically counselled on. [4]
- 5. This page gives no dose regimens by design. Doses depend on indication, renal function, volume status, age and comorbidity, and belong in a prescribing reference used by the treating clinician.

## Adverse effects

- 1. Effect: Hyperkalaemia
- 1. Detail: A predictable consequence of removing the aldosterone stimulus. The risk is higher in renal disease and with potassium supplements, potassium-sparing diuretics, ACE inhibitors, direct renin inhibitors or NSAIDs. [1]
- 2. Effect: Hypotension and dizziness
- 2. Detail: Most likely in patients with an activated renin-angiotensin system, such as those on high-dose diuretics, which is why volume or salt depletion is corrected before starting. [1]
- 3. Effect: Renal impairment
- 3. Detail: ARBs may reduce renal function in patients whose filtration is highly dependent on the renin-angiotensin system. Renal function is monitored regularly and therapy stopped if a clinically significant decline occurs. [1]
- 4. Effect: Overall tolerability
- 4. Detail: The class is generally well tolerated with a low incidence of side effects, and a lower incidence of cough and angio-oedema than ACE inhibitors. [1]
- 1. Effect: Fetal toxicity
- 1. Detail: Carried as a boxed warning. Reduced fetal kidney perfusion may lead to renal dysgenesis, oliguric or anuric

renal failure, oligohydramnios, skeletal or skull deformities, pulmonary hypoplasia and fetal death, and reduced placental and umbilical blood flow can restrict growth.

1. Action: Therapy is discontinued immediately if pregnancy is detected, unless treatment is judged lifesaving for the mother. [1]
2. Effect: Acute renal failure
2. Detail: Particularly in renal artery stenosis, chronic kidney disease, severe heart failure or volume depletion. Higher doses are associated with an increased risk of acute kidney injury, especially alongside heart failure, diarrhoea or sepsis.
2. Action: Renal function is monitored regularly, and the dose reduced or the drug discontinued if function declines significantly. [1]
3. Effect: Angio-oedema
3. Detail: Rare with this class and less frequent than with ACE inhibitors, but reported, so it cannot be treated as impossible.
3. Action: Recognition and withdrawal of the drug, as with any angio-oedema attributed to renin-angiotensin blockade. [1]
4. Effect: Vasodilatory shock in overdose
4. Detail: Overdose typically presents as hypotension and shock from vasodilation, with a high cardiac index and low systemic vascular resistance - a vasoplegic rather than cardiogenic pattern, and one that can be prolonged.
4. Action: Management is supportive and delivered in a critical care setting; the pattern is recognised partly by the failure of therapies aimed at calcium channel blocker toxicity. [1]
1. Drug: Olmesartan
1. Effect: Associated with sprue-like enteropathy. It has also been linked in one analysis to increased cardiovascular mortality in high-risk populations, possibly through excessive blood pressure reduction. [1]
2. Drug: ARBs as a class
2. Effect: Rare drug-induced liver injury, most cases cholestatic and mild with no consistent dose-dependent pattern, suggesting an idiosyncratic mechanism. Close monitoring during the first weeks of therapy is recommended. [1]
3. Drug: ARBs as a class
3. Effect: Other rare reactions include urticaria, anaphylaxis, vasculitis, neutropenia, leukopenia and liver function test abnormalities. [1]

## Contraindications, precautions and interactions

1. Pregnancy, under a boxed warning, because of fetal renal and skeletal toxicity. [1]
2. Hypersensitivity to the drug or its excipients. [1]
3. Coadministration with aliskiren in a patient diagnosed with diabetes. [1]
1. Bilateral renal artery stenosis, and heart failure complicated by hypotension, where filtration and pressure both depend on an active renin-angiotensin system. [1]
2. Volume or salt depletion, including patients on high-dose diuretics, where correction before starting therapy reduces symptomatic hypotension. [1]
3. Kidney failure with an estimated glomerular filtration rate below 15 mL/min per 1.73 m squared, where dose reduction or discontinuation is described for symptomatic hypotension or uncontrolled hyperkalaemia. [1]
4. Ascites and liver failure, where drugs that diminish renal perfusion or raise acute kidney injury risk are avoided. [1]
5. Breastfeeding, where no published evidence supports safe use and the effects on a nursing infant are unknown. [1]
6. Older adults, in whom a lower starting dose is considered because of increased sensitivity and hypotension risk. [1]
1. With: ACE inhibitors and direct renin inhibitors
1. Effect: Combination increases the risk of hypotension, acute renal failure and hyperkalaemia. Concurrent use is no longer recommended and should be avoided. [1]
2. With: Potassium supplements, potassium-sparing diuretics and NSAIDs

2. Effect: Further elevation of serum potassium, particularly in renal disease; in some patients avoiding the combination altogether is safer. [1]
3. With: Lithium
3. Effect: Serum lithium concentrations rise with the risk of toxicity, so levels are monitored and doses adjusted during coadministration. [1]
4. With: Other antihypertensive medications
4. Effect: The blood pressure-lowering effect is enhanced, which may require the dose of either agent to be adjusted. [1]

## Comparison tables

1. Title: ARBs compared with ACE inhibitors
1. Caption: The two classes share a pathway and most of their indications. Every genuine difference traces back to whether bradykinin metabolism is affected.
1. Key: feature
1. Label: Feature
1. Is Row Header: true
2. Key: acei
2. Label: ACE inhibitors
3. Key: arb
3. Label: ARBs
1. Feature: Point of action
1. Acei: Block conversion of angiotensin I to angiotensin II
1. Arb: Block angiotensin II at the AT1 receptor [1]
2. Feature: Effect on bradykinin
2. Acei: Bradykinin breakdown is inhibited, so kinins accumulate
2. Arb: Bradykinin metabolism is unaffected [1][2]
3. Feature: Dry cough
3. Acei: Reported in 10% to 20% of patients
3. Arb: Lower incidence, though rare cases are reported [2][1]
4. Feature: Angio-oedema
4. Acei: Rare but potentially life-threatening, and a permanent bar to the class
4. Arb: Less frequent, but reported and not excluded [2][1]
5. Feature: Hyperkalaemia
5. Acei: Reported in 2% to 6% of patients
5. Arb: Same mechanism, same requirement for periodic potassium monitoring [2][1]
6. Feature: Pregnancy
6. Acei: Contraindicated because of documented fetopathy
6. Arb: Contraindicated under a boxed warning [2][1]
7. Feature: Using both together
7. Acei: Not recommended
7. Arb: Not recommended - hypotension, acute renal failure and hyperkalaemia all increase [1]
2. Title: Member-specific points worth knowing
2. Caption: Facts that belong to one drug rather than the class. Actual therapy is governed by a prescribing reference and the treating clinician.
1. Key: drug
1. Label: Drug
1. Is Row Header: true

2. Key: prodrug
2. Label: Prodrug?
3. Key: feature
3. Label: What sets it apart
1. Drug: Losartan
1. Prodrug: No
1. Feature: Uricosuric through URAT1 inhibition; active metabolite EXP3174 carries much of the effect [1]
2. Drug: Telmisartan
2. Prodrug: No
2. Feature: Longest half-life at about 24 hours; conjugated rather than CYP-metabolised [1]
3. Drug: Irbesartan
3. Prodrug: No
3. Feature: Highest oral bioavailability at 60% to 80%; predominantly hepatic clearance [1]
4. Drug: Valsartan
4. Prodrug: No
4. Feature: A half-life of about 6 hours; the ARB component of the sacubitril combination [1]
5. Drug: Olmesartan
5. Prodrug: Yes
5. Feature: Associated with sprue-like enteropathy; strong nocturnal blood pressure control [1]
6. Drug: Candesartan
6. Prodrug: Yes
6. Feature: Protein binding above 99%; carries a heart failure indication as well as hypertension [1]
7. Drug: Azilsartan
7. Prodrug: Yes
7. Feature: CYP2C9 metabolism produces only inactive metabolites [1]
8. Drug: Eprosartan
8. Prodrug: No
8. Feature: The one member recommended to be taken with food [1]

## High-yield exam pearls

1. Pearl: The difference between ACE inhibitors and ARBs is bradykinin, not potency.
1. Why: ACE degrades bradykinin as well as producing angiotensin II, so inhibiting it lets kinins accumulate and irritate the airway. ARBs act downstream at the receptor and leave bradykinin metabolism untouched, which is why cough and angio-oedema are less frequent with them. [1][2]
2. Pearl: AT1 and AT2 receptors do opposite things.
2. Why: AT1 activation raises blood pressure through vascular smooth muscle contraction, higher systemic vascular resistance, sympathetic activation and sodium retention. AT2 activation causes vasodilation and natriuresis and is anti-inflammatory and antifibrotic. Blocking AT1 selectively leaves the protective arm intact. [1]
3. Pearl: Losartan lowers uric acid; the rest of the class does not.
3. Why: It inhibits urate transporter 1 in the renal proximal tubules, making it uricosuric - a genuinely member-specific property rather than a class effect. [1]
4. Pearl: Telmisartan avoids the cytochrome P450 system entirely.
4. Why: It is not metabolised by CYP enzymes and instead undergoes conjugation, mainly glucuronidation, which reduces the potential for CYP-mediated interactions. Its half-life of about 24 hours is also the longest in common use. [1]
5. Pearl: Olmesartan is the sprue-like enteropathy drug.
5. Why: It is the only ARB specifically associated with that presentation, which mimics coeliac disease and is a classic

single-fact examination target. [1]

6. Pearl: Three members are prodrugs.

6. Why: Candesartan, olmesartan and azilsartan are given as prodrugs requiring enzymatic hydrolysis in the gastrointestinal tract to release the active moiety; the rest are administered in active form. [1]

7. Pearl: Dual RAAS blockade is a trap, not a strategy.

7. Why: KDIGO strongly advises against combining ACE inhibitors, ARBs and direct renin inhibitors in chronic kidney disease regardless of diabetes status, because of increased adverse events including hypotension, acute renal failure and hyperkalaemia. [1]

## Common exam traps

1. Trap: "An ARB is completely safe in a patient who had angio-oedema on an ACE inhibitor."

1. Correction: The incidence is lower because ARBs do not elevate bradykinin, but angio-oedema has been reported in patients using ARBs. It is a reduced risk rather than an abolished one. [1]

2. Trap: "ARBs avoid the hyperkalaemia problem that ACE inhibitors have."

2. Correction: They do not. Blocking AT1 still removes the aldosterone stimulus, so potassium rises by the same route. Serum potassium needs periodic monitoring, and concurrent use with other potassium-raising agents must be avoided. [1]

3. Trap: "An ARB is a safer alternative in pregnancy than an ACE inhibitor."

3. Correction: ARBs carry the same boxed warning. Maternal use can cause fetal renal failure, lung dysplasia, cranial hypoplasia, limb contractures and fetal or neonatal death, and therapy should be discontinued as soon as pregnancy is detected. [1]

4. Trap: "Blocking the system at two points gives twice the benefit."

4. Correction: Combining an ARB with an ACE inhibitor or a direct renin inhibitor increases hypotension, acute renal failure and hyperkalaemia, and concurrent use is no longer recommended. [1]

5. Trap: "All ARBs behave the same pharmacokinetically."

5. Correction: Oral bioavailability alone ranges from about 25% for valsartan to 60% to 80% for irbesartan, and half-lives run from roughly 6 hours for valsartan to about 24 hours for telmisartan. Three members are prodrugs and one is uricosuric. [1]

6. Trap: "A higher ARB dose is always the safer way to intensify blood pressure control."

6. Correction: Higher doses are associated with an increased risk of acute kidney injury, particularly in patients with comorbid conditions such as heart failure, diarrhoea or sepsis. [1]

## Self-test questions

1. Question: Where in the renin-angiotensin-aldosterone system do ARBs act?

1. They inhibit renin release from juxtaglomerular cells
2. They prevent conversion of angiotensin I to angiotensin II
3. They selectively block angiotensin II at the type 1 receptor
4. They antagonise aldosterone at the mineralocorticoid receptor

1. Explanation: ARBs selectively inhibit the binding of angiotensin II to the angiotensin type 1 receptor. Direct renin inhibitors act on renin production, ACE inhibitors prevent the conversion of angiotensin I to angiotensin II, and aldosterone antagonists work at the mineralocorticoid receptor - four different points on the same pathway. [1]

2. Question: Why does switching from an ACE inhibitor to an ARB usually resolve a drug-induced cough?

1. ARBs are shorter-acting, so the airway recovers between doses
2. ARBs do not elevate bradykinin, which is the mediator responsible for the cough
3. ARBs suppress the cough reflex centrally
4. ARBs are given at lower equivalent doses, so airway irritation is reduced

2. Explanation: The incidence of cough and angio-oedema with ARBs is lower than with ACE inhibitors because ARBs

do not elevate bradykinin levels. The cough on an ACE inhibitor is caused by kinin accumulation in the lung, so a drug that leaves bradykinin metabolism alone removes the cause. [1][2]

3. Question: Which ARB has uric acid-lowering properties?

1. Telmisartan
2. Losartan
3. Candesartan
4. Olmesartan

3. Explanation: Among ARBs, losartan functions as a uricosuric agent by inhibiting urate transporter 1 in the renal proximal tubules, thereby lowering uric acid levels. This is specific to losartan and is not shared by the rest of the class. [1]

4. Question: Which ARB is associated with sprue-like enteropathy?

1. Irbesartan
2. Valsartan
3. Olmesartan
4. Eprosartan

4. Explanation: Olmesartan has been associated with sprue-like enteropathy. Other rare adverse effects reported across the class include urticaria, anaphylaxis, vasculitis, neutropenia, leukopenia and liver function test abnormalities. [1]

5. Question: A patient with chronic kidney disease is already taking an ACE inhibitor. What is the position on adding an ARB?

1. It is recommended, because dual blockade slows progression more effectively
2. It is strongly advised against, because of increased adverse events
3. It is recommended only if the patient has diabetes
4. It is acceptable provided the ACE inhibitor dose is halved

5. Explanation: KDIGO strongly advises against combining ACE inhibitors, ARBs and direct renin inhibitors in individuals with chronic kidney disease, regardless of diabetes status, because of the increased risk of adverse events. The combination raises the risk of hypotension, acute renal failure and hyperkalaemia. [1]

6. Question: Which of these is the correct pairing of an ARB with a distinguishing pharmacokinetic feature?

1. Telmisartan - extensively metabolised by CYP3A4, with a half-life of about 6 hours
2. Irbesartan - bioavailability of about 25%, cleared almost entirely by the kidney
3. Telmisartan - not metabolised by CYP enzymes, half-life of about 24 hours, predominantly hepatic clearance
4. Losartan - a prodrug requiring gastrointestinal hydrolysis before it becomes active

6. Explanation: Telmisartan is not metabolised by CYP enzymes and instead undergoes conjugation, primarily glucuronidation. Its terminal half-life is approximately 24 hours and hepatic clearance accounts for the majority of its elimination. Irbesartan's bioavailability is 60% to 80%, and losartan is not a prodrug - candesartan, olmesartan and azilsartan are. [1]

7. Question: Which contraindication do ARBs share with ACE inhibitors?

1. Asthma
2. Pregnancy
3. Atrial fibrillation
4. Hypothyroidism

7. Explanation: ARBs, like ACE inhibitors, are contraindicated during pregnancy because of the risk of reduced fetal kidney perfusion, which may lead to renal dysgenesis, oliguric or anuric renal failure, oligohydramnios, skeletal or skull deformities, pulmonary hypoplasia and fetal death. Therapy should be discontinued immediately if pregnancy is detected. [1]

## Frequently asked questions

1. Question: How is an ARB different from an ACE inhibitor?

1. Answer: An ACE inhibitor stops angiotensin II being made; an ARB lets it be made but blocks it at the AT1 receptor. Because angiotensin-converting enzyme also breaks down bradykinin, only the ACE inhibitor causes kinins to accumulate, and that is the source of the cough and much of the angio-oedema risk. [1][2]

2. Question: Do ARBs still cause high potassium?

2. Answer: Yes. Blocking AT1 still removes the signal for aldosterone release, so potassium retention happens by the same route as with an ACE inhibitor. Periodic monitoring of serum potassium is part of therapy, and other potassium-raising drugs need care. [1]

3. Question: Can an ARB be combined with an ACE inhibitor for extra effect?

3. Answer: No. Combining agents that block the renin-angiotensin system at more than one point raises the risk of hypotension, acute renal failure and hyperkalaemia, and the combination is no longer recommended. KDIGO advises strongly against it in chronic kidney disease. [1]

4. Question: Why must ARBs be stopped in pregnancy?

4. Answer: Fetal kidney perfusion depends on angiotensin II. Blocking it can cause renal dysgenesis, oliguric or anuric renal failure, oligohydramnios, skull and skeletal deformities, pulmonary hypoplasia and fetal death, so the class carries a boxed warning and therapy is stopped as soon as pregnancy is detected. [1]

5. Question: Is one ARB better than another?

5. Answer: The receptor blockade is a class effect, so the practical differences are pharmacokinetic and incidental: losartan lowers uric acid, telmisartan has the longest half-life and avoids CYP metabolism, irbesartan is the best absorbed, and olmesartan is the one linked to sprue-like enteropathy. [1]

6. Question: What is monitored during ARB treatment?

6. Answer: Blood pressure, renal function and serum electrolytes, especially potassium. Where an ARB is used in heart failure, ejection fraction is followed as well, and during acute decompensation natriuretic peptide levels and a chest x-ray are used to assess the picture. [1][4]

## References

[1] Angiotensin II Receptor Blockers (StatPearls). StatPearls Publishing / NCBI Bookshelf 2025.

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[4] Losartan: medicine for high blood pressure (NHS). National Health Service (UK) 2026.

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