

Loop diuretics

The most powerful diuretics in routine use, blocking the Na-K-2Cl cotransporter of the thick ascending limb, and the only diuretic class with ototoxicity as a specific adverse effect.

Online guide: <https://www.medguideglobal.com/pharmacology/cardiovascular/loop-diuretics>

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Quick revision

One Liner: Loop diuretics compete with chloride at the Na-K-2Cl cotransporter of the thick ascending limb; the segment they block reabsorbs a large share of filtered sodium, which explains both their potency and the depth of the electrolyte disturbance they cause.

1. Mechanism: competition with chloride for the Na-K-2Cl (NKCC2) cotransporter at the apical membrane of the thick ascending limb of the loop of Henle. [1]
2. Four agents make up the class: furosemide, bumetanide, torsemide and ethacrynic acid. [1]
3. Approved for oedema associated with congestive heart failure, liver cirrhosis and renal disease including nephrotic syndrome, and for hypertension though not as first-line therapy. [1]
4. Blocking the cotransporter also removes the driving force for calcium and magnesium reabsorption, so both ions are lost. [1]
5. Ototoxicity is specific to this class and is not shared by any other diuretic, presenting as sensorineural hearing loss, tinnitus, vertigo or dizziness. [2]
6. Deafness and tinnitus occur most often after large bolus doses in acute settings, because the injury tracks the high serum concentration achieved. [2]
7. Ethacrynic acid is the most ototoxic member and can cause permanent sensorineural hearing loss, but it is not a sulfonamide derivative. [1][2]
8. Hyponatraemia, hypokalaemia, hypochloraemia, hypomagnesaemia and metabolic alkalosis form the characteristic electrolyte picture. [1]
9. Oral bioavailability separates the agents: furosemide averages about 50%, while bumetanide and torsemide are closer to 80%. [1]

Overview

1. Loop diuretics act at the thick ascending limb of the loop of Henle, competing with chloride for the Na-K-2Cl cotransporter on the apical membrane and inhibiting reabsorption of sodium and chloride there. Because this segment reabsorbs a large share of the filtered sodium load, blocking it produces the most powerful diuresis available in routine practice. The class comprises furosemide, bumetanide, torsemide and ethacrynic acid. [1]
2. Approved use centres on oedema associated with congestive heart failure, liver cirrhosis and renal disease including nephrotic syndrome. These agents are also approved for hypertension, although they are not first-line for that purpose, and pulmonary oedema and severe hypertension with renal disease are recorded off-label roles. [1]
3. Two features dominate their safety profile. The first is depth of electrolyte disturbance - hyponatraemia, hypokalaemia, hypochloraemia, hypomagnesaemia, metabolic alkalosis and calcium loss. The second is ototoxicity, which is described as specific to loop agents and not shared by any other diuretic, and which becomes most likely when high serum concentrations are reached quickly. [1][2]

Classification and drug examples

Intro: A small class best divided by chemistry, because the sulfonamide-derived agents and the single non-sulfonamide agent behave differently in allergy and differ in ototoxic potential.

1. Name: Sulfonamide-derived loop diuretics

1. Note: The three agents in routine use. Cross-allergenicity with sulfonamide antibiotics carries only a low potential, but hypersensitivity to sulfonamides is still listed as a contraindication.

1. Name: Furosemide

Furosemide

1. Route: Oral/IV/IM

1. Note: The reference agent of the class; oral bioavailability averages about 50% and its half-life is short. [1]

2. Name: Bumetanide

2. Route: Oral/IV

2. Note: Oral bioavailability closer to 80%, with the shortest half-life of the group. [1]

3. Name: Torsemide

Torsemide

3. Route: Oral/IV

3. Note: Bioavailability close to 80% and the longest half-life of the three sulfonamide-derived agents. [1] [1]

2. Name: Non-sulfonamide loop diuretic

2. Note: A single agent, retained largely because its chemistry differs from the rest of the class.

Name: Ethacrynic acid

Route: Oral/IV

Note: Not a sulfonamide derivative, which makes it the option where sulfonamide allergy is a concern; it is also the most ototoxic member of the class. [1][2] [1]

Mechanism of action

Summary: Loop diuretics compete with chloride for the Na-K-2Cl cotransporter on the apical membrane of the thick ascending limb, blocking sodium and chloride reabsorption in the nephron segment that handles the largest reabsorbable sodium load outside the proximal tubule.

Target: Na-K-2Cl (NKCC2) cotransporter, apical membrane of the thick ascending limb

Cidal Static: not applicable

Kill Kinetics: not applicable

1. Step: 1

1. Title: The drug reaches the luminal side of the thick ascending limb

1. Detail: The site of action is the apical membrane of the thick ascending limb of the loop of Henle, so the drug must be present in tubular fluid to work. [1]

2. Step: 2

2. Title: It competes with chloride at the NKCC2 cotransporter

2. Detail: Binding is competitive with chloride at the Na-K-2Cl cotransporter, which is what blocks the transporter rather than merely slowing it. [1]

3. Step: 3

3. Title: Sodium and chloride reabsorption is inhibited

3. Detail: With the cotransporter blocked, sodium and chloride remain in the tubular fluid and a large natriuresis follows. [1]

4. Step: 4

4. Title: Potassium recycling stops and divalent cations are lost

4. Detail: Potassium can no longer be returned to the lumen, and the loss of that recycling removes the driving force for calcium and magnesium reabsorption in the same segment. [1]

5. Step: 5

5. Title: The same transporter family in the inner ear explains the ototoxicity

5. Detail: Inhibition of the NKCC1 channel in the stria vascularis, together with ischaemia there, is the described

mechanism of the reversible hearing loss and tinnitus characteristic of the class. [2]

Major clinical uses

1. Drug: Furosemide
1. Organism: Not applicable
1. Indication: Oedema associated with congestive heart failure
1. Line: first-line
1. Note: The best-established use of the class; response is judged on weight, fluid status and renal function rather than on urine output alone. [1]
2. Drug: Loop diuretics as a class
2. Organism: Not applicable
2. Indication: Oedema associated with liver cirrhosis
2. Line: targeted
2. Note: Electrolyte depletion and dehydration in this setting can precipitate hepatic encephalopathy. [1][5]
3. Drug: Loop diuretics as a class
3. Organism: Not applicable
3. Indication: Oedema of renal disease, including nephrotic syndrome
3. Line: targeted
3. Note: Retained as the diuretic of choice where reduced filtration limits thiazide effectiveness. [1][3]
4. Drug: Bumetanide
4. Organism: Not applicable
4. Indication: Oedema where oral response to furosemide is inadequate
4. Line: targeted
4. Note: Higher and more predictable oral bioavailability is the pharmacological reason for the switch. [1]
5. Drug: Loop diuretics as a class
5. Organism: Not applicable
5. Indication: Hypertension, and off-label in acute pulmonary oedema
5. Line: adjunct
5. Note: Approved for hypertension but not as first-line therapy for it; pulmonary oedema and severe hypertension with renal disease are recorded off-label uses. [1]
6. Drug: Ethacrynic acid
6. Organism: Not applicable
6. Indication: Oedema requiring a loop diuretic where sulfonamide allergy is a concern
6. Line: reserve
6. Note: Chosen for its chemistry rather than its potency, and used with the highest ototoxic risk in the class in mind. [1][2]

Pharmacokinetics

1. Drug: Furosemide
1. Route: Oral/IV/IM
1. Absorption: Average oral bioavailability about 50%
1. Csf Penetration: Not clinically relevant
1. Metabolism: See prescribing reference
1. Elimination: Predominantly renal
1. Half Life: Approximately 1.5 to 2 hours, extending to about 2.6 hours in renal dysfunction
1. Dose Adjust: Renal impairment; monitor electrolytes and fluid status [1]
2. Drug: Bumetanide

2. Route: Oral/IV
2. Absorption: Oral bioavailability closer to 80%
2. Csf Penetration: Not clinically relevant
2. Metabolism: See prescribing reference
2. Elimination: Predominantly renal
2. Half Life: Approximately 1 hour, extending to about 1.3 to 1.6 hours in renal dysfunction
2. Dose Adjust: Renal impairment; monitor electrolytes and fluid status [1]
3. Drug: Torsemide
3. Route: Oral/IV
3. Absorption: Oral bioavailability closer to 80%
3. Csf Penetration: Not clinically relevant
3. Metabolism: Hepatic, principally by CYP2C9 with minor contributions from CYP2C8 and CYP2C18
3. Elimination: Predominantly hepatic - metabolism accounts for roughly 80% of total clearance and urinary excretion for roughly 20% where renal function is normal. The drug still reaches its site of action in the tubular lumen by active proximal secretion, as the rest of the class does
3. Half Life: Approximately 3 to 4 hours, extending to about 5 to 6 hours in renal dysfunction
3. Dose Adjust: Renal impairment; monitor electrolytes and fluid status [1][4]
1. The bioavailability difference between furosemide and the other oral agents is clinically useful: a patient whose oral response is disappointing may respond to an agent with more reliable absorption rather than needing a different class. [1]
2. Elimination is not uniform across the class. Furosemide and bumetanide are cleared predominantly by the kidney, whereas torsemide is cleared predominantly by hepatic metabolism, which is why hepatic impairment and CYP2C9 interactions matter more for torsemide than for the other two. [4]
3. Half-lives are short across the class and lengthen in renal dysfunction, which is part of why fluid status, weight, electrolytes and renal function are monitored periodically rather than once. [1]
4. Baseline auditory testing is described as necessary where large bolus doses at high infusion rates are planned, with periodic sequential monitoring of auditory function afterwards. [2]
5. This page gives no dose regimens by design. Doses depend on indication, renal function, route and previous diuretic exposure, and belong in a prescribing reference used by the treating clinician.

Adverse effects

1. Effect: Electrolyte depletion
1. Detail: Hyponatraemia, hypokalaemia, hypochloraemia, hypomagnesaemia and metabolic alkalosis are the characteristic disturbances of this class. [1]
2. Effect: Calcium and magnesium loss
2. Detail: A direct consequence of blocking the cotransporter and the potassium recycling that supports divalent cation reabsorption. [1]
3. Effect: Hyperuricaemia
3. Detail: Raised urate secondary to loop diuretic use can result in acute gout attacks or flares. [1]
4. Effect: Prerenal azotaemia
4. Detail: Diuretic-induced hypovolaemia can raise urea and creatinine, which is why both are followed during treatment. [2]
1. Effect: Ototoxicity
1. Detail: Described as an adverse effect specific to loop agents and not shared by any other diuretic, presenting as sensorineural hearing loss, tinnitus, vertigo or dizziness, and attributed to ischaemia in the stria vascularis together with inhibition of NKCC1 there.
1. Action: Baseline auditory testing before planned large bolus doses at high infusion rates, with periodic reassessment

during treatment. [2]

2. Effect: Permanent sensorineural hearing loss with ethacrynic acid

2. Detail: Ethacrynic acid has greater ototoxic potential than the rest of the class and has been reported to cause hearing loss that does not recover.

2. Action: Reserve for situations where its non-sulfonamide chemistry is the deciding factor, and weigh the hearing risk explicitly. [1][2]

3. Effect: Precipitation of hepatic encephalopathy in cirrhosis

3. Detail: Severe electrolyte depletion and dehydration from loop therapy can precipitate encephalopathy in patients with cirrhosis, which is a metabolic complication rather than direct liver injury.

3. Action: Follow electrolytes and volume status closely when treating ascites or oedema in liver disease. [5]

1. Drug: Ethacrynic acid

1. Effect: The most ototoxic drug in the group, and the only member that is not a sulfonamide derivative. [1][2]

2. Drug: Loop diuretics as a class

2. Effect: Clinically apparent acute liver injury is exceedingly rare, if it occurs at all, and the class carries the lowest hepatotoxicity likelihood rating. [5]

Contraindications, precautions and interactions

1. Anuria, where no useful tubular response is possible. [1]

2. Hypersensitivity to furosemide, bumetanide, torsemide or to sulfonamides. [1]

3. Hepatic coma and severe electrolyte depletion. [1]

1. Renal impairment, which both increases ototoxic risk and lengthens the half-life of every member of the class. [1]

2. Cirrhosis with ascites, because electrolyte depletion and dehydration can precipitate hepatic encephalopathy. [5]

3. Established gout, given that treatment-related hyperuricaemia can provoke an acute attack. [1]

4. Older patients and those with cardiac or metabolic disease, in whom abrupt fluid shifts and prerenal azotaemia are of particular concern. [2]

1. With: Aminoglycosides and platinum-containing chemotherapy

1. Effect: Both potentiate the ototoxic potential of loop diuretics. [2]

2. With: Digoxin

2. Effect: Diuretic-induced potassium and magnesium loss is the reason digoxin interactions receive specific attention during loop therapy. [1]

3. With: Thiazide-like diuretics

3. Effect: Deliberately combined for sequential nephron blockade in chronic diuretic tolerance, with additive electrolyte risk. [2]

Resistance mechanisms

1. Mechanism: Acute tolerance and the braking phenomenon

1. Detail: Some time after a dose, rebound retention of sodium occurs through increased reabsorption in the distal tubules, blunting the net effect of the drug.

Diminishing response between doses in chronic heart failure

1. Countermeasure: Choosing the correct dose and dosing frequency, attending to the timing of diet, and restricting dietary salt. [2]

2. Mechanism: Chronic tolerance from distal tubular adaptation

2. Detail: With prolonged therapy the kidney adapts structurally in the distal tubule so that sodium retention increases, which reduces the response to a previously adequate regimen.

Long-term loop diuretic therapy

2. Countermeasure: Sequential nephron blockade by co-administering a thiazide-like diuretic, alongside reassessment of the loop agent itself. [2]

- 3. Mechanism: Proximal tubular sodium reabsorption escaping the block
- 3. Detail: Sodium reclaimed upstream of the loop never reaches the blocked transporter, so the drug cannot act on it.
- Diuretic resistance in decompensated heart failure
- 3. Countermeasure: Combining acetazolamide with a loop diuretic has demonstrated effectiveness in diuretic resistance because it acts on proximal sodium reabsorption. [2]
- 4. Mechanism: Incomplete or variable oral absorption
- 4. Detail: An apparently resistant patient may simply be absorbing too little drug, since oral bioavailability differs substantially between members of the class.
- Poor oral response to furosemide with a good intravenous response
- 4. Countermeasure: Reassessment of route, or a change to an agent with higher oral bioavailability. [1]
- Stewardship Note: Failure of a diuretic to increase urine output is described as grounds to stop the agent and look for undiagnosed renal pathology, rather than as an automatic signal to escalate.

Comparison tables

Title: Comparing the loop diuretics

Caption: Chemistry, absorption and half-life are what separate these agents. Actual therapy and dosing belong to the treating clinician working from a current prescribing reference.

- 1. Key: drug
- 1. Label: Drug
- 1. Is Row Header: true
- 2. Key: sulfonamide
- 2. Label: Sulfonamide derivative?
- 3. Key: bioavailability
- 3. Label: Oral bioavailability
- 4. Key: halflife
- 4. Label: Half-life (normal renal function)
- 5. Key: note
- 5. Label: Distinguishing point
- 1. Drug: Furosemide
- 1. Sulfonamide: Yes
- 1. Bioavailability: About 50% on average
- 1. Halflife: About 1.5 to 2 hours
- 1. Note: The reference agent; variable absorption is its main limitation [1]
- 2. Drug: Bumetanide
- 2. Sulfonamide: Yes
- 2. Bioavailability: Closer to 80%
- 2. Halflife: About 1 hour
- 2. Note: Shortest half-life, more reliable oral absorption [1]
- 3. Drug: Torsemide
- 3. Sulfonamide: Yes
- 3. Bioavailability: Closer to 80%
- 3. Halflife: About 3 to 4 hours
- 3. Note: Longest acting of the sulfonamide-derived agents [1]
- 4. Drug: Ethacrynic acid
- 4. Sulfonamide: No
- 4. Bioavailability: See prescribing reference
- 4. Halflife: See prescribing reference

4. Note: Option in sulfonamide allergy; the most ototoxic member of the class [1][2]

High-yield exam pearls

1. Pearl: Ototoxicity is the one adverse effect no other diuretic shares.

1. Why: It is described as a specific adverse effect of loop agents alone, mediated by ischaemia in the stria vascularis and by inhibition of the NKCC1 channel there, and it is potentiated by aminoglycosides and platinum-containing chemotherapy. [2]

2. Pearl: The route and speed of administration matter more than the total exposure for hearing.

2. Why: Deafness and tinnitus arise most frequently with large bolus doses given in acute settings, because the toxicity follows the peak serum concentration rather than cumulative use. [2]

3. Pearl: Ethacrynic acid is the escape route in sulfonamide allergy.

3. Why: It is the only member that is not a sulfonamide derivative, which makes it the safer choice where a sulfonamide allergy is a concern, though it is also the most ototoxic agent in the group. [1][2]

4. Pearl: Loops lose calcium and magnesium; thiazides retain calcium.

4. Why: Blocking the Na-K-2Cl cotransporter removes the recycling of potassium that sustains the gradient for divalent cation reabsorption, so calcium and magnesium are lost - the mirror image of the thiazide effect. [1][3]

5. Pearl: Swapping furosemide for bumetanide or torsemide is partly an absorption decision.

5. Why: Furosemide bioavailability averages around 50% and is variable, whereas bumetanide and torsemide are closer to 80%, so an inadequate oral response is not always an inadequate dose. [1]

6. Pearl: A gout flare during decongestion is often drug-related.

6. Why: Hyperuricaemia secondary to loop diuretic use can precipitate acute gout attacks, so a new monoarthritis in a patient being diuresed deserves that explanation before others. [1]

Common exam traps

1. Trap: "Loop diuretic hearing loss is always permanent."

1. Correction: The ototoxicity described for this class is characteristically reversible, arising from stria vascularis ischaemia and NKCC1 inhibition. Ethacrynic acid is the member specifically linked to permanent sensorineural hearing loss. [2][1]

2. Trap: "A sulfonamide allergy rules out every loop diuretic."

2. Correction: There is only a low potential for cross-allergenicity, and ethacrynic acid is not a sulfonamide derivative at all, so a loop agent remains available. [1]

3. Trap: "If the diuretic stops working, the answer is always a bigger dose."

3. Correction: Tolerance has two distinct forms. Acute tolerance produces a braking phenomenon of rebound sodium retention, while chronic tolerance reflects structural adaptation of the distal tubule; salt restriction, dose timing and sequential nephron blockade are all recognised responses. [2]

4. Trap: "Loop diuretics are hepatotoxic in cirrhosis."

4. Correction: Clinically apparent liver injury from loop diuretics is exceedingly rare if it occurs at all. The hazard in cirrhosis is precipitation of hepatic encephalopathy through electrolyte depletion and dehydration, which is a metabolic consequence rather than direct liver toxicity. [5]

Self-test questions

1. Question: Which transporter do loop diuretics inhibit, and where?

1. The Na⁺/Cl⁻ channel in the distal convoluted tubule

2. The Na-K-2Cl (NKCC2) cotransporter at the apical membrane of the thick ascending limb

3. The epithelial sodium channel in the cortical collecting duct

4. The sodium-potassium ATPase on the basolateral membrane of the proximal tubule

1. Explanation: Loop diuretics compete with chloride to bind the Na-K-2Cl cotransporter on the apical membrane of the thick ascending limb, inhibiting sodium and chloride reabsorption at the segment that handles a large fraction of the filtered load. [1]

2. Question: Why do loop diuretics cause loss of calcium and magnesium?

1. They chelate divalent cations within the tubular lumen
2. They inhibit parathyroid hormone secretion
3. Blocking the cotransporter prevents the potassium recycling that sustains the gradient driving calcium and magnesium reabsorption
4. They increase intestinal excretion of both ions

2. Explanation: Blockade of the Na-K-2Cl cotransporter leaves potassium unable to return to the lumen, and the resulting loss of the electrical driving force means calcium and magnesium ions are lost with the urine. [1]

3. Question: In which circumstance is loop diuretic ototoxicity most likely?

1. After many months of low-dose oral therapy
2. After large bolus doses given in an acute setting, producing high serum concentrations
3. Only when the drug is combined with a thiazide
4. Only in patients with normal renal function

3. Explanation: Deafness and tinnitus most frequently follow large bolus doses in acute settings, and the toxicity is attributed to the higher serum concentrations reached. Renal impairment and concurrent aminoglycosides or platinum agents raise the risk further. [2][1]

4. Question: Which loop diuretic is not a sulfonamide derivative?

1. Furosemide
2. Bumetanide
3. Torsemide
4. Ethacrynic acid

4. Explanation: Ethacrynic acid stands apart chemically from the other members and is therefore the safer option where a sulfonamide allergy is a concern. It is, however, the most ototoxic agent in the group and has been linked to permanent sensorineural hearing loss. [1][2]

5. Question: What does the term braking phenomenon describe in diuretic therapy?

1. The fall in blood pressure that limits further diuresis
2. Rebound sodium retention from increased distal tubular reabsorption after a dose, representing acute tolerance
3. The ceiling above which further drug produces no additional urine output in any patient
4. Progressive deafness with repeated dosing

5. Explanation: Acute tolerance develops some time after a dose and causes rebound retention of sodium through increased reabsorption in the distal tubules. Correct dose selection, dosing frequency, timing of diet and dietary salt restriction are the described countermeasures. [2]

6. Question: A patient established on oral furosemide has a poor response, but responds well to the intravenous route. What is the most likely pharmacokinetic explanation?

1. Furosemide is destroyed by gastric acid
2. Furosemide oral bioavailability averages about 50%, lower and more variable than bumetanide or torsemide at around 80%
3. Furosemide undergoes extensive hepatic first-pass conversion to an inactive metabolite
4. Oral furosemide is not absorbed at all in heart failure

6. Explanation: Average oral bioavailability of furosemide is around 50%, whereas bumetanide and torsemide reach approximately 80%. That difference is one reason a change of agent or of route can restore a response without any change in the underlying disease. [1]

7. Question: Which of the following is a recorded contraindication to loop diuretic therapy?

1. Anuria

2. Hypercalcaemia

3. Atrial fibrillation

4. Type 2 diabetes mellitus

7. Explanation: Anuria is listed alongside hypersensitivity to the drug or to sulfonamides, hepatic coma and severe electrolyte depletion. Hypercalcaemia is not a contraindication, since the class increases rather than reduces calcium excretion. [1]

Frequently asked questions

1. Question: Why are loop diuretics stronger than thiazides?

1. Answer: Because the thick ascending limb reabsorbs far more of the filtered sodium than the distal convoluted tubule does. Blocking the larger segment removes more sodium, which is why loop agents are used for decongestion and thiazides for sustained blood pressure control. [1][3]

2. Question: Is loop diuretic hearing loss reversible?

2. Answer: The ototoxicity described for the class is characteristically reversible and is attributed to stria vascularis ischaemia and NKCC1 inhibition. Ethacrynic acid is the exception specifically associated with permanent sensorineural hearing loss. [2][1]

3. Question: Can a patient with a sulfonamide allergy receive a loop diuretic?

3. Answer: Cross-allergenicity between sulfonamide antibiotics and these diuretics carries only a low potential, and ethacrynic acid is not a sulfonamide derivative at all. The decision rests with the treating clinician using the individual allergy history. [1]

4. Question: What is done when a loop diuretic seems to stop working?

4. Answer: The recognised approaches are reassessing dose and dosing frequency, attending to diet timing and salt restriction, adding a thiazide-like diuretic for sequential nephron blockade, or combining with acetazolamide to address proximal sodium reabsorption. Absent urine output is instead a reason to stop and look for renal pathology. [2]

5. Question: Do loop diuretics damage the liver?

5. Answer: Clinically apparent acute liver injury from this class is exceedingly rare, if it occurs at all. The liver-related danger is indirect: electrolyte depletion and dehydration can precipitate hepatic encephalopathy in cirrhosis. [5]

References

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