

// THE POTASSIUM DILEMMA

Why does hyperkalaemia stop heart failure and kidney therapy?

Several cornerstone heart and kidney therapies act on the renin-angiotensin-aldosterone system and raise potassium. When potassium rises, therapy is often cut back, and patients lose the protection it was meant to give.

Andreas Fercher, CEO, Elyte Diagnostics · 15 October 2026 · 6 min read

SUMMARY

RAAS inhibitors reduce death and hospitalisation in heart failure and slow kidney decline, but they raise serum potassium. After a hyperkalaemia episode, therapy was reduced in 25–57% of patients across four European countries. Patients whose therapy was reduced spent more time in hospital, stopping therapy has not been shown to protect the kidney, and KDIGO 2024 advises lowering potassium before reducing or stopping these drugs.

25–57%

of patients had RAASi reduced after hyperkalaemia

Germany, Spain, Sweden, UK [1]

1 in 25

fewer patients hospitalised within six months when therapy was maintained

Four-country study, observational [1]

4.0–4.5 mmol/L

the potassium range with the lowest mortality risk

CKD Prognosis Consortium [5]

The potassium dilemma

ACE inhibitors, angiotensin receptor blockers, sacubitril/valsartan, and mineralocorticoid receptor antagonists are the backbone of treatment in heart failure with reduced ejection fraction and in albuminuric chronic kidney disease. Each reduces aldosterone production or action, and with it potassium secretion in the distal nephron. Chronic kidney disease, heart failure, and diabetes impair potassium handling further.

The dilemma follows from this. The patients who gain most from these drugs are also those most likely to develop hyperkalaemia, and withdrawing therapy in response may lower the immediate potassium risk while raising the longer-term cardiovascular and kidney risk.

The risk is common and it recurs. In a Danish population cohort of 31,649 people with newly diagnosed heart failure, 25% had a potassium value above 5.0 mmol/L within the first year, rising to 44% among those with stage 4 kidney disease [6]. After a first episode, a second followed within six months in 40% of people with chronic kidney disease and 49% of people with heart failure [7].

What happens after an episode

A recent study across four European health systems followed 40,059 adults with chronic kidney disease, heart failure, or both, who had a hyperkalaemia episode while taking a RAAS inhibitor [1]. The researchers compared prescriptions in the 120 days before and after the episode. Therapy counted as reduced if all RAAS inhibitors were stopped, if a class was dropped, or if a dose was cut by a quarter or more.

The share of patients whose therapy was reduced was 44.4% in Germany, 24.8% in Spain, 34.7% in Sweden, and 57.1% in the United Kingdom. In the UK, more than a third of patients, 37.9%, had every RAAS inhibitor stopped.

Figure 1. Share of patients whose RAAS-inhibitor therapy was reduced after a hyperkalaemia episode [1]



Reduced = all RAASi stopped, a class dropped, or a dose cut by 25% or more, comparing the 120 days after the episode with the 120 days before. Hyperkalaemia definitions differed between countries. Bars are scaled from 0 to 60%.

What follows a reduction

In Sweden, 41.9% of 20,824 patients had their therapy reduced after hyperkalaemia [2]. When those patients were matched with similar patients whose therapy was maintained, the reduced group spent 33.6 days in hospital per person-year over the following six months, against 24.7 days in the maintained group.

Across the four-country study, maintaining therapy was associated with one fewer first hospitalisation within six months for every 25 patients [1]. In a separate US cohort, the six-month rate of heart failure hospitalisation, heart failure emergency visits, or progression to kidney failure was 17.5% when therapy was stopped and 18.2% when it was reduced, against 10.6% when it was maintained or increased [3]. A meta-analysis of kidney disease cohorts found a 48% higher risk of death when treatment was stopped because of hyperkalaemia [4].

These are observational findings. Patients whose therapy is reduced are often older, frailer, or more acutely ill, and some reductions follow a decline that would have happened anyway. The studies adjusted for this in different ways, but none can fully remove it. Most of the European data cited here were funded by a manufacturer of potassium binders. The direction of the evidence is nonetheless consistent: across countries and study designs, reducing therapy after hyperkalaemia is associated with more hospital use and higher mortality.

Why the decision is hard

Hyperkalaemia rarely causes symptoms before it affects the heart's rhythm. The relationship between potassium and mortality is U-shaped: risk is lowest between about 4.0 and 4.5 mmol/L and rises on both sides [5]. At 5.5 mmol/L, the risk of death was 22% higher than at 4.2 mmol/L in an analysis of more than 1.2 million people [5].

A clinician who sees a single raised value therefore faces a trade-off: an uncertain short-term risk of arrhythmia against a longer-term loss of protection. The potassium result is immediate and visible, while the benefit lost by cutting therapy is delayed, so the measurable short-term hazard tends to drive the decision, often without an equally reliable process for rechecking potassium and restarting treatment. Once a patient is stable, guidelines schedule potassium checks weeks to months apart [8, 9]. Between those checks, the clinician has little information about where the patient's potassium is heading, and reducing the dose can look like the safer choice.

What the guidelines now say

“Hyperkalemia associated with use of RASi can often be managed by measures to reduce the serum potassium levels rather than decreasing the dose or stopping RASi.” KDIGO 2024 [8]

The KDIGO algorithm for potassium above 5.5 mmol/L starts with correctable causes, then diuretics, bicarbonate, and potassium exchange agents, and places a dose reduction or discontinuation last, noting that discontinuation is associated with more cardiovascular events [8]. The Austrian Society of Nephrology's 2026 consensus goes further for kidney disease: stopping RAAS inhibition because of hyperkalaemia is indicated only when serum potassium stays above 6.0 mmol/L despite all potassium-lowering options [10].

What stopping therapy does and does not achieve

Stopping a RAS inhibitor lowers the immediate potassium risk. The evidence does not show that it protects the kidney. In the STOP-ACEi trial, 411 patients with advanced chronic kidney disease (eGFR below 30 ml/min/1.73 m²) were randomised to stop or continue therapy; after three years, eGFR did not differ between the groups (12.6 against 13.3 ml/min/1.73 m²), and kidney failure or kidney replacement therapy occurred in 62% and 56% respectively [11].

Two target trial emulations point the same way. In 2,305 Japanese patients who developed hyperkalaemia (5.5 mmol/L or above) on RAS inhibitors, stopping was associated with fewer episodes of severe hyperkalaemia but a 16% higher risk of death, with no difference in kidney outcomes [12]. In 1,434 French patients with advanced kidney disease, stopping was associated with twice the three-year risk of major cardiovascular events and a 34% higher risk of starting kidney replacement therapy [13].

PRACTICE POINTS

- A single raised potassium value is a reason to look again, not automatically a reason to stop protective therapy. Very high or rapidly rising values, symptoms, acute kidney injury, or ECG changes need immediate action.
- When therapy has been reduced, a plan to review and restart it matters as much as the reduction itself.
- The decision depends on knowing where potassium is heading between visits. The next paper in this series looks at what the guidelines say about how often to check it.

REFERENCES

1. Svensson MK, Fischereder M, Kalra PR, et al. Estimated number needed to treat to avoid a first hospitalization by maintaining instead of reducing RAASi therapy after hyperkalemia. *Kidney360*. 2024;5:1813–1823. doi:10.34067/KID.0000000000000561. Funded by AstraZeneca.
2. Svensson MK, Murohara T, Lesén E, et al. Hyperkalaemia-related reduction of RAASi treatment associates with more subsequent inpatient care. *Nephrol Dial Transplant*. 2024;39(8):1258–1267. doi:10.1093/ndt/gfae016. Funded by AstraZeneca.
3. Kanda E, Rastogi A, Murohara T, et al. Clinical impact of suboptimal RAASi therapy following an episode of hyperkalemia. *BMC Nephrol*. 2023;24:18. doi:10.1186/s12882-022-03054-5. Funded by AstraZeneca.
4. Tang C, Wen XY, Lv JC, et al. Discontinuation of renin-angiotensin system inhibitors and clinical outcomes in chronic kidney disease: a systematic review and meta-analysis. *Am J Nephrol*. 2023;54(5–6):234. doi:10.1159/000531000.
5. Kovesdy CP, Matsushita K, Sang Y, et al. Serum potassium and adverse outcomes across the range of kidney function: a CKD Prognosis Consortium meta-analysis. *Eur Heart J*. 2018;39:1535–1542. doi:10.1093/eurheartj/ehy100.
6. Thomsen RW, Nicolaisen SK, Hasvold P, et al. Elevated potassium levels in patients with congestive heart failure: occurrence, risk factors, and clinical outcomes. *J Am Heart Assoc*. 2018;7:e008912. doi:10.1161/JAHA.118.008912. Funded in part by AstraZeneca.
7. Adelborg K, Nicolaisen SK, Hasvold P, et al. Predictors for repeated hyperkalemia and potassium trajectories in high-risk patients: a population-based cohort study. *PLoS One*. 2019;14(6):e0218739. doi:10.1371/journal.pone.0218739. Funded in part by AstraZeneca.
8. KDIGO CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int*. 2024;105(4S):S117–S314. doi:10.1016/j.kint.2023.10.018.
9. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021;42(36):3599–3726. doi:10.1093/eurheartj/ehab368.
10. Lindner G, Windpessl M, Pirklbauer M, et al. Konsensusempfehlungen zur Diagnose und Therapie der Hyperkaliämie der Österreichischen Gesellschaft für Nephrologie 2026. *Wien Klin Wochenschr*. 2026;138(Suppl 2):S11–S48. doi:10.1007/s00508-026-02708-w.
11. Bhandari S, Mehta S, Khwaja A, et al. Renin-angiotensin system inhibition in advanced chronic kidney disease. *N Engl J Med*. 2022;387(22):2021–2032. doi:10.1056/NEJMoa2210639. Funded by the National Institute for Health Research and the Medical Research Council.
12. Hashimoto N, Sakaguchi Y, Hattori K, et al. Discontinuing renin-angiotensin system inhibitors after incident hyperkalemia and clinical outcomes: target trial emulation. *Hypertens Res*. 2025;48(7):2034–2044. doi:10.1038/s41440-025-02218-8.
13. Maisons V, Costes-Albrespic M, Liabeuf S, et al. Impact of renin-angiotensin system inhibitors discontinuation in advanced chronic kidney disease: a target trial emulation. *Clin Kidney J*. 2026;19(10):sfag304. doi:10.1093/ckj/sfag304.

AF

Andreas Fercher, CEO, Elyte Diagnostics, Graz

Published 15 October 2026 · Last updated 20 October 2026

Disclosure: Elyte Diagnostics is developing a home potassium test. This paper does not discuss that product.