Renal Artery Stenosis

- 10% are **Fibromusclular dysplasia**, mainly young women
- In >90% cases, Atherosclerosis is the underlying cause
- Most atherosclerotic lesions are ostial
- Many patients will also have atherosclerotic disease in intra-renal vessels "**ischaemic nephropathy**"
- HTN occurs because the kidney detects reduced perfusion and sends out ***Renin*** to activate the Renin-Angiotensin pathway to ↑ BP in an attempt to ↑ kidney perfusion.

- These patients typically have a high burden of systemic artherosclerotic disease elsewhere & at ↑ risk of CVD → aggressive secondary prevention of CVD. Including **aspirin**.

Some things to consider

- Rise in creatinine with ACEi is more pronounced in the presence of diuretic-induced volume depletion. *Which might have been some of the issue for Bob*
- If >20% rise then RAS stenosis should be considered
- A similar rise can occur with intrarenal vascular disease e.g. hypertensive nephrosclerosis and in hypovolaemic patients
- When RAS stenosis reaches a critical level, reduction in BP with any agent can reduce eGFR
- Correction of stenosis with angioplasty may help…
- Those taking RAS blockade have significantly lower mortality

Nephrol Dial Transplant. 2012 Apr;27(4):1403-9. doi: 10.1093/ndt/gfr496. Epub 2011 Oct 12.

Dispelling the myth: the use of renin–angiotensin blockade in atheromatous renovascular disease

Constantina Chrysochou [1], Robert N Foley, James F Young, Kaivan Khavandi, Ching M Cheung, Philip A Kalra

Affiliations + expand

PMID: 21993376 DOI: 10.1093/ndt/gfr496

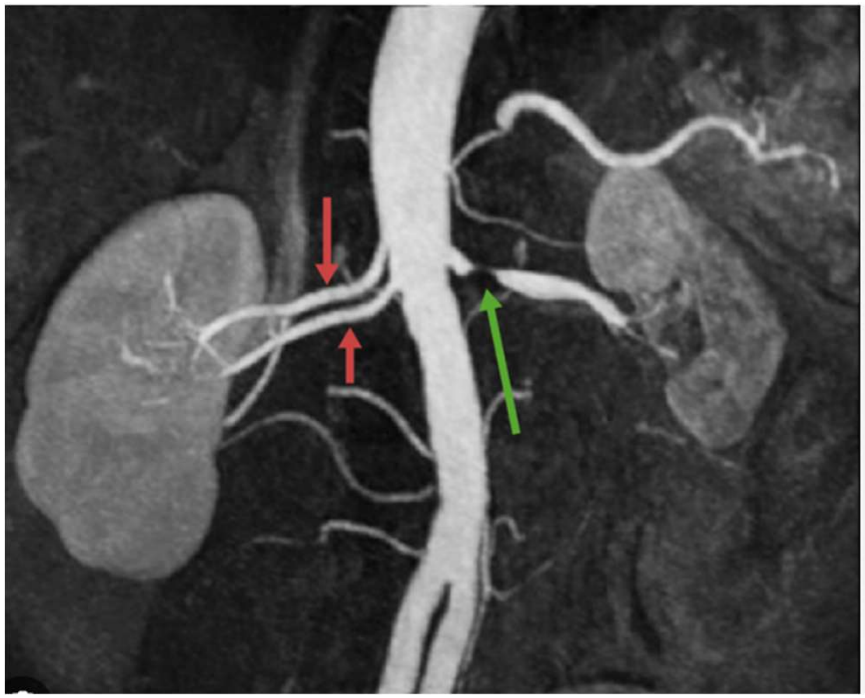
Prospective Observational Study
621 patients with atherosclerotic renovascular disease (ARVD)
Treatment with ACE-I and ARBs was associated with the following effects:

1. Patients with bilateral and significant RAS were able to tolerate these medications safely in most cases.

2. On multivariate time-adjusted analysis, patients receiving RAB were significantly less likely to die ($P < 0.02$)

3. In a small sub-group of patients who were unable to tolerate RAB, renal artery revascularization enabled safe re-commencement of these drugs.

This is Bob’s CTA. Will he benefit from renal artery angioplasty?



UpToDate Guidance

Probably Not, as HTN is not truly resistant

We suggest revascularization in addition to medical therapy rather than medical therapy alone in patients with severe unilateral atherosclerotic renal artery stenosis (typically defined as a stenosis >70 percent) if they have a **high likelihood** of benefitting from intervention, defined by the presence of one or more of the following:

1.Recurrent flash pulmonary edema

2. True resistant hypertension with uncontrolled blood pressure

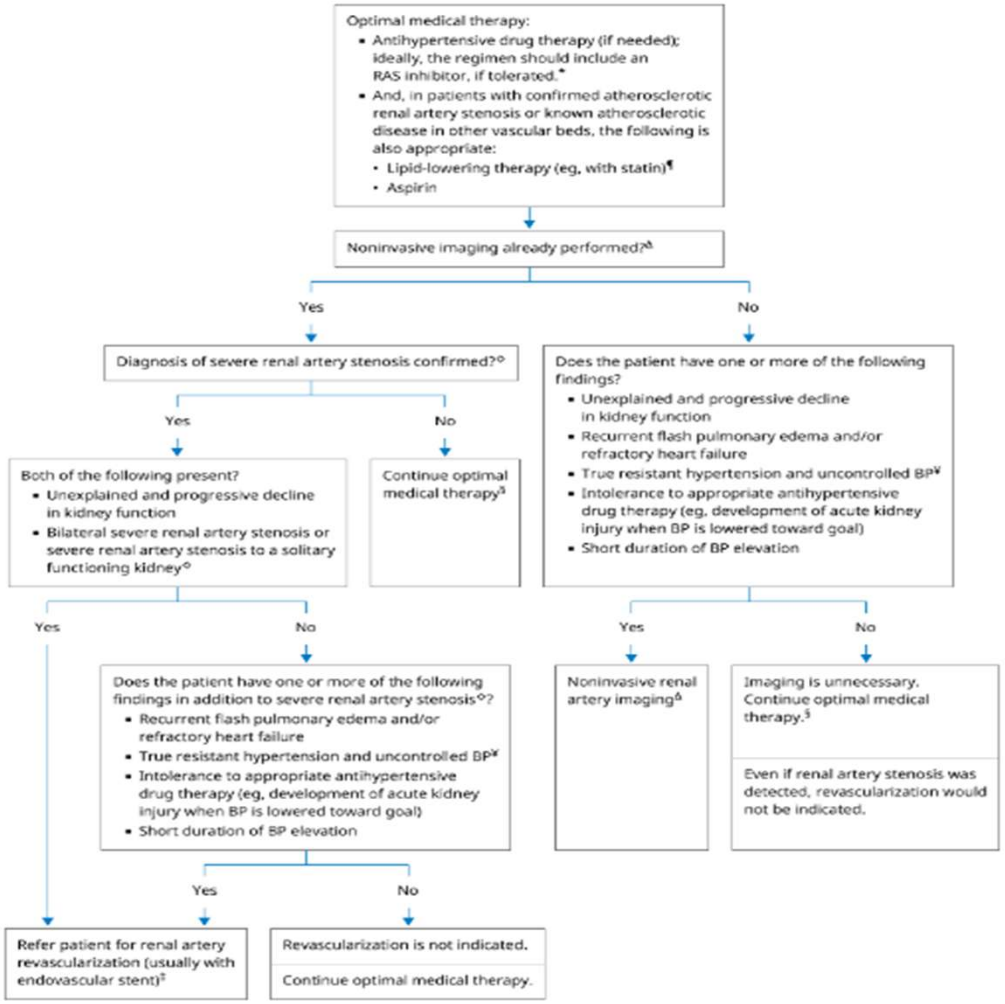
3. Patients who **develop acute kidney injury** and/or persistent hyperkalemia when blood pressure is reduced toward goal (even with antihypertensive medications **other** than an ARB or ACE inhibitor) may benefit from revascularization.

4. A short duration of blood pressure elevation prior to the diagnosis of renovascular disease (**eg, <1 year)** predicts a fall in blood pressure after renal revascularization.

Among other patients with unilateral atherosclerotic renal artery stenosis (ie, patients with none of the above listed indications and/or nonsevere stenosis), we recommend **against** revascularization.

In general, the RAS inhibitor can be continued if the eGFR declines by less than 30 to 40% and does not continue to worsen, and if the patient's BP is not lowered excessively (eg, systolic pressure <110 mmHg)

Severe = >70%



UpToDate Guidance

RAS Blockade in CKD –General comments

"No, taking an ACE inhibitor won't hurt your poker game."

They're good ☺

Nephrologists

The NEW ENGLAND JOURNAL of MEDICINE

CURRENT ISSUE SPECIALTIES TOPICS

ORIGINAL ARTICLE

Renin–Angiotensin System Inhibition in Advanced Chronic Kidney Disease

Authors: Sunil Bhandari, Ph.D., Samir Mehta, M.Sc., Arif Khwaja, Ph.D., John G.F. Cleland, M.D., Natalie Ives, M.Sc., Elizabeth Brettell, B.Sc., Marie Chadburn, Ph.D., and Paul Cockwell, Ph.D., for the STOP ACEi Trial Investigators* Author Info & Affiliations

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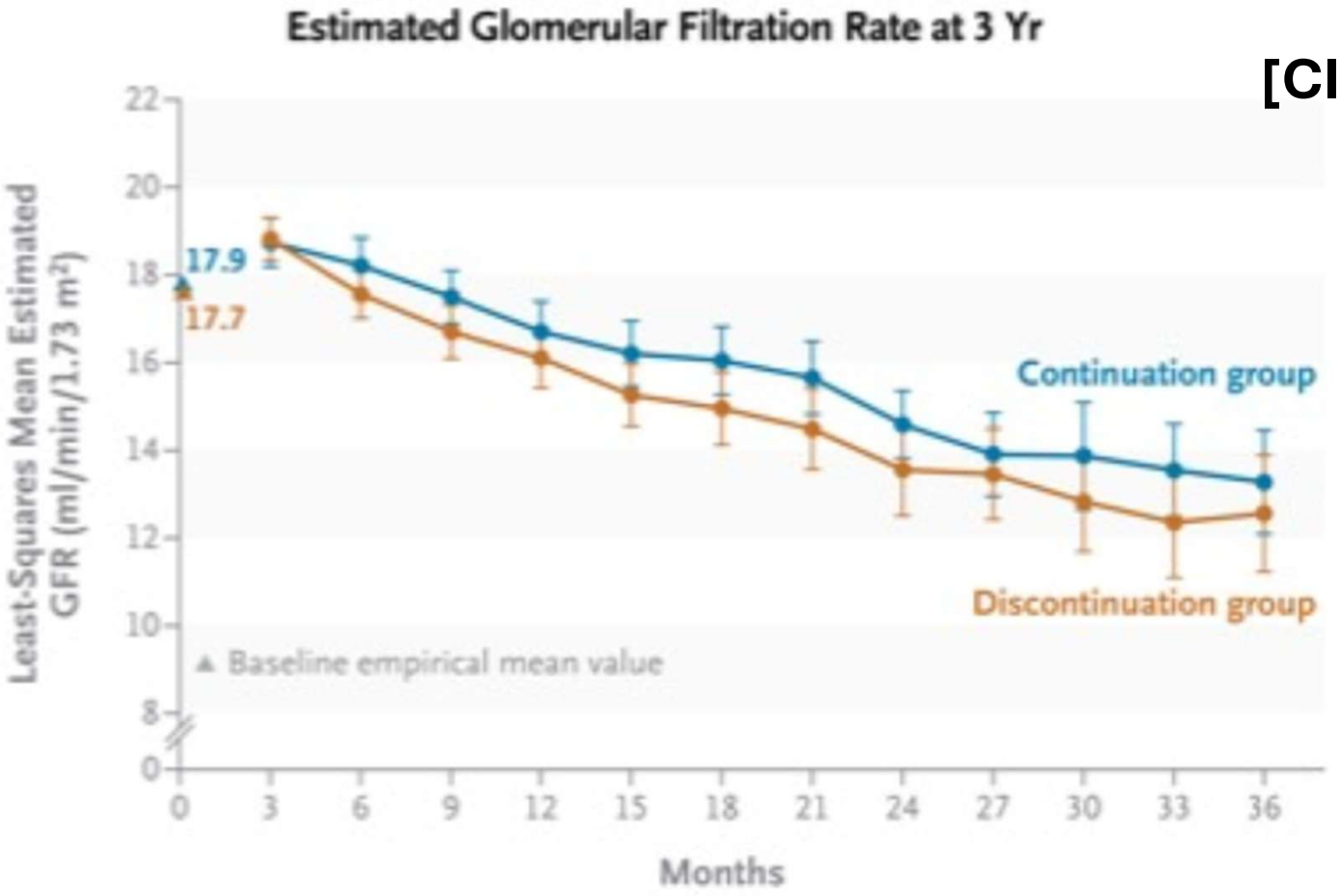
Multicenter, open-label trial
Patients with advanced and progressive CKD (Egfr<30) randomly assigned to continue or stop RAS blockade drugs
Primary outcome: eGFR at 3 years
Secondary outcomes: ESKD, hospitalisation etc.
Prespecified subgroups were defined according to age, eGFR, type of diabetes, mean arterial pressure, and proteinuria
Enrolled 411 patients in 39 UK centres

Cause of CKD

Characteristic	RAS Inhibitor Discontinuation Group (N=206)	RAS Inhibitor Continuation Group (N=205)
Source of chronic kidney disease — no. (%)‡		
Glomerulonephritis: primary, secondary, or multisystem	45 (22)	31 (15)
Tubulointerstitial disease	3 (1)	3 (1)
Hereditary including ADPKD	42 (20)	39 (19)
Renal vascular disease or hypertension	32 (16)	36 (18)
Diabetic nephropathy	43 (21)	44 (21)
Other cause	21 (10)	30 (15)
Unknown	37 (18)	34 (17)
Blood pressure — mm Hg		
Median systolic (IQR)	136 (129 to 147)	138 (126 to 147)
Median diastolic (IQR)	77 (70 to 82)	77 (70 to 82)

[CI], −2.5 to 1.0; P=0.42),



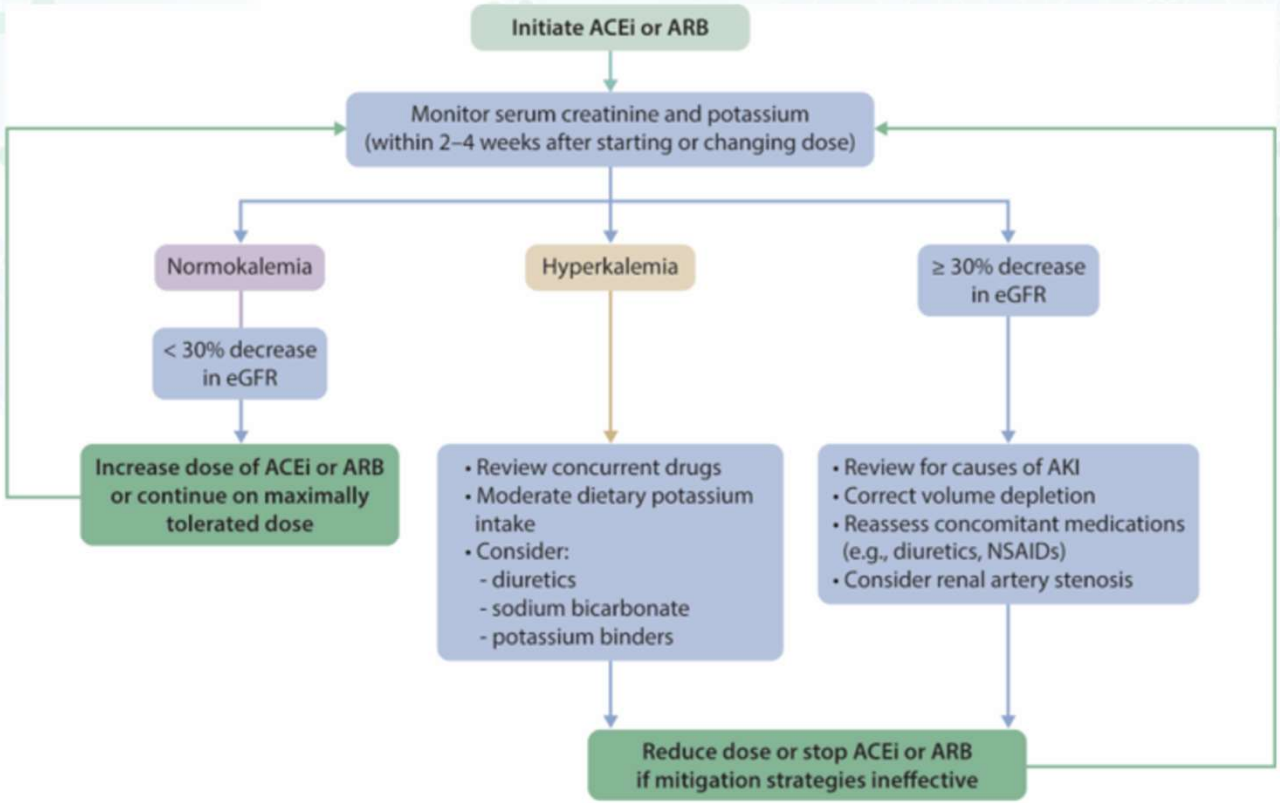
Serious Adverse Events

Variable	Discontinuation Group	Continuation Group
Patients — no./total no. (%)	107/206 (52)	101/205 (49)
No. of events	237	253
No. of cardiovascular events	108	88

trial did not have sufficient power to investigate the effect of the discontinuation of RAS inhibitors on cardiovascular events or mortality. However, because our findings are consistent with a lack of advantage for such discontinuation with respect to kidney function, there is little rationale to conduct a larger randomized trial to investigate cardiovascular safety.

MANAGEMENT – ACUTE CHANGES IN eGFR

Initial dips in eGFR are expected following initiation of hemodynamically active therapies, including both RASI and SGLT2i. GFR reductions of ≥30% from baseline exceed the expected variability and warrant evaluation.



Case 5

Grace

62-year-old female

History of hypertension -18 years

Two prior hospitalizations with AKI:

- 5 years ago: septic shock (peak 450, recovered to baseline Cr 122)
- 2 years ago: volume depletion from gastroenteritis (Cr peaked at 230, returned to 123)

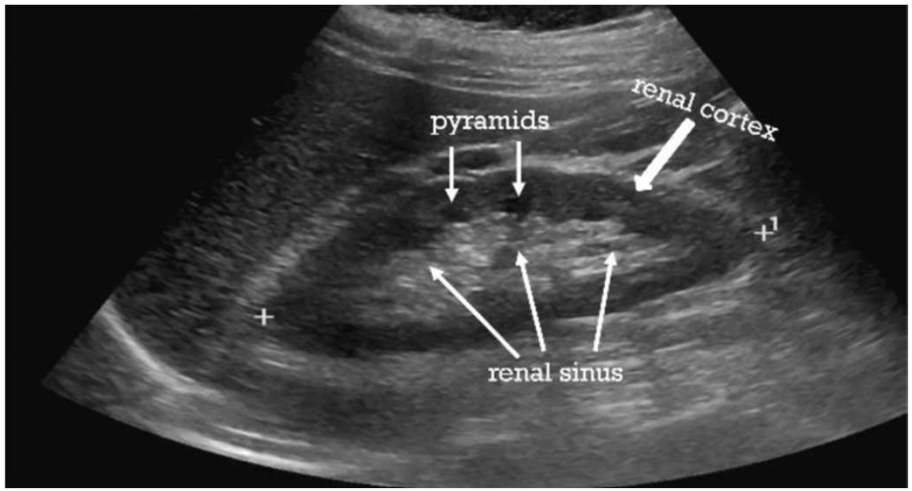
No diabetes

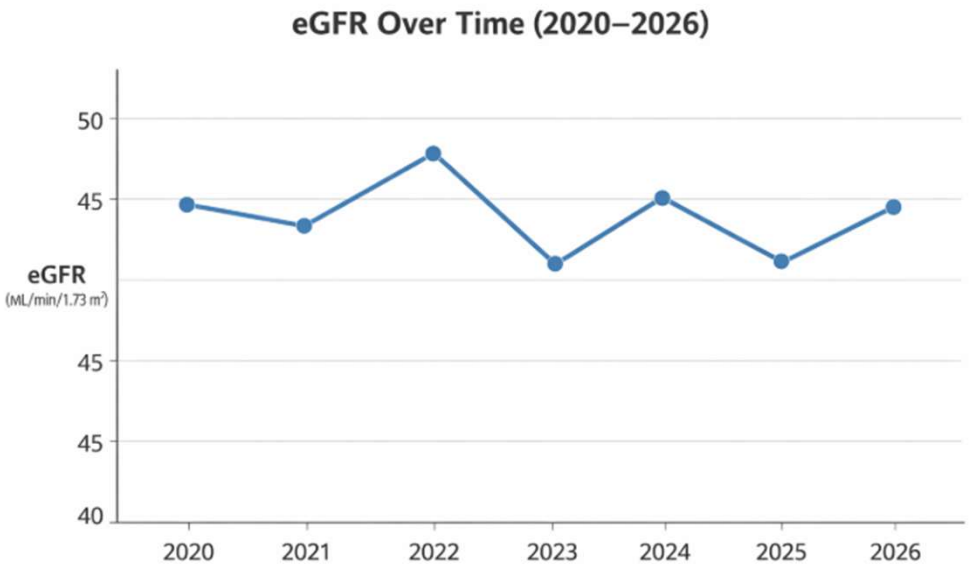
Former NSAID use (intermittent for arthritis, stopped 2 y ago)

- **Medications:**
- Losartan 100 mg daily
- Chlorthalidone 12.5 mg daily
- Atorvastatin 20 mg daily

Vitals:
BP: 132/78 mmHg
HR: 70 bpm
BMI: 27 kg/m^2

Renal Ultrasound:
Mild bilateral cortical thinning
Normal kidney size
No hydronephrosis





Test	Current	1 Year Ago	3 Years Ago
Creatinine	128 umol/L	131	125
eGFR	44 mL/min/1.73m^2	43	43
Urine ACR	7 mg/mmol	8	5
Potassium	4.8 mmol/L	4.7	4.6
Bicarbonate	23 mmol/L	24	23

Urinalysis:
1+ protein
No hematuria
No active sediment