

# PERSPECTIVES IN HYPERTENSION

## Pros and cons of lower systolic blood pressure targets in chronic kidney disease

MARIO FUNES HERNANDEZ

University of Minnesota, Division of Nephrology and Hypertension, Minneapolis, MN, USA

TARA I. CHANG

Stanford University School of Medicine, Division of Nephrology, Stanford, CA, USA



Knowing what systolic blood pressure (SBP) to target for patients with chronic kidney disease (CKD) can be confusing. The 2021 KDIGO guideline tells us to aim for SBP <120 mm Hg<sup>1</sup>, the 2025 ESC guideline says 120–129 mm Hg<sup>2</sup>, and the 2025 AHA/ACC guidelines splits the difference with a recommendation to target SBP <130 mm Hg “with encouragement to achieve SBP <120 mm”.<sup>3</sup> Part of the confusion stems from the fact that the evidence for lower SBP targets is strong for cardiovascular (CV) outcomes, mixed for kidney outcomes, and highly dependent on how we measure blood pressure (BP).

All BP-target trials used standardized office BP, where the patient rests quietly for 5 minutes with attention paid to proper positioning and correct cuff size before  $\geq 2$  BP readings are taken and averaged. Standardized BP readings can be 5–15 mm Hg lower than the typical rushed clinic BP. Because intra-individual variability is so large, we cannot just subtract a fixed number from non-standardized measurements and call it standardized. If we try to treat to SBP <120 using non-standardized readings, we are at high risk of overtreatment that could lead to adverse clinical events.

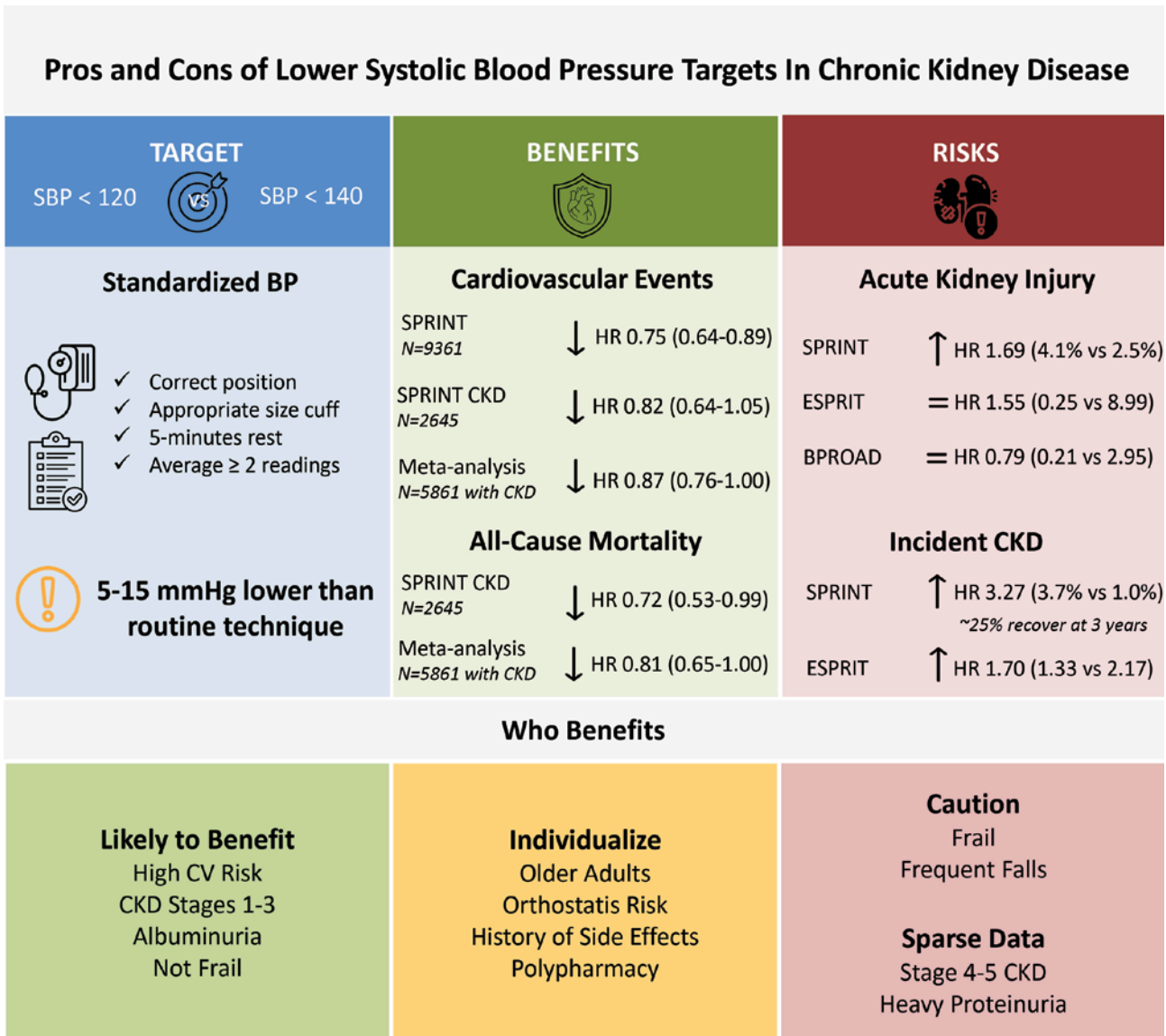
Now back to the different SBP targets among the different guidelines. The differences are not just academic; it changes medication burden, monitoring, and adverse event risk in clinical practice. Notably, the findings from recent trials

are actually fairly consistent: lower (<120 mm Hg) SBP targets reduce CV events and likely reduce mortality overall, and the benefits in CKD are relatively similar to the general population.

The first trial to show a benefit of targeting SBP<120 was SPRINT, which randomized high-CV risk adults to SBP <120 vs <140 mm Hg and intentionally enriched for CKD.<sup>4</sup> In the full cohort, intensive control reduced the primary composite CV outcome (hazard ratio [HR] 0.75, 95% confidence interval [CI] 0.64–0.89). In the CKD subgroup (n=2,645; mean estimated glomerular filtration rate [eGFR] ~48 mL/min/1.73m<sup>2</sup>), the effect size was similar (HR 0.82, CI 0.64–1.05; no significant interaction), and all-cause mortality was lower in the SBP<120 group (HR 0.72, CI 0.53–0.99).<sup>4</sup> The catch: SPRINT excluded diabetes, eGFR <20, and heavy proteinuria (>1 g/day).

Since SPRINT, there have been several additional randomized trials that extended the benefits of targeting SBP<120 mm Hg versus < 140 mm Hg on CV events to older adults<sup>5</sup> and to patients with diabetes.<sup>6,7</sup> However, in all these trials the proportion of patients with CKD was very low (2–8%). A meta-analysis pooling ~5,861 CKD patients across six randomized trials found lower BP targets reduced CV events (RR 0.87, p=0.05) and suggested lower all-cause mortality (RR 0.81, p=0.051).<sup>8</sup>

Figure 1



But what about kidney outcomes? For patients with baseline CKD, the results are mostly neutral. In SPRINT's CKD subgroup, the prespecified kidney outcome (≥50% eGFR decline or kidney failure) did not differ between treatment groups, and while there was earlier eGFR decline, the differences were attenuated after the first months, consistent with a hemodynamic effect.<sup>4,5</sup> ESPRIT reported more kidney outcomes in the intensive group, driven mainly by sustained ≥40% eGFR decline; almost nobody reached kidney failure, and a meaningful fraction later recovered to eGFR ≥60 mL/min/m<sup>2</sup>.<sup>6</sup> BROAD similarly showed no difference in CKD progression between treatment arms.<sup>7</sup>

There is some evidence to suggest that more intensive SBP lowering confers a higher risk of incident CKD. In SPRINT, the <120 mm Hg group experienced more incident CKD, defined as a ≥30% decline in eGFR to <60 mL/min/1.73 m<sup>2</sup> (HR 3.27, 95% CI 2.43-4.40), though absolute rates were still low (3.7% vs 1.0% at 3 years).<sup>4</sup> An important nuance: in SPRINT, about one quarter of "incident CKD" cases later recovered and no longer met criteria at final visit, and biomarker sub-studies suggest these drops often reflect hemodynamics rather than intrinsic injury.<sup>9,10</sup>

Regarding the risk for acute kidney injury (AKI), this adverse kidney event occurred more often in the SBP <120 mm Hg group in SPRINT, but

there were no between-group differences in ESPRIT and BPROAD Falls/syncope/hypotension are relatively uncommon in trials, but real-world frailty, polypharmacy, and volume depletion are more common than trial eligibility allows.

So, what is the practicing clinician to do? Here's how we would translate current evidence into practice. (See **Figure 1**)

- If we're going to target SBP <120 mm Hg, we must measure BP in a standardized way.
- Consider targeting SBP < 120 mm Hg for the CV and mortality benefits
- Explain to the patients that kidney benefits are less certain and prepare them for the early dip in kidney function that can occur with lower SBP targeting
- Patients with advanced CKD, frailty, and heavy albuminuria were not well represented in the trials, so guidance on SBP targets is less clear for these and other subgroups.

As for most things in medicine, BP management in CKD should be personalized to each patient's clinical circumstances.

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Mario Funes Hernandez – [mfunes@umn.edu](mailto:mfunes@umn.edu)

Tara I. Chang – [tichang@stanford.edu](mailto:tichang@stanford.edu)

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