

REVIEW

Debate on the 2025 Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Emphasis on Defense Against the BP Threshold and Why We May Not Get There Easily

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ABSTRACT: Elevated blood pressure (BP) is the most important noncommunicable disorder worldwide. Finding and effectively managing elevated BP is the single greatest public health benefit we can accomplish, as it will reduce premature death and enable patients to live longer free of the disabilities that target organ damage inflicts on the brain, heart, kidneys, and legs. Hypertension guidelines are an invaluable source of information on how to detect elevated BP, how to evaluate people for situations where hypertension is a symptom of other disorders, and how to apply the various treatments that lower BP effectively in patients. Determining the point at which treating high BP is more likely to result in benefit than harm is a marriage of science and art. There is no right answer to what clearly constitutes hypertension when using a systolic or diastolic BP to define it. The science shows the mathematics behind the reduction of BP and the number of lives saved and target organs preserved. The art comes into play when a decision is made that, when a systolic or diastolic BP exceeds a certain level, it becomes reasonable to intervene at that point with treatment. Caregivers play an important role in monitoring and educating patients with hypertension—especially in the detection of unintended effects of treatment, such as excessive BP lowering, symptomatic hypotension, and impacts on laboratory tests and well-being. Nonadherence to prescribed therapies is a barrier to effectively managing chronic disorders like hypertension. Having a solid foundation in the science behind the guidelines and recognizing that the application of guidelines requires some clinical judgment gleaned from balancing the risks and benefits of treatment in each individual patient, is the basis for healthy exchanges of ideas, like this pro and con series which discusses the science and furthers the art. This review has taken the con side of several issues in the latest American College of Cardiology/American Heart Association 2025 Hypertension Guideline.

Key Words: blood pressure ■ caregivers ■ guideline ■ humans ■ judgements

Of all the things done clinically on behalf of a patient, little compares as favorably for the health of the brain, heart, and kidneys as evaluating and managing elevated blood pressure (BP). When a controlled trial of hypertension conducted in Baltimore demonstrated that it was feasible and beneficial to lower elevated BP with long-term oral medication in 1966,¹ in patients who were not in an accelerated phase of their hypertension, it signaled a paradigm change. Shortly thereafter the Veterans Administration (VA) studies,^{2,3} the development of the

High Blood Pressure Education Program,⁴ and ultimately an impressive series of subsequent clinical trials in hypertension provided the basis for a series of Hypertension Guidelines in the United States, and around the world.

Figure 1 shows an example of the evolution of hypertension thresholds, in this case, using the diastolic BP, for diagnosing the presence of hypertension, beginning with the original Joint National Committee Report up to the present American College of Cardiology (ACC)/American Heart Association (AHA) Guideline. Defining

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Nonstandard Abbreviations and Acronyms

ACC	American College of Cardiology
ACCORD	Action to Control Cardiovascular Risk in Diabetes
AHA	American Heart Association
BP	blood pressure
BROAD	Blood Pressure Control Target in Diabetes
CVD	cardiovascular disease
ESPRIT	Effects of Intensive Systolic Blood Pressure Lowering Treatment in Reducing Risk of Vascular Events
HOPE	Heart Outcomes Prevention Evaluation
HR	hazard ratio
PREVENT	Predicting Risk of Cardiovascular Disease Events
STEP	Strategy of Blood Pressure Intervention in Elderly Hypertensive Patients

and treating hypertension has been, literally, a work in progress over the past 48 years.

In this opinion piece, the goal is to outline and defend concerns about the role of guidelines in the management of elevated BP, divided into 4 areas: what are guidelines meant to do, who are they for, measuring BP, and a commentary on hypertension thresholds and goal BPs. The rationale for these 4 areas is to (1) point out that guidelines are not straitjackets and are understood to make room for clinical judgment; (2) point out that for whom the guidelines are written, that is, the audience, is a crucial point in guideline coverage, since they need to address the actionable aspects they recommend; (3) point out that a key component in translating a guideline recommendation into clinical practice is predicated on measuring BP accurately; and (4) review, in depth, the new recommendation for lower BP thresholds and treatment goals (ie, 120 mm Hg systolic), particularly in those at high cardiovascular risk.

GUIDELINES

The Oxford English Dictionary defines guideline like this:

A line drawn, marked, or placed as a guide, e.g. in positioning a tool or producing a drawing; a constructed line used for guidance. (Oxford University Press. (n.d.). Guideline, n. In Oxford English Dictionary. Retrieved July 9, 2025, from <https://doi.org/10.1093/OED/3652253681>).

In this sense, a guideline acts as a sort of boundary, like the rope-lines alongside the path on a mountain to keep climbers from falling and to show how far the limits

of safety extend. When the first hypertension guideline was published in the United States in 1977,⁵ there was an accompanying editorial by JAMA editor William Barclay that offered what I submit as sage advice regarding the guideline development process:¹⁴

The report should be viewed as a useful guide and not as a rigid directive on how to manage high BP. One should be aware that such reports are compromises and do not necessarily reflect the conviction of individual committee members. And:

Unfortunately, statements by committees, especially if issued by prestigious organizations or by the government become regarded as having more authority than they deserve.

There is more in the 1977 editorial. It takes <3 minutes to read it (4 paragraphs), and it is worth the invested time to do so. Having served on several guideline committees, I have been part of the process whereby we read, interpret, and critique the literature, mix in some of our own experiences in managing patients, and formulate statements on what the data shows and what we should do about it. The last 2 items, evidence statements and recommendations, are the key outward-facing components of guidelines. How the strength of a recommendation is determined, and what the level of evidence is to support that recommendation are vital in understanding what constitutes Guideline-Based Management or Medicine. In this regard, the current ACC/AHA 2025 guideline document has 5 Class (or Strength) of Recommendation categories and 5 Level of Evidence categories. Although they are organized a little differently in the new US guidelines, the principles are similar to the Class (or Strength) of Recommendation and Level of Evidence categories of the European Societies of Cardiology¹⁵ (2024) and the European Society of Hypertension¹⁶ (2023) guidelines.

In the words of the ACC/AHA 2025 writing committee, the new guideline is meant to: "... provide recommendations applicable to patients with or at risk of developing cardiovascular disease (CVD). The focus is on medical practice in the United States, but these guidelines are relevant to patients throughout the world. Although guidelines may be used to inform regulatory or payer decisions, the intent is to improve quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances and should not replace clinical judgment." (from the Preamble in the new guidelines).¹³

AUDIENCE

When you consider the variety of patients seen in a general practice setting compared with those seen in specialty settings, the complexity of providing care for a patient with hypertension, who may or may not have

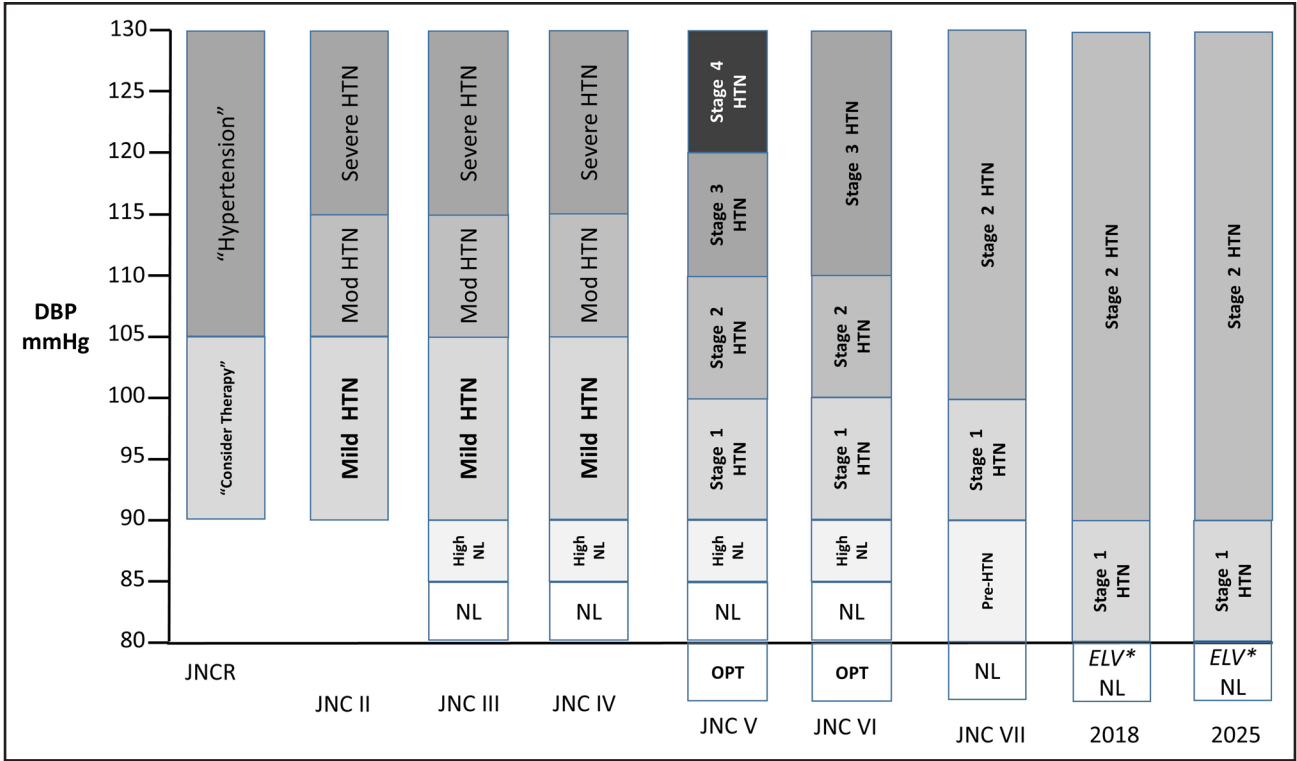


Figure 1. This figure shows the evolution in thinking about the threshold that defines hypertension over the past 5 decades, from a perspective of the diastolic blood pressure (BP) value alone. Beginning with the Joint National Committee Report (JNCr), which did not so much define hypertension, but recommended therapy start when the diastolic value was at or above 105 mm Hg.⁵ Over the ensuing Joint National Committee (JNC) reports the terms mild, moderate, and severe hypertension^{6,7} came, and went, being replaced by stages. Stages have shrunk from 4,⁸ to 3,⁹ and now are at 2.¹⁰ The introduction of prehypertension in JNC 7 sparked a lot of controversy. I skipped showing JNC 8 because we did not publish a formal table of BP definitions/values, however, we did use 90 mm Hg diastolic as a treatment threshold/goal in the first 5 recommendations.¹¹ Values for the last two bars are from the 2018¹² and 2025 guidelines.¹³ ELV*, elevated, defined as systolic BP 120 to 129 mm Hg and diastolic BP <80 mm Hg. HTN indicates hypertension; Mod, moderate; NL, normal; and OPT, optimal.

multiple other comorbidities, becomes daunting. The time pressures on health care providers are considerable, particularly when they are managing chronic disorders like high BP in addition to the frequent concurrent comorbidities of overweight/obesity and diabetes.^{17,18} In the Framingham Offspring Study, 1 in 5 men and one in 6 women had hypertension without an additional cardiovascular risk factor, while 8% of men and twelve percent of women had >4 cardiovascular risk factors.¹⁹ As clinical trial evidence and personal experience attest, the value of treating elevated BP is amplified greatly when more and more additional risk factors are present, and the use of risk calculators like Predicting Risk of Cardiovascular Disease Events (PREVENT) is justifiably recommended.²⁰ While the development of hypertension management guidelines is a welcome endeavor, and a useful update of progress in clinical trials, to care for the majority of hypertension patients it has to be situated among the guidelines for other common comorbidities (obesity, diabetes, hyperlipidemia, heart failure, and chronic kidney disease, to name a few). The electronic health record, with its ability to provide practice alerts, and the usage of consultants with deeper expertise in the concurrent comorbidities,

can be helpful. However, managing a complicated, uncontrolled hypertensive patient with multiple comorbidities, whose treatment adherence is hard to gauge, and who may be reluctant to treatment intensification despite an explanation of the benefits in doing so makes practicing Guideline-Based Management or Medicine in the current era quite challenging. If the historical trends in recommendations from the hypertension guidelines are an indicator of the future, I believe that the use of artificial intelligence or other apps that can both summarize the clinical picture of the patient and prioritize and integrate changes into the existing treatment regimen will become almost mandatory going forward.

MEASUREMENT

I hope the reader will permit a brief digression to make a point about the measurement of BP. In the mid-1980s, I attended an industry-sponsored Investigator meeting in which 2 BP medications would be tested against each other in Black versus White hypertension patients.²¹ One aspect of this meeting was the requirement to show proficiency in BP measurement by Investigators and

Coordinators. I failed to pass the BP measurement test and then had to do the preparatory segment and repeat the test (I passed on the second try). Over the subsequent years I have read and reread the directions for measuring BP, and participated in the ACC/AHA and National Heart Lung and Blood Institute updates on measuring this incredibly important metric.^{22,23} When Michael Rakotz assumed a leadership role in the American Medical Association, he asked what were significant problems with hypertension (in 2015). I answered that few people taking a BP actually did it correctly. Following this he organized the Check BP Challenge at the annual American Medical Association meeting in Chicago in the spring of 2016, recruiting medical students in the United States who indicated they had been taught how to take a BP to evaluate how well they actually did it. Six trained patients who knew what to look for and rated each student on a set number of steps in BP measurement determined how many were correctly done. An Apple iPad was given to any student who got it correct (ie, did all 11 steps correctly). One hundred fifty-nine students enrolled in our study. We gave away 1 iPad. Of the 11 steps, the average number of steps undertaken correctly in our medical student cohort was 4. When we submitted our results to an academic journal widely read by medical school deans and others who have a role in medical student curriculum development, it was rejected without review. After its eventual publication, a query came from an op-ed writer for *JAMA*.²⁴ Figure 2 reflects the legacy of that interview. There appeared to be substantial interest in this finding.

One of the consequences of poor BP measurement technique is that the errors made in measurement, resulting from the way most office practices undertake it,

result in a higher BP compared with measurement using proper technique.²⁵ How much difference? Compiled in the study just cited, the systolic BP is about 10 mm Hg higher, and the diastolic value is about 7 mm Hg higher in the usual office measurement when compared with the protocol-based way of BP measurement.²⁵ Although the new ACC/AHA guideline acknowledge the value of measurements outside the office (section 3.1.4), using home BP measurements runs the same risk as casual office BPs. One thing I recommend to health care practitioners who manage patients who do home BPs is to have the patient show them what they actually do at home when they measure BP. When asked why they (the patients) do it that way, the typical response is: "... that was how it is done in my doctor's office." Few people actually read the directions that come with the home monitors. If you have patients measuring home BPs using a casual office model as their learning template, there is a reasonable likelihood the home value will also register higher. It is valuable to be sure one is thoroughly knowledgeable in how to measure a BP correctly oneself, and to ensure that patients are taught to do the same.²⁶ Moreover, it is extremely useful to reinforce such training (for both oneself and one's patients) at some set interval, such as yearly.²⁷ The new ACC/AHA guideline support using a BP monitor that has undergone validation in the discussion of section 3.1.1,^{13,28} and recommend the www.validatebp.org website to find validated monitors.

THRESHOLDS AND GOALS

Herein lies what is, in my opinion, the most important section in a hypertension guideline. Since there is no

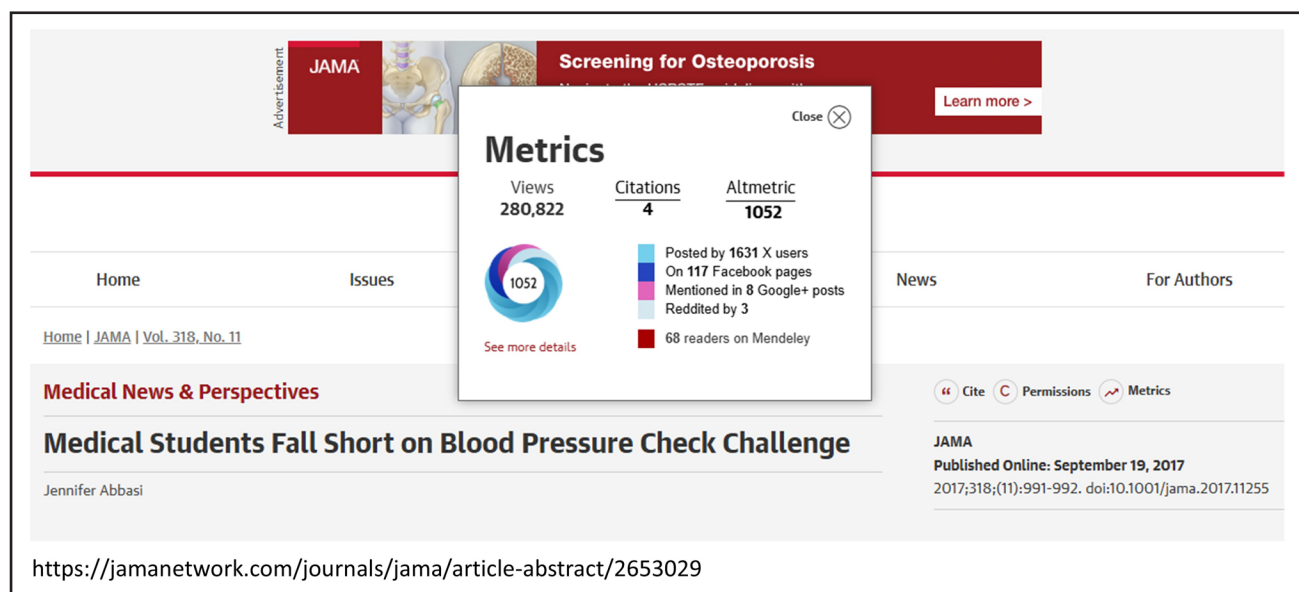


Figure 2. Screenshot of JAMA website accessed on July 10, 2025.

Citation (URL) depicted at the bottom of the graphic. The key issue is within the box marked Metrics and under the heading views. The Medical News and Perspectives piece was authored by Abbasi²⁴.

actual threshold value below which end points are virtually absent and above which end points occur commensurate with the rising level of BP, the definition of hypertension becomes defined as a value of systolic or diastolic BP above which the benefits of treatment outweigh the risks of therapy. This is akin to the phrasing in the definition of Class 1 recommendations shown in Figure 3 for the 2025 ACC/AHA guideline, where Benefit >>> Risk. In the new ACC/AHA hypertension guideline,¹³ the threshold to diagnose hypertension outlined in table 4 in that document is the same as in the 2018 ACC/AHA guideline.¹² Stage 1 hypertension is a systolic BP of 130 to 139 mm Hg inclusive, or a diastolic BP of 80 to 89 mm Hg inclusive. If the average of the systolic BPs is lower than 130 mm Hg and yet the average of the diastolic BPs is 80 to 89 mm Hg, the patient has stage 1 hypertension. The higher category or stage of the systolic or diastolic BP defines the category.

Epidemiology indicates that the cardiovascular risk of elevated BP doubles for every 20 mm Hg systolic BP increase. Specifically, the Lewington meta-analysis of almost 1 million people without prior vascular disease showed that every 20 mm Hg increase in systolic BP

doubled the risk of death from ischemic heart disease or stroke in the course of an average of 12 years of follow-up.²⁹ In the Framingham study of about 1800 patients without hypertension (>140/90 mm Hg at that time), with initial blood systolic pressure in the 130 to 139 mm Hg range at baseline, there were more cardiovascular events when compared with Framingham participants in the 120 to 129 mm Hg systolic range (high normal) or those with a systolic <120 mm Hg (optimal).³⁰ Arguing the logic in reverse, there is the potential for a large reduction in outcomes when the systolic is reduced from 180 to 160 mm Hg. There is about half that many outcomes prevented by reducing from 160 to 140 mm Hg. Reducing from 140 to 120 mm Hg yields about a fourth as many outcomes prevented. It begs the question: where do you stop? The Lewington study stopped at 115 mm Hg systolic because there is so little data about outcomes in adults with a systolic BP <115 mm Hg. At least 3 considerations result from this reasoning.

First, it is hard to apply research BP data to a typical (often called casual) office BP. For reasons covered previously casual office BPs are typically higher

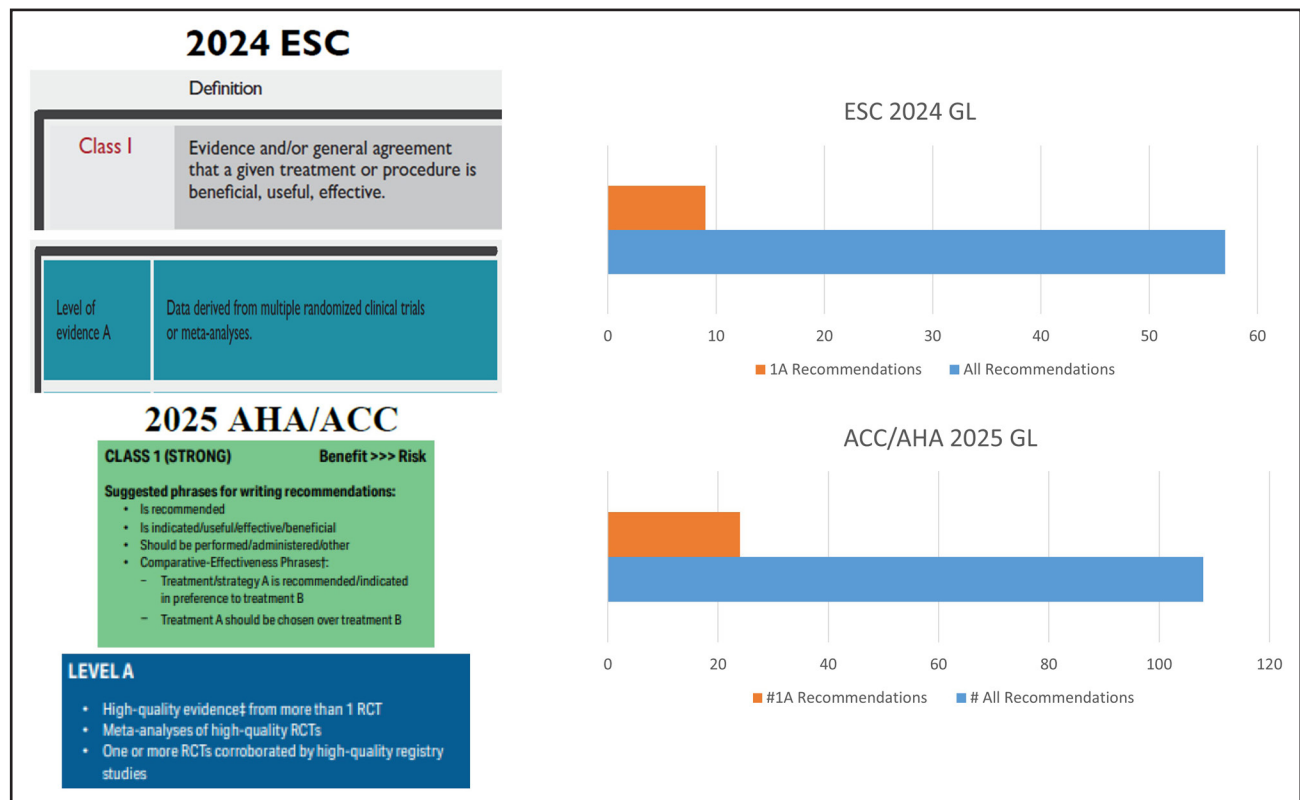


Figure 3. Shown are the definitions for a Class of Recommendation value of 1, and the Level of Evidence value of A, for both the European Society of Cardiology 2024 hypertension guideline (upper left) and the American College of Cardiology (ACC)/American Heart Association (AHA) 2025 guideline (lower left).^{13,15}

1A recommendations are considered the most compelling things we should be doing in the care of hypertension patients. The percentage of all recommendations for hypertension care which merited the rating of 1A in the guideline committee's estimate, 16% for European Society of Cardiology (ESC; **upper right**) and 22% for ACC/AHA (**lower right**), underscores how challenging it is to marry the science and art of hypertension care. Note: the Y axes are of different ranges for the ESC and ACC/AHA graphs.

than research-grade BPs. This is why so many guidelines (including the new ACC/AHA ones) recommend out-of-office BP measurements to confirm the office values.^{13,15,16,31} Second, high BP does not occur in most people in the absence of other cardiovascular risk factors, and the treatments prescribed for those comorbidities. In the Framingham data just cited,³⁰ >30% of the women and >40% of the men were current smokers. Using epidemiology data to guide clinical outcomes expected by selective intervention on a particular risk factor like BP is a potential slippery slope. In addition, there can be unforeseen pitfalls in using prior clinical trial data to inform outcomes in a newer clinical trial. For example, the ACCORD trial (Action to Control Cardiovascular Risk in Diabetes) relied on outcome data prior to ACCORD initiation to estimate how many cardiovascular events would occur in type 2 diabetics with hypertension. They postulated that the control group (assigned to a systolic BP of <140 mm Hg systolic) would experience about a 4% per year event rate.³² This was predicated in part on the HOPE trial (Heart Outcomes Prevention Evaluation) which demonstrated a 4.4% higher cardiovascular event rate in diabetics not assigned to ramipril compared with those who received ramipril.³³ The actual event rate in the standard-therapy arm in the BP study of ACCORD was 2.09%,³⁴ and the BP arm of the ACCORD study, as originally published, did not meet its primary end point.³²

Last, there is a practical issue involved here. In order to do a quality BP measurement would require a major rethink of how office measurement of BP is conducted. To get the quality of BP produced in a clinical trial like SPRINT (Systolic Blood Pressure Intervention Trial) requires training (and periodic reinforcement of that training) in the technique, adequate time to do the measurement with at least duplicate readings, and adequate space and personnel to accommodate this. Greater use of home BP monitoring is a possible solution to this major renovation in in-office patient flow, but the same things apply (technical training and training reinforcement, time, and perusal of data in a way that allows it to be summarized and actionable) to home BP measurements. It may be that someday we will have the same luxury in hypertension that our colleagues in diabetes care have with continuous glucose monitoring. At this time, that seems less practical until we develop well-validated devices and software technology that can sift through, clean, and present the BP data in a format where action can be taken, or not, as needed.

Referring to section 5.2.7 in the new guidelines, there is encouragement to achieve SBP <120 mm Hg in adults with confirmed hypertension presented as a 1A recommendation for those with increased risk for CVD, defined as a PREVENT prediction of 10-year CVD events of >7.5%.^{13,20} The same wording is applied to those with a PREVENT prediction of 10-year CVD events <7.5% (ie, not at increased risk of CVD), but is labeled as 2b-B-NR.

Let's look at the supporting documentation for these 2 recommendations, taking the increased CVD risk first.

In the first cited references, a systematic review and meta-analysis of published trials where 120 mm Hg systolic was a goal by Whelton and colleagues, the key paragraph (to me) reads like this in the Discussion: "Our results suggest intensive SBP treatment may increase the risks of hypotension, syncope, injurious falls, electrolyte abnormality, and acute kidney injury or acute renal failure. However, these events were uncommon, representing numbers needed to harm ranging from 514 (hypotension) to 2941 (injurious falls) for the SBP <130 mm Hg versus higher systolic BP target comparison. Our finding that intensive BP reduction significantly reduced the risk of all-cause mortality is important, not only because mortality is an important outcome per se but also because it is usually available for all trial participants and is not subject to bias in ascertainment. While the potential for adverse effects during more intensive treatment of hypertension is important, they pale in comparison to the potential for benefit resulting from a reduction in major CVD events and mortality."³⁵

A few concerns about this: the CVD outcomes are often combined when assessing benefit, but the adverse events are often listed individually. I would have liked to see the adverse events combined as well. The discussion of mortality is important and is probably the most robust outcome in randomized clinical trials of hypertension, and it is generally reported. However, observing the hazard ratios (HR) of mortality data in figure 3 of the Whelton manuscript, as well as the supplement, it is less impressive than the HRs of the CV events in general, and their sensitivity analyses do acknowledge that sometimes reevaluations of the mortality benefit do not quite achieve statistical significance.

The second study cited in support of the 1A recommendation enrolled Chinese patients 60 to 80 years of age and randomized them to a systolic BP of 110 to 130 mm Hg versus a systolic BP of 130 to 150 mm Hg.³⁶ Per the abstract of this study, the primary outcome was "... a composite of stroke, acute coronary syndrome (acute myocardial infarction and hospitalization for unstable angina), acute decompensated heart failure, coronary revascularization, atrial fibrillation, or death from cardiovascular causes".³⁶ This was a randomized clinical trial, as compared with the systematic review of Whelton. A few things to point out here. The first is that the STEP (Strategy of Blood Pressure Intervention in Elderly Hypertensive Patients) randomized clinical trial of Zhang and colleagues did achieve a statistically significant reduction in the primary outcome in the <130 mm Hg systolic group. They did so needing <2 antihypertensive medications in either the Standard or the Intensive group, despite an almost 20% prevalence of diabetes in their participants. In the 3.34 years of follow-up, a time frame similar to that of SPRINT, the primary outcome occurred

in 1.0% per year in the Intensive-treatment group compared with 1.4% per year in the Standard-treatment participants, compared with that of SPRINT, in which outcomes occurred in 1.65% per year in the Intensive compared with 2.19% in the Standard groups. The differences between STEP and SPRINT outcomes are interesting given that there are a larger number of outcomes included in the primary composite of the STEP study. Amongst those additional outcomes, the investigators reported that coronary revascularization (HR, 0.69 [95% CI, 0.40–1.18]), atrial fibrillation (HR, 0.96 [95% CI, 0.55–1.68]), and death from cardiovascular causes (HR, 0.72 [95% CI, 0.39–1.32]) were not different between the Intensive-treatment and the Standard-treatment participants. In the STEP study, at 1 year of follow-up, the Standard-treatment systolic BP was 135.3 and the Intensive-treatment value was 127.5 mm Hg, and the balance of time to the study inclusion at just over 3 years, showed a similar average of systolic pressure between the groups. I can see support for the 130 mm Hg systolic recommendation, but am struggling with the 120 mm Hg one. Zhang and colleagues also noted no benefit on kidney function with the lower BP goal group; more on this later. They observed no significant differences in adverse events with the exception of hypotension being more common in the Intensive-treatment group.

The third citation is the BPROAD trial (Blood Pressure Control Target in Diabetes), reported by Bi and colleagues, which enrolled >12 000 type 2 diabetics in China and randomized their systolic BP treatment goals to <120 mm Hg versus <140 mm Hg, with a 5-year follow-up.³⁷ The primary outcome in BPROAD was: "... a composite of nonfatal stroke, nonfatal myocardial infarction, treatment or hospitalization for heart failure, or death from cardiovascular causes".³⁷ Participants were at least 50 years old, on 1.4 antihypertensive medications at enrollment, with a prior cardiovascular event in 22% to 23%. After a year of follow-up, the average systolic BP in the Intensive-treatment arm was 121.6 mm Hg and 133.2 mm Hg in the Standard-treatment group, achieved by an average of 2.1 antihypertensive medications in the Intensive-treatment arm versus 1.3 to 1.4 antihypertensive medications in the Standard-treatment arm. After just over 4 years of follow-up of primary outcome events: "... occurred in 393 patients (1.65 events per 100 person-years) in the intensive-treatment group and 492 patients (2.09 events per 100 person-years) in the standard-treatment group (HR, 0.79 [95% CI, 0.69–0.90]; $P<0.001$)."³⁷ Again, side effects were stated to be similar between the 2 treatment arms, but in the headings of Conditions of Interest and Clinical Safety Alerts (table 3 in the Bi manuscript), the investigators did note more hypotension and more hyperkalemia in the Intensive-treatment arm. Again, no benefit on kidney function was observed.

Finally, for the 1A recommendation in the current ACC/AHA guideline, the study by Liu and colleagues, the

ESPRIT trial (Effects of Intensive Systolic Blood Pressure Lowering Treatment in Reducing Risk of Vascular Events), is cited. It enrolled 11 255 Chinese participants: "... (4359 with diabetes and 3022 with previous stroke)" who were assigned to intensive treatment (office systolic <140 mm Hg) or standard treatment (office systolic <120 mm Hg)³⁸ and followed for 3.4 years. The primary outcome was defined as: "... a composite of myocardial infarction, revascularization, hospitalization for heart failure, stroke, or death from cardiovascular causes".³⁸ The intensive group achieved an average systolic BP of 119.1 mm Hg using 2.8 antihypertensive medications, while the standard group achieved a systolic BP of 134.8 mm Hg using 2.1 antihypertensive medications. Liu and colleagues observed that: "... the primary outcome event occurred in 547 (9.7%) participants in the intensive treatment group and 623 (11.1%) in the standard treatment group (HR, 0.88 [95% CI 0.78–0.99]; $P=0.028$)."³⁸ These outcomes work out to 2.85% and 3.26% per year (dividing the reported percentages by the 3.4 years of follow-up). Regarding adverse events, Liu and colleagues noted that: "Serious adverse events occurred in 2366 (42.1%) participants from the intensive treatment group and 2378 (42.2%) participants from the standard treatment group (HR, 1.01 [95% CI, 0.95–1.07]; table 2, appendix 1 p 37)". Serious adverse events of syncope occurred more frequently in the intensive-treatment group (24 [0.4%] of 5624) than in the standard-treatment group (8 [0.1%] of 5631; HR, 3.00 [95% CI, 1.35–6.68]). All these participants were hospitalized and recovered, 2 resulted in fractures, one in each group. Among them, 5 in the intensive treatment group and 2 in the standard-treatment group were caused by hypotension. There was no significant between-group difference in serious adverse events of hypotension (0.1% versus 0.1%), electrolyte abnormality (0.2% versus 0.2%), injurious falls (0.5% versus 0.4%), or acute kidney injury (0.1% versus <0.1%).³⁸ There was a composite kidney end point in ESPRIT, defined as: end-stage renal disease, a sustained decline in eGFR to <10 mL/minute per 1.73 m², death from renal causes, or a sustained decline $\geq 40\%$ in estimated glomerular filtration rate (eGFR) from baseline³⁸ which occurred in 169 (3%) of participants in the intensive arm compared with 102 (1.8%) in the standard arm.

Of the 3 randomized clinical trials cited for the 1A recommendation, all 3 were in non-US populations, and were heavily represented by diabetic patients. Serious adverse events were similar between intense versus standard systolic BP arm assignments, but serious adverse events include most of the outcomes since heart attack, stroke, and heart failure are typically hospitalized, and serious adverse events usually include death as well. If they are similar between groups, and 1 group (the standard arm) has more primary outcomes occurring, what makes up the serious adverse events in the intense arms where less primary outcomes occur?

As for the 2b-B-NR recommendation in lower CVD risk patients with hypertension a single study is cited.³⁹ This study was conducted in Brazil, enrolling prehypertension patients who failed to reach BP goals after 3 months of lifestyle treatment. The primary outcome in this study was incident hypertension defined as 2 sets of 2 standardized BP measurements averaging either ≥ 140 mm Hg systolic or ≥ 90 mm Hg diastolic. Treatment was randomly assigned, as either a combination of 12.5 mg of chlorthalidone plus 2.5 mg of amiloride (372 participants) or a matching placebo (358 participants) in a one-to-one fashion for a period of 18 months. At completion, the systolic BP was 123.5 mm Hg in the active treatment group and 125.6 mm Hg in the placebo. This was not a CVD outcome trial. There were 3 heart attacks (2 in the diuretic group and 1 in the placebo group), and 1 death in each group, during treatment. From an adverse event standpoint, the low density lipoprotein cholesterol (LDL-cholesterol) and uric acid were significantly higher in the diuretic group, and the potassium (despite the amiloride) was significantly lower in the diuretic group at the end of 18 months. At this point, it is worth rereading the wording of the 2b-B-NR recommendation. The <120 mm Hg systolic goal outlined in the recommendation is acknowledged to be a weak (2b) one.

Finishing this section on BP thresholds and goals, it is worth discussing the kidneys since they are also potential casualties of the hypertension process. The new ACC/AHA guideline has recommendations regarding chronic kidney disease in section 5.3.8. These recommend a systolic goal of <130 mm Hg and are 1A when it comes to preventing CVD, and 1B-R when it comes to slowing CKD progression using an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin receptor blocker (ARB). The Kidney Disease Improving Global Outcomes recommends a systolic BP goal of 120 mm Hg for the purpose of reducing CVD (not chronic kidney disease [CKD] progression).⁴⁰ A recent opinion piece outlines some thoughts on why preventing CKD progression through more stringent BP control is so disappointing.⁴¹

Despite how I may sound here, I think the ACC/AHA BP guideline are well done and the initial section on what is new (table 1) is a great start when you are already familiar with past guidelines. I spent the bulk of this review covering thresholds and goals because I think will continue to be interesting and controversial over the interval until the next set of guidelines, especially in light of shortcomings outlined in the previous section on measurement. I retain some concern over the systolic BP threshold lowering as this will mandate more medication and nonadherence to prescribed therapies is a barrier to managing effectively chronic disorders like hypertension.^{42–44}

THINKING AHEAD

As alluded to in this commentary, taking care of patients with hypertension is truly risk management. Although

calculators that can estimate risk have been available for many years, there is still reticence to use them in primary care settings, for a lot of reasons.⁴⁵ In the new guidelines, the PREVENT calculator for predicting CVD risk is recommended. Time will tell if this will catch on. A recent issue of the *Journal of the American College of Cardiology* features 2 articles on PREVENT.^{46,47} In the Cho study, the performance of PREVENT was assessed at 4 large academic health systems: Penn Medicine (Philadelphia), Vanderbilt (Nashville), Massachusetts General Hospital (Boston), and Mount Sinai Icahn (New York). There were substantial differences in PREVENT model performance over a 10-year span between each of these centers, underscoring the challenge of predicting CVD outcomes in geographically and socially diverse populations like we have in the United States. As Lu points out in the JACC editorial commentary on PREVENT,⁴⁷ further calibration of PREVENT will likely be needed to plug some of the gaps in its current capabilities. PREVENT seems to be decent when compared with the older pooled cohort equations like the atherosclerotic cardiovascular disease (ASCVD) calculator in the Multi-Ethnic Study of Atherosclerosis (MESA) experience,⁴⁸ but the ideal predictor remains somewhat elusive to date.

My final comment is more in the nature of a speculation. We have witnessed a remarkable palette of CVD benefits and kidney function benefits with nonsteroidal mineralocorticoid antagonists,⁴⁹ sodium-glucose linked type 2 transporter inhibitors,⁵⁰ and glucagon-like peptide receptor 1a agonists/glucose-dependent insulinotropic polypeptide agonists,⁵¹ often noted with only modest BP differences between groups given active medication versus those treated with placebo. Perhaps future recommendations will showcase much greater use of these agents since they address the issue of obesity, a common finding in hypertension in many countries, while providing benefit in both CVD outcomes and kidney outcomes, while current antihypertensive agents do not address the full cardio-renal-metabolic spectrum.⁵⁰

ARTICLE INFORMATION

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