Blood Pressure Management
Beyond the Guidelines

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See related article, pp e13–e115

Since 1977, national blood pressure (BP) management guidelines have served the medical community and patients well.[1] Implementation of these guidelines has contributed to a decline in the mean BP level in the United States and to a remarkable reduction in age-adjusted cardiovascular disease (CVD) mortality rates.[2]

However, the decline in CVD death rates has leveled in the past 5 years, and CVD remains the leading cause of death in the United States and most industrialized countries. These trends are cause for concern because they indicate we are losing some progress toward achieving the ultimate goal of the American Heart Association, as expressed in its mission statement: Building healthier lives, free of cardiovascular diseases and stroke.[3] Although management of other risk factors is a part of the picture, perhaps the largest opportunity for moving toward that goal can be found in effective BP management. Could we better equip ourselves to address cardiovascular disease by focusing on the issues involved in the residual risk of elevated BP? Certainly, some of the residual risk lies in nonadherence to treatment. But, let us examine the residual risk from the current approach to BP goals of therapy.

The 2017 American College of Cardiology/American Heart Association Blood Pressure Management Guideline begins the process of addressing the need for more progress in reducing CVD events and deaths by implementing lower goal BP based on the best available evidence from randomized controlled trials (RCTs).[1] Effective implementation of the new systolic BP (SBP) goal of 130 mm Hg is expected to further reduce CVD mortality. However, the new guideline (of which I was a writing committee member and fully endorse) has the limitations of any evidence-based guidelines. Because of the lack of evidence from RCTs for younger patients and patients at a lower 10-year CVD risk, the new guideline necessarily leaves some important treatment decisions to the discretion of clinicians.

Specifically, the guideline, based on RCT evidence, recommends that adults with an SBP of 130 to 139 mm Hg with a 10-year CVD risk score ≥10% begin both lifestyle modification and BP-lowering medication. For those at lower risk, the recommendation is to begin lifestyle therapy alone and re-evaluate in 3 to 6 months. Lacking RCT evidence for medication therapy among low-risk patients in this BP range, the guideline leaves unanswered the question of how to proceed if lifestyle therapy does not achieve the goal of <130/80 mm Hg. Likewise, the guideline recommends lifestyle therapy for those with elevated BP (120–129/<80 mm Hg). Event-based RCT evidence for this population of mostly young adults simply does not exist and likely will not in the future, considering the logistical challenges of funding and executing such RCTs.

This leaves clinicians with a dilemma if we want to meaningfully move toward a goal of a population free of CVD and stroke. There is ample evidence from longitudinal observational studies and meta-analyses of RCTs that risk imposed by BP exists from an SBP of ≈115 mm Hg; and there is evidence of benefit in reducing CVD risk for higher risk patients by treating to an SBP ≈120 mm Hg.[1,2]

Furthermore, the success from efforts to date driven by previous BP guidelines has shifted the distribution of BP downward. This is a good and desired result. However, it brings into question whether the past strategy, of focusing on those at highest short-term risk, represents a sufficient approach for reducing cardiovascular risk in the population as a whole. Until recently, the majority of CVD events occurred in adults with a BP >140/90 mm Hg. With the reduction in population mean BP level, that reality is changing. The Table compares the percentage of incident CVD events from an analysis of pooled data from the Framingham Heart Study, Cardiovascular Health Study, and Atherosclerosis Risk in Communities Study in the 1980s to 1990s to pooled data from the Reasons for Geographic and Racial Differences in Stroke study, the Multi-Ethnic Study of Atherosclerosis study, and the Jackson Heart Study in the 2000s. In earlier decades, a majority of the CVD events occurred in patients with a BP >140/90 mm Hg, but in the more recent analysis, the majority of events occurred in patients with a BP <140/90 mm Hg.[4] The reality of most events occurring at lower BPs may dictate different treatment strategies going forward, including intervening with BP-lowering therapy earlier and at a lower BP. These data also raise the issue of whether a strategy to prevent hypertension—that is, prevent the age-related rise in BP—may represent the ideal strategy.

In all industrialized societies, BP rises with age. This is not true for some nonindustrialized hunter-gatherer human societies nor for animals, including primates.[5] The rise in BP, especially SBP, with age is neither physiological nor inevitable.

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Table. Percentage of Incident CVD Events in People With Blood Pressure <140/90 mm Hg[4]

CVD Event	1980s–1990s	2000s
Stroke	23%	63%
CHD	31%	63%
HF	26%	60%

CHD indicates coronary heart disease; CVD, cardiovascular disease; and HF, heart failure.

The diet of most societies, characterized by excess dietary sodium and calorically dense foods, is the main contributor to the rise in BP with age. The early rise in BP negatively impacts renal function and vascular stiffness, which in turn accelerate the rise in BP.

Small increases in BP at a young age track through adulthood and BP levels at age 30 years predict total mortality better than other measures (Figure).[6,7] As well, there is evidence that both lifestyle and BP-lowering medication given at BP levels <140/90 mm Hg prevent the further rise in BP[8,9] and medication regresses left ventricular mass.[10]

These issues—the abnormal rise in BP with age, the unhealthy food environment, the shift in the population distribution of BP, and the occurrence of the majority of CVD events at BPs below guideline target BP (even the recent American College of Cardiology/American Heart Association guideline)—raise serious questions including (1) Will current strategies, driven by RCT evidence and a focus on older, high-risk patients, move us toward the goal of a population free from CVDs and stroke? (2) Should there be a stronger focus on the biology of BP and CVD? (3) Is it now time to deal with the issue at the hypertension core by working together to facilitate a major change in food policy leading to a healthy food environment?

In conclusion, my suggestions for clinicians that go beyond what can be recommended in an evidenced based guideline:

In most patients, regardless of CVD risk score, if a goal BP of ≤130/80 mm Hg is not achieved after 6 months of appropriate lifestyle therapy, BP-lowering medication should be initiated (IIa B-NR).

In patients <40 years of age with elevated BP (SBP, 120–129 mm Hg) and with a family history of hypertension, diabetes mellitus, or dyslipidemia, if goal BP of <120/80 mm Hg is not achieved after 6 months of appropriate lifestyle therapy, BP-lowering medication should be considered (IIb C-LD).

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None.

References

1. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71:e13–e115. doi: 10.1161/HYP.0000000000000065.
2. Bundy JD, Changwei L, Stuchlik P, Xiaoqing B, Kelly TN, Mills KT, He H, Chen J, Whelton PK, He J. Systolic blood pressure reduction and risk of cardiovascular disease and mortality: a systematic review and network meta-analysis. *JAMA Cardiol*. 2017;2:775–781.
3. Jones DW, Peterson ED, Bonow RO, Masoudi FA, Fonarow GC, Smith SC, Solis P, Girgus M, Hinton PC, Leonard A, Gibbons RJ. Translating research into practice for healthcare providers: the American Heart

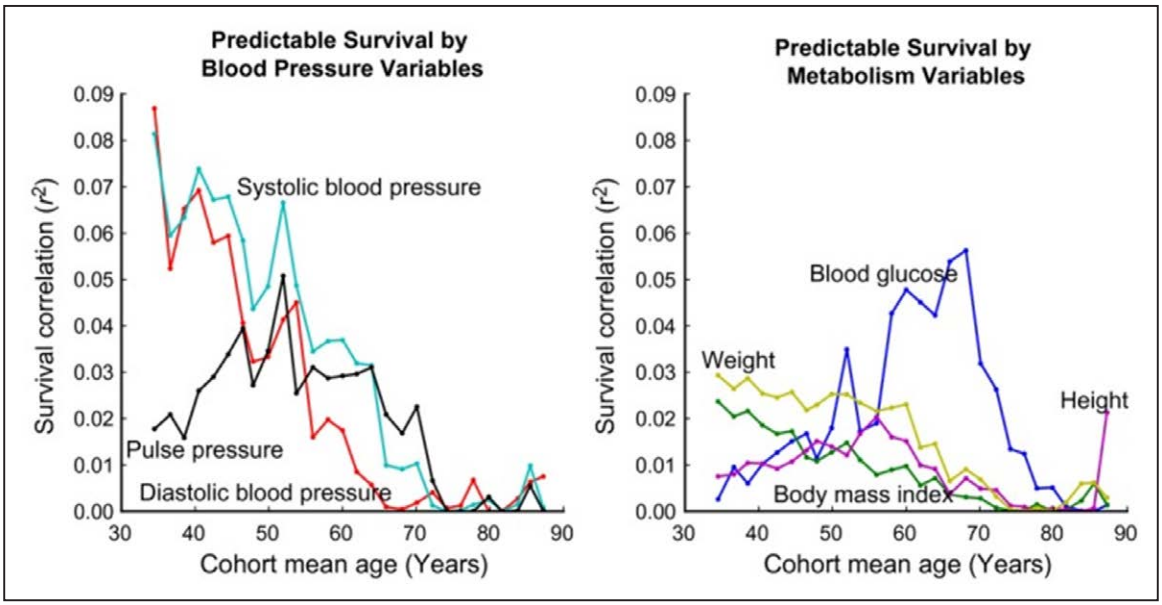

Figure. The individual variables' ability to predict survival changes over time. Blood pressure, body mass index, and weight are predictive of mortality primarily from ages 35 to 60 y and while blood glucose is most predictive from ages 57 to 73 y. Predicting all-cause mortality from basic physiology in the Framingham Heart Study.[10]

Association's strategy for building healthier lives, free of cardiovascular diseases and stroke. *Circulation*. 2008;118:687–696.
4. Tajeu GS, Booth JN 3rd, Colantonio LD, et al. Incident cardiovascular disease among adults with blood pressure <140/90 mm Hg. *Circulation*. 2017;136:798–812. doi: 10.1161/CIRCULATIONAHA.117.027362.
5. Gurven M, Blackwell AD, Rodríguez DE, Stieglitz J, Kaplan H. Does blood pressure inevitably rise with age?: longitudinal evidence among forager-horticulturalists. *Hypertension*. 2012;60:25–33. doi: 10.1161/HYPERTENSIONAHA.111.189100.
6. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008;117:3171–3180. doi: 10.1161/CIRCULATIONAHA.107.730366.
7. Zhang WB, Pincus Z. Predicting all-cause mortality from basic physiology in the Framingham Heart Study. *Aging Cell*. 2016;15:39–48. doi: 10.1111/acel.12408.
8. Whelton PK, Buring J, Borhani NO, Cohen JD, Cook N, Cutler JA, Kiley JE, Kuller LH, Satterfield S, Sacks FM. The effect of potassium supplementation in persons with a high-normal blood pressure. Results from phase I of the Trials of Hypertension Prevention (TOHP). Trials of Hypertension Prevention (TOHP) Collaborative Research Group. *Ann Epidemiol*. 1995;5:85–95.
9. Julius S, Kaciroti N, Egan BM, Nesbitt S, Michelson EL; Trial of Preventing Hypertension (TROPHY) Investigators. TROPHY study: outcomes based on the Seventh Report of the Joint National Committee on Hypertension definition of hypertension. *J Am Soc Hypertens*. 2008;2:39–43. doi: 10.1016/j.jash.2007.07.005.
10. Fuchs SC, Poli-de-Figueiredo CE, Figueiredo Neto JA, et al. Effectiveness of chlorthalidone plus amiloride for the prevention of hypertension: the PREVER-Prevention Randomized Clinical Trial. *J Am Heart Assoc*. 2016;5:e004248.

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