Hypertension and Target Organ Damage: Don't Believe Everything You Think!

George A. Mensah, MD, MPH[1,2]

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[1] Center for Translation Research and Implementation Science; National Heart, Lung, and Blood Institute; National Institutes of Health, Bethesda, Maryland, USA.
[2] Division of Cardiovascular Sciences; National Heart, Lung, and Blood Institute; National Institutes of Health, Bethesda, Maryland, USA.

Address correspondence to George A. Mensah, MD, FACC; Center for Translation Research and Implementation Science (CTRIS) ; National Heart, Lung, and Blood Institute; National Institutes of Health; One Rockledge Center, 6705 Rockledge Drive, Suite 6070, Bethesda, MD 20892; 301.496.1051; George.Mensah@nih.gov

Raised systolic blood pressure (BP) is a powerful independent risk factor for cardiovascular mortality and death from all causes.[1] It is also a major cause of clinical and pre-clinical damage to the heart, brain, retina, kidneys, and arterial blood vessels. Damage to these organs typically manifests as coronary heart disease, heart failure, stroke, other cardiovascular diseases and impaired renal function or end-stage kidney failure. The myriad pathophysiological mechanisms associated with the spectrum of target organ damage are shown in Table 1. Although these mechanisms are individually and collectively important, we now know that the magnitude of systolic BP elevation beyond the theoretical minimum risk exposure level and the presence of comorbid risk factors account for most of the observed organ damage and related death and disability. This knowledge has not always been common or without controversy.

Half a century ago, most clinicians believed that the rise in systolic BP level with advancing age was a benign physiological response to age-related arterial stiffening. In fact, systolic BP was believed to be normal as long as it did not exceed "100 plus your age."[2] Inherent in this belief was the concept of the BP dividing line above which hypertension and related risk of target organ damage were present, and BP level below that line was considered normal, with the risk of damage considered very low. Thus, a 70-year-old woman with a systolic BP of 170 mm Hg did not need treatment. Objective data from multiple epidemiological studies and hypertension clinical trials have proved these beliefs to be false. Other beliefs proven false include the notion of a benign clinical course in "borderline," "mild," or "high-normal" hypertension, especially within the context of multiple comorbid cardiovascular risk factors such as a strong family history of premature CVD, dyslipidemia, cigarette smoking, physical inactivity, poor nutrition, diabetes, obesity, and cardiometabolic syndrome. Similarly, the misconception that left ventricular hypertrophy was an appropriate compensatory physiological response to raised blood pressure has been dispelled by compelling epidemiological data initially gleaned from the Framingham Heart Study.[3,4]

Another more contemporary belief relates to the burden and severi-

Table 1. Spectrum of hypertension-related target organ damage

Organ	Known clinical and pre-clinical damage	Predominant mechanisms
Heart	Ischemic heart disease Sudden cardiac death Atrial fibrillation Left ventricular hypertrophy Adverse left ventricular geometric remodeling Left ventricular diastolic dysfunction Heart failure with reduced ejection fraction Heart failure with preserved ejection fraction	Atherosclerosis Atherothrombosis Endothelial dysfunction Oxidative stress Arrhythmogenesis Pressure effect on cardiac chambers Myocardial cellular hypertrophy Increased cardiac afterload
Brain	Hemorrhagic stroke Ischemic stroke Small vessel cerebral ischemic disease Vascular dementia Cognitive impairment	Pressure-related arterial wall stress Atherosclerosis Reduced cerebral blood flow Cumulative effects of small infarcts Disruption of the blood-brain barrier
Central Arteries	Aortic dissection Decreased aortic compliance	Arteriosclerosis Atherosclerosis Pressure-related arterial wall stress
Peripheral Arteries	Increased peripheral arterial stiffness Peripheral atherosclerosis	Arteriosclerosis Atherosclerosis Endothelial dysfunction Oxidative stress Inflammation Immunity Vascular rarefaction
Kidneys	Proteinuria Hypertensive nephropathy Decline in renal function Chronic kidney disease End-stage renal disease	Increased glomerular pressure Accelerated nephron loss Increased glomerular filtration Disruption of kidney BP regulation
Eyes	Hypertensive retinopathy Ischemic optic neuropathy Hypertensive optic neuropathy Choroidal neovascularization Retinal vascular occlusion	Pressure-related arteriolar stress Increased vascular growth factors Atherothrombosis
Heart Valves	Progression of valve calcification in aortic stenosis Aortic valve sclerosis Mitral annular calcification	Increased BP effects on valves Atherosclerosis

BP, blood pressure.

ty of hypertension in African Americans. It is well-known that African Americans have a high prevalence of hypertension (41% compared with 28% in non-Hispanic Whites); hypertension starts at much earlier ages; it is considered more difficult to control; and it is more frequently complicated by target organ damage and premature death.[5] Consistent with this narrative, long-standing suboptimal blood pressure control in African Americans is common, even in the presence of a history of cardiovascular disease or multiple cardiovascular risk factors. Not surprisingly, hypertension-related mortality in non-Hispanic Black men is nearly three-fold the rate seen in non-Hispanic White and Hispanic men; and in Black women, the disparity in mortality rate exceeds two-fold that of non-Hispanic White women. Given these observations, it is not surprising that some physicians believed or questioned whether hypertension in African Americans may be a different disease.[6,7]

The epidemiologic data suggest little to no evidence that hyperten-

sion in African Americans is a different disease and that suboptimal blood pressure control in this population should be the norm. As Cooper and Rotimi put it nearly two decades ago, "level for level," a similar risk of complications exists with blood pressure elevation among Blacks and Whites.[8] Additionally, hypertension prevalence in African Americans is not the highest in the world; higher rates of hypertension prevalence have been reported in majority Caucasian populations of Spain, Finland, Germany, and the Russian Federation.[9,10] More importantly, recent evidence suggests that hypertension control rates exceeding 80% is possible in African Americans[11,12] and the mortality benefit of intensive treatment to a systolic BP target of 120 mm Hg and below in persons without diabetes but otherwise at high cardiovascular risk is also seen in African Americans.[13]

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Other compelling epidemiological data should also help dispel misconceptions and erroneous beliefs about hypertension and its target organ damage. In the majority of patients (men, women, Black, White, youth and the elderly), hypertension-related target organ damage is most affected by: the level of systolic blood pressure; socioeconomic and demographic factors that impact access to care and quality of care received; comorbid risk factors; and adequacy of treatment to target blood pressure levels. Continued commitment to raising awareness about the clinical and public health importance of high blood pressure and the need for effective prevention, detection, evaluation, treatment, and control to target blood pressure levels is essential. In this regard, we should be guided by the totality of emerging but well-founded epidemiologic evidence and not necessarily believe everything we think or have thought in years past!

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Conflict of Interest Disclosure

None

Disclaimer

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