Prognostic Value of Blood Pressure Variability (p 461)

Combined major macrovascular, renal events and all-cause mortality

(2,345 events) P for trend <0.001

There is now good evidence that visit-to-visit variability (VVV) in systolic blood pressure (SBP) is a risk factor for cardiovascular events, but it is not clear whether it provides additional predictive information beyond traditional risk factors, including mean SBP, particularly in patients with diabetes mellitus. We used the opportunity afforded by the ADVANCE trial (Action in Diabetes and Vascular Disease: Preterax and Diamicron Modified Release Controlled Evaluation) and the ADVANCE-ON (ADVANCE-Observational) post-trial follow-up study in patients with diabetes mellitus, to analyze the predictive capacity of VVV of SBP in 9114 patients without major macrovascular or renal events or death in the first 24 months after randomization, on major outcomes during the next 7.6 years of follow-up. As can be seen in Figure 2A, VVV in SBP was significantly associated with increased 8-year risk of vascular events and all-cause mortality, independent of mean SBP and other traditional risk factors. In addition, VVV in SBP provided additional predictive information beyond that obtained from mean SBP and the traditional risk factors. With the advent of linked clinical records and home monitoring of BP, it is becoming practical to use VVV in SBP to improve individual risk stratification beyond using mean SBP and other factors. Our findings suggest that reduced VVV in SBP may be an important therapeutic target in patients with type 2 diabetes mellitus.

Healthy Vascular Aging (p 267)

In middle-aged and elderly nonhypertensive individuals, the residual lifetime risk for hypertension is estimated to be as high as 90%. Age-related increases in arterial stiffness and blood pressure are thereby widely accepted as an inevitable part of the aging process in acculturated societies. However, limited data derived from populations leading traditional hunter-gatherer lifestyles suggest that the age-associated increase in blood pressure may not be inevitable. Using data collected from 3196 Framingham Study participants aged ≥50 years, Niiranen et al examined the prevalence, correlates, and prognosis of healthy vascular aging, defined as absence of hypertension and pulse wave velocity <7.6 m/s (mean+2 SD of a reference sample aged <30 years). In their investigation, the authors demonstrate that the prevalence of healthy vascular aging decreases from 30.3% in patients aged 50 to 59 years to 1.0% in those aged ≥70 years. The researchers observed that those who met 6 of the American Heart Association’s Life’s Simple 7 goals had 10-fold greater odds of having healthy vascular aging, compared with people who achieved ≤1 goals. Healthy vascular aging was associated with a 55% lower risk of cardiovascular disease relative to absence of healthy vascular aging. The results of Niiranen et al suggest that maintaining normal vascular function in old age is possible, but challenging, in individuals acculturated to a Western lifestyle. Although their data are observational, these findings support intervention strategies targeting modifiable factors and behaviors to prevent or delay vascular aging and the associated risk of cardiovascular disease.

Angiotensin II and Vascular Dysfunction After Preeclampsia (p 382)

Women who have had preeclampsia during pregnancy are significantly more likely to develop cardiovascular disease (CVD). Despite the remission of clinical symptoms postpartum, these women subsequently develop CVD at a younger age and are more likely to die of CVD than women who have healthy pregnancies. Despite this obvious relationship between preeclampsia and CVD risk, the mechanisms responsible remain unclear. In this issue of Hypertension, Staniewicz et al report that women who have had preeclampsia exhibit reduced endothelium-dependent vasodilation and exaggerated vasoconstrictor sensitivity to angiotensin II compared with age and parity-matched women with healthy pregnancies. Furthermore, this reduced endothelium-dependent dilation is mediated, in part, by reductions in nitric oxide-dependent vasodilation and is rescued by inhibition of the angiotensin II type 1 receptor. These findings suggest that, despite the remission of clinical symptoms postpartum, a persistent deficit of NO signaling and vascular dysfunction may contribute to accelerated CVD progression and increased lifetime CVD risk in these patients. Furthermore, angiotensin receptor blockade may be a clinically relevant, mechanism-specific therapeutic strategy for the management of elevated CVD risk in this population.
Clinical Implications

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