**HYPERTENSION UPDATE: *QUO VADIMUS*?**

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Hypertension continues to represent a formidable challenge to human health and to healthcare worldwide. Exciting new developments in clinical and fundamental hypertension research are poised to change how we understand and approach the clinical management of hypertension. The recently concluded Systolic Blood Pressure Intervention Trial (SPRINT), testing the effect of additionally lowering the systolic blood pressure (BP) compared to the current guidelines target (<120 mm Hg vs. <140 mm Hg), was closed earlier than planned due to demonstrating significant cardiovascular (CV) benefits and reduced all-cause mortality. Where do we go from here? (*Quo Vadimus*?) These results indicate the need to go both forward and back into the translational continuum that connects fundamental discoveries to the prevention and cure of hypertension: moving forward, we will need to reassess the potential impact of these findings on the current clinical treatment targets for BP, which may necessitate further clinical investigations of patients with additional existing conditions, such as diabetes, investigations across the age spectrum, or of the effects of BP measurement methodologies. The significant beneficial CV effects of lowering BP by an d additional 20 mmHg indicates that we will also need to go ”back-to-the-bench” to better understand the fundamental underlying biological connections between these specific CV conditions and hypertension, and to test whether these new findings could become the basis for more efficient and specific treatments for hypertension or for the prevention of specific CV conditions. Meanwhile, recent advances in fundamental research have emerged to support reported clinical associations between hypertension, immunity, arterial stiffening, and genetics. Investigational research performed in several experimental models, in which hypertension occurs spontaneously or it is induced either by aldosterone,salt, or Angiotensin II, has been elucidating cellular and molecular pathways responsible for these associations. Key regulators identified included T cells, chemokines, and redox stress. Likewise, similar experimental studies demonstrated that arterial stiffening preceded hypertension and reversing stiffening prevented hypertension in the models used. By further pursuing these pathways, we may develop new biomarkers that could signal risk of developing hypertension or new pharmacological targets for prevention and management of hypertension. Future significant benefits for the health of hypertensive and CV patients will require the continuous bench-to-bedside, as is traditional, but also the back-to-the-bench cooperation between fundamental, clinical, and implementation research to close the remaining translation gaps in hypertension.