Vascular remodeling in hypertension can be categorized into 3 large categories: 1) Small arterial remodeling, 2) large arterial remodeling and 3) capillary rarefaction. Small arterial remodeling is due to eutrophic remodeling of the arterioles in response to chronic inflammation, oxidative stress, renin angiotensin aldosterone system activation, sympathetic nervous system activation and high salt intake. Although the initial remodeling is a compensatory mechanism to protect the major organs, increased remodeling and subsequent narrowing of the lumen of the arterioles results in increased systemic vascular resistance and right shifting of the sodium/blood pressure relationship, leading to elevation of blood pressure. Large arterial remodeling is characterized by hypertrophic remodeling of the aorta and large arteries that increases with aging. Certain risk factors such as diabetes, smoking, metabolic syndrome, dyslipidemia are known to accelerate large arterial remodeling with aging. Increase in aortic stiffness, due to the progression of large arterial remodeling, leads to increase in pulse pressure and increase in systolic blood pressure (SBP). The increase in SBP and pulse pressure leads to accelerated eutrophic remodeling of the arterioles that leads to increase in mean blood pressure and acceleration of the large arterial remodeling. Thus, the interplay between small and large arterial remodeling is important in the pathogenesis of hypertension.