Reactive oxygen species (ROS) play a crucial role in cell signaling, but excessive ROS levels lead to oxidative stress, which contributes to various diseases, including hypertension. This complex condition involves the interplay of multiple systems, including the heart, vasculature, kidneys, brain, and immune cells, where oxidative stress acts as a unifying factor. Despite evidence linking oxidative stress to hypertension, clinical trials using ROS scavengers and antioxidants have yielded inconsistent results, highlighting the need for a deeper understanding of redox mechanisms. Recent research highlights the role of the endoplasmic reticulum (ER) in oxidative stress and hypertension. The ER is responsible for essential cellular processes, and stressors like oxidative stress can lead to ER stress, activating the unfolded protein response (UPR). While the UPR initially promotes cell survival, prolonged activation can lead to detrimental effects, including apoptosis, inflammation, and fibrosis. The interconnectedness of oxidative stress and ER stress is crucial in hypertension, with ER stress influencing endothelial dysfunction, cardiovascular remodeling, and kidney damage. Inhibition of ER stress has been shown to improve vascular function and reduce blood pressure in experimental models. Understanding the intricate interactions between oxidative stress and ER stress is vital for elucidating the underlying mechanisms of redox-dependent processes in hypertension. Further research in this area could provide valuable insights for the development of novel therapeutic strategies to address hypertension and associated cardiovascular dysfunction. A Primer in Oxidative Stress ROS are generated by the incomplete reduction of molecular oxygen (O2) and can be either free radicals or nonradicals. They are produced through metabolic and enzymatic reactions within cells, with NADPH oxidase being the major source in the cardiovascular and renal system. The intracellular redox state is regulated by antioxidant systems, which maintain redox homeostasis. These systems include enzymes like superoxide dismutase (SOD), catalase, glutathione peroxidase, peroxiredoxins, and the thioredoxin system. The transcription factor Nrf2 plays a key role in regulating antioxidant enzymes and genes involved in antioxidant and anti-inflammatory processes.