Cell death has been recognized in the cardiovascular system for centuries. In Virchow’s 1858 lectures, he described atherosclerosis as producing new tissue, followed by cell death: “Thus, we have here an active process which really produces new tissues, but then hurries on to destruction in consequence of its own development.” Degraded and really produces new tissues, but then hurries on to destruction by cell death: “Thus, we have here an active process which described atherosclerosis as producing new tissue, followed.

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ATVB in Focus
Cell Death in Cardiovascular Disease
Series Editor: Martin R. Bennett

Articles in this series:

Cell Death in Cardiovascular Disease
Martin R. Bennett
involved in the degradation and recycling of the building blocks of organelles, proteins, and other components of the cytoplasm important for cellular homeostasis. Despite the fact that autophagy can lead to cell survival, recent studies indicate that apoptosis and autophagy involve complementary pathways and that autophagic degeneration may be a part of apoptosis, at least in some cell types.

This review series in ATVB on “Cell Death in the Cardiovascular System” is therefore very timely. The reviews cover the relationship between cell death and inflammation, particularly covering the innate immune system (Zheng et al), the evidence for and role of autophagy in vascular disease (Schijvers et al), and how ER stress and the unfolded protein response regulates apoptosis (Scull and Tabas). Importantly, the reviews demonstrate that we are moving beyond simple descriptions of cell death in disease. The elucidation of mechanisms underlying cell death has allowed the causal role of cell death to be elucidated in disease. These studies show that very little cell death (as measured) can result in profound consequences for the tissue, and that prevention of cell death is both part of the mechanism of action of established drugs, and a fertile ground for new therapeutics.

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