One of the undesirable aspects of modern life in economically developed countries is the high prevalence of cardiovascular diseases, which constitute the leading cause of death. In the year 2000, 2,846,429 people died from these diseases in the countries of the former "socialist bloc," accounting for 60% of all deaths (WHO, 2004). Roughly half of these cases were due to a single condition—ischemic heart disease (IHD). It is therefore understandable that both clinical and experimental cardiologists are striving to improve this unfavorable situation. However, this is an extremely difficult task, and its resolution will likely require many years of intensive research, ranging “from molecule to bedside.”

The underlying cause of IHD is an imbalance between the supply and demand of oxygen and metabolic substrates in cardiac cells. During ischemia, however, not only is the delivery of nutrients impaired, but also the removal of metabolic waste products is disrupted. Anaerobic metabolism predominates, metabolites accumulate in the tissue, and disturbances in ionic and neurohumoral homeostasis occur, ultimately impairing the contractile function of the heart muscle. Time plays a crucial role in the entire process, determining the boundary between reversible and irreversible damage: while the effects of short-term ischemia are almost entirely reversible, prolonged perfusion restriction leads to permanent impairment of essential cardiac cell functions.

Activation of anaerobic glycolysis leads to the accumulation of lactate and other metabolites, intracellular pH decreases, and ATP stores become depleted. The final stage of perfusion restriction is cell death and breakdown, either through necrosis or apoptosis, resulting in myocardial infarction. The only way to prevent or at least mitigate this process is early restoration of blood flow through the coronary circulation, thereby re-establishing oxygen supply to cardiac cells. The extent of ischemia-reperfusion injury to the heart muscle depends not only on the severity and duration of the ischemic insult, but also on the heart's intrinsic resistance to oxygen deprivation.