



## Adrenergic Mechanisms in Myocardial Ischemia

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Stress-induced myocardial ischemia is a frequent manifestation of coronary heart disease, and sympathetic activation is an important precipitating and aggravating factor in such stress induced ischemia. However, the complex interplay between the sympathetic initiation of myocardial ischemia, ischemia-induced alterations in sympathetic neurotransmission, as well as changes in adrenoceptor density and post-receptor signal transduction that can occur during ischemia remains incompletely understood. Not only the activation of myocardial  $\beta_1$  adrenoceptors, but also the activation of coronary  $\beta_2$ -adrenoceptors can contribute to myocardial ischemia. However, the role of  $\beta_1$ -adrenoceptor-mediated increases in contractility relative to heart rate in the initiation of ischemia is not clear, and the significance of  $\beta_2$ -adrenoceptor mediated changes in coronary vasomotor tone, as well as the responsible  $\beta_2$ -adrenoceptor subtypes are highly controversial. Malignant arrhythmias may be triggered by both  $\beta_1$ - and  $\beta_2$ -adrenergic mechanisms. Current research in this field is focussed not only on the underlying physiological and pathophysiological mechanisms, but also on clinical treatment strategies, e. g. , by  $\beta_1$ -blockade,  $\beta_2$ -blockade, bradycardic agents and calcium antagonists. Recent findings were presented and future research directions discussed during the 61<sup>st</sup> International Titisee Conference, held at the Schwarzwald-Hotel, Titisee, March 29-31, 1990 under the sponsorship of the Boehringer Ingelheim Foundation. Dr. Hasso Schroeder and Dr. Hermann Frohlich deserve special thanks for their generous support and pleasant organization of the meeting. The publication of the proceedings has been made possible by grants from Astra Chemicals, Bayer, ICI, Dr. Karl Thomae, and Upjohn.

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