

Preface

It is a fundamental property of all forms of life to adapt to changes in the environment. Such adaptations are commonly seen when individuals are exposed to high altitude, cold and hot conditions, or become apparent during ontogenetic development, exercise, and different diseases. These are, however, not limited—as is often done—to modifications that seem favorable to the individual. It is also becoming clear that these adaptations do not take place only at the whole body level, but all organs including the heart are also involved in this process. Cardiac adaptation to a wide variety of stressful situations such as pressure and volume overload, loss of functional tissue, or oxygen deprivation is usually associated with dramatic alterations in the circulating level of different hormones and growth factors providing signals to genetic apparatus in the cell. Different intracellular pathways are thus activated for remodeling of subcellular organelles, including extracellular matrix, sarcolemma, sarcoplasmic reticulum, mitochondria, and myofibrils. Particularly, changes in gene expression and protease activation play a critical role in inducing subcellular remodeling in the heart. In fact, remodeling of subcellular organelles is invariably associated with alterations in their function, and may serve as a compensatory mechanism for adaptation of cells and organs.

When the heart is confronted with an increased workload over a prolonged period, it usually tends to cope with the situation by increasing its muscle mass, a phenomenon referred to as cardiac hypertrophy. Initially, hypertrophy plays a compensatory role since it enables the heart to adapt to excessive hemodynamic load. However, the compensatory nature of hypertrophy if left unattended, deteriorates with time, and eventually ends in heart failure. Although the mechanisms of transition from compensatory cardiac hypertrophy to heart failure are not fully understood, depressed contractility during development of heart failure suggests an adaptive process that conserves the energy level. On the other hand, it can also be argued that contractile failure is a consequence of events associated with maladaptation. It is a real challenge not only to prevent the transition from compensated heart to failure but also to develop ways to manage subcellular and metabolic alterations during the development of contractile dysfunction. It follows that the processes of adaptation and maladaptation play an important role in the pathogeny

of serious cardiovascular diseases, such as hypertension, valvular diseases, congenital heart disease, myocardial infarction, and different cardiomyopathies as well as during adaptation to exercise and high altitude hypoxia.

This book summarizes the present knowledge of different mechanisms involved in the development of positive and negative consequences of cardiac adaptation. Particular attention was paid to the still underestimated adaptive cardiac responses during development, to adaptation to the frequently occurring pressure and volume overload as well as to cardiac changes, induced by enduring exercise and chronic hypoxia. Our effort was to put together the rapidly developing basic and clinically relevant information on adaptive mechanisms and thus contribute to the better understanding of possible prevention and therapy of life-threatening cardiovascular diseases.

The presentation of the subject matter in the form of 24 manuscripts on cardiac adaptations, as developed by several investigators for this book is organized in three parts. Part I dealing with developmental aspects of cardiac adaptation includes seven chapters on comparative and molecular aspects of cardiac development, prenatal and postnatal developments, coronary vascular development, and ontogenetic adaptation to hypoxia as well as cardiac and arterial adaptation during aging. Part II is devoted to cardiac adaptations to overload on the heart and includes eight chapters. Discussion in this part of the book is centered around the mechanisms of cardiac hypertrophy due to pressure overload, volume overload, exercise, gender difference, high altitude, and different pathological situations. Part III of this monograph includes nine articles on molecular and cellular mechanisms of cardiac adaptation. These chapters highlight the roles of sympathetic nervous system with respect to α -adrenoceptor and β -adrenoceptor mechanisms in the development of cardiac hypertrophy. In addition, the modulatory role of mitochondria, autophagy, adenosine, growth factors of different proteins and hormones in cardiac adaptation under several pathophysiological situations is discussed.

We are grateful to Ing. M. Markova from Prague as well as Dr. Vijayan Elimban and Ms Eva Little of Winnipeg for their help in editing the manuscripts. Cordial thanks are also due to Ms. Portia Formento, Springer USA, for her continuous advice and understanding during the editorial process. We hope this book will be of great value to students, fellows, scientists, clinicians, and surgeons. In addition, we believe that cardiovascular investigators will find this book highly useful in their studies for finding solutions to prevent and reverse cardiovascular abnormalities in diverse pathological conditions.

Prague
Winnipeg

Bohuslav Ostadal
Naranjan S. Dhalla



<http://www.springer.com/978-1-4614-5202-7>

Cardiac Adaptations

Molecular Mechanisms

Ostadal, B.; Dhalla, N.S. (Eds.)

2013, XVIII, 466 p., Hardcover

ISBN: 978-1-4614-5202-7