PREFACE

Cerebral ischemia is a global public health threat and one of the important causes of mortality and loss of independence in those affected. Moreover, cerebral ischemia is one of the most common causes of dementia, which sooner or later develops in more than half of patients after an ischemic episode.

This book presents a new picture of ischemic brain disease that knows no geographic boundaries and continues to attract the attention of a large community of scientists, physicians, engineers, and related health professionals by synthesizing the latest modern data on disease progression mechanisms and possible care for patients with this disease. The authors present the characteristics of cerebral ischemia from pregnancy and childhood through adolescence to adulthood. Post-ischemic brain injury in animals and humans leads to proteomic, genomic, and structural changes in various brain structures, starting in the hippocampus, showing changes identical to those seen in Alzheimer's disease. Cerebral ischemia is the second naturally occurring disease after Alzheimer's disease, which primarily causes the death of pyramidal neurons in the CA1 region of the hippocampus. The main pathology is considered to be post-ischemic changes in the hippocampus, especially in its CA1 area, underlying episodic memory impairment, which is the earliest and most important clinical symptom of post-ischemic dementia. Ischemia is also responsible for severe changes in the temporal lobe, which is the target area of the main axon exit network of the hippocampus. Both regions are structurally and functionally connected to each other and are essential for basic memory learning processes. An increased risk of post-ischemic dementia has been shown to be associated with age, hippocampal neuronal atrophy, and neurodegenerative changes associated with the development of amyloid plaques and neurofibrillary tangles, along with increased expression of amyloid processing and tau protein genes. The presented data comes from the latest research by leading and renowned scientists around the world who provide new evidence for understanding the processes involved in ischemia and recirculation. In the future, it is likely that the manipulation of ischemia-activated genes associated with Alzheimer's disease and their proteins will offer new hope for the development of causal therapies urgently needed to prevent or treat post-ischemic brain neurodegeneration.

The chapters discuss possible diagnostics, the choice of therapy, the prognosis of outcome, and the long-term course of the disease in people after cerebral ischemia. The book provides vital knowledge about the current state of post-ischemic processes in the brain and possible post-ischemic care based on a persistent search for solutions by scientists and clinicians. It therefore seems timely for such a book to introduce the potential reader to the scope and complexity of the current state of new avenues of experimental and clinical research on post-ischemic lesions. I would like to thank all the authors for their reliable and responsible work on the creation of this book. In 13 chapters, various aspects of ischemic brain disease are discussed, that collectively provide a comprehensive picture of pathogenesis, associated risk factors, potential new biomarkers, and

therapeutic targets. I believe this book will encourage scientists and clinicians to delve into this field and take on the difficult challenge of working on an effective treatment for the aftermath of cerebral ischemia.

Ryszard Pluta, MD, PhD
Professor and Head of Laboratory of Ischemic and Neurodegenerative Brain Research,
Mossakowski Medical Research Institute,
Polish Academy of Sciences,
Warsaw, Poland.
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