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Nota di contenuto	Intro -- Contents -- Preface -- Chapter I -- Reorganization of Elementary Functions (REF) after Brain Injury and in the Intact Brain: A Novel Understanding of Neurocognitive Organization and Reorganization -- Abstract -- Introduction -- Conceptualisations of Posttraumatic Functional Recovery -- Functional Localization and Recovery -- Mechanisms of Posttraumatic Functional Recovery -- The REF-Model -- Elementary Functions -- Algorithmic Strategies -- Reorganization of Elementary Functions (REF) -- Algorithmic Modules -- Neural, Behavioural and Conscious Flexibility in the Normal and Brain Injured Individual -- Implications of the REF-Model for Posttraumatic Rehabilitative Training -- Generalization of Institutionalised Training to Reallife Situations -- Utilization of Advanced Technology in Neurorehabilitation -- Training Aimed at Developing AMs -- Implications of the REF-Model for the Prognosis Regarding Posttraumatic Cognitive Performance and Rehabilitation -- The Importance of the Pretraumatic Experience of the Patient -- Brain Reserve and Cognitive Reserve -- Localization and Recovery of Cognitive Functions - Rehabilitation and Reconceptualisation -- Conclusion -- Acknowledgment -- References -- Chapter II -- The Splitting of the Brain: A Reorientation towards Fiber Tracts Damage in Amnesia -- Abstract -- Introduction -- Fiber -Behavior Interactions

with a Focus on Conscious Memory -- Memory - Systems and Divisions -- Memory and Time -- Memory and Content -- Memory Disorders -- Fiber Systems Relevant for Conscious Memory Processing -- Association Fibers with Yet Unclear Contribution to Conscious Mnemonic Processing -- Association Fibers with (Likely) Functions in Memory -- Striatal Fibers -- Commissural Fibers -- Thalamic Fibers (Peduncles) -- The Capsula Interna -- Other Cortico-Subcortical Fiber Bundles Related to Memory Processing.

Conditions Affecting the Integrity of the Brain's White Matter -- Conclusion -- References -- Chapter III -- Molecular Determinants of Hypoxic-Ischemic Injury in Developing Brain and Potential Strategies for Neuroprotection -- Abstract -- Abbreviations -- Introduction -- Pathophysiology of Neonatal Brain Injury -- Spectrum of Cell Death in Hypoxic-Ischemic Brain Injury -- Role of Mitochondria in Apoptosis and Necrosis -- Excitotoxicity and Neonatal Hypoxic-Ischemic Brain Injury -- Apoptosis as the Principle Mechanism of Hypoxic- Ischemic Neuronal Death -- Sex-Specific Activation of Cell Death Signaling Pathways -- Anti-Apoptotic X-Linked IAP (XIAP) and Neuroprotection against HI Brain Injury -- Excitatory Synaptic Function -- Neuronal Pentraxins -- Neuronal Pentraxins Interact with the AMPA-Type Glutamate Receptors -- NP1 Gene Silencing Is Neuroprotective -- Conclusion -- Acknowledgments -- References -- Chapter IV -- Comet Assay as a Useful Tool to Assess Brain Damage -- Abstract -- 1. Introduction -- 2. DNA Damage -- 3. Comet Assay Technique -- 4. Methodology of the Comet Assay -- 4.1. Preparation of Microscopic Slides -- 4.2. Cell Lysis -- 4.3. Alkali Unwinding -- 4.4. Electrophoresis -- 4.5. Neutralization -- 4.6. DNA Staining -- 4.7. Scoring the Comets -- 5. Advantages of the Comet Assay -- 6. Comet Assay Applications -- 7. Use of the Comet Assay to Measure the Brain Damage -- Conclusion -- References -- Chapter V -- Responsiveness and Prognosis in the Severe Disorder of Consciousness -- Abstract -- Introduction -- Consciousness and Descriptors of Consciousness -- Clinical and Behavioral Scales -- Residual Responsiveness in the VS: Electrophysiological and Neuroimaging Studies -- Heart Rate Variability -- HRV and the Brain -- HRV, Responsiveness and Outcome in the Severe Disorder of Consciousness -- Conclusion -- References.

Chapter VI -- Cerebral Radiation Necrosis: Causes, Management and Prognosis -- Abstract -- Classification of Radiation Injury to the Brain -- Incidence of Radiation Cerebral Necrosis -- Pathophysiology of Radiation Induced Necrosis to the Brain -- Management and Prognosis -- New Treatment Approaches for CRN -- Conclusion -- References -- Chapter VII -- Exercise as a Therapeutic Approach after Brain Damage -- Introduction -- Physical Activity and Exercise -- Age and Exercises -- Enrichment Environment and Morphological Changes -- Neurogenesis and Physical Activity -- Physical Activity and its Effects on Neurotransmission -- Physical Activity and Growth Factors -- Conclusion -- References -- Chapter VIII -- Neuroprotection in Experimental Cerebral Ischemia -- Abstract -- Introduction -- Histopathological Changes in Mongolian Gerbils Brain Submitted to Different Durations of Cerebral Ischemia -- Motoric Behavior Changes in Mongolian Gerbils Submitted to Different Durations of Cerebral Ischemia -- Oxidative Stress Rate in Mongolian Gerbils Brain Submitted to Different Durations of Cerebral Ischemia -- Neurological Status and Mortality Rate in Mongolian Gerbils Brain Submitted to Different Durations of Cerebral Ischemia -- Neuroprotective Efficiency of NMDA Receptor Blockade in Mongolian Gerbils after Various Duration of Cerebral Ischemia in Gerbils -- Neuroprotective Efficiency of NMDA Receptor Blockade in the Striatum and CA3 Hippocampus after Various

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MK-801 Effect on Neurological Status and Mortality Rate in Mongolian Gerbils Brain Submitted to Different Durations of Cerebral Ischemia -- Conclusion -- Acknowledgments -- References -- Chapter IX -- Initiation of Inflammatory Mechanisms in Acute Brain Damage -- Abstract -- Introduction -- Conclusion -- References -- Index -- Blank Page.

Sommario/riassunto

This book presents current research in the study of the causes, management and prognosis in brain damage including the implications for therapeutic intervention in the reorganization of elementary functions (REF) after brain injury; a reorientation towards fiber tracts damage in amnesia; the use of alkaline comet assay to assess DNA damage and repair in brain damage; responsiveness and prognosis in the severe disorder of consciousness; the pathophysiology, management and prognosis of cerebral radiation necrosis; exercise as a therapeutic approach after brain damage; neuroprotection in experimental cerebral ischemia; and initiation of inflammatory mechanisms in acute brain damage.
