If this book had been written 30 or 40 years ago, it could have been titled “Brain recovery? Could it occur?” In fact, the concept that each area of the brain has a rigid network of connections and then a fixed and immutable function was too strongly rooted to accept an alternative idea that an adult brain could modify itself in response to an injury. We used to think that the brain, once damaged, could not repair itself.

If this book had been written 10 years ago, it could have been titled “Neurobiological Aspects of Brain Recovery.” In fact, breakthroughs in neuroscience have undoubtedly shown that though individual neurons might be damaged beyond repair, the brain exploits its neuroplasticity properties and tries to repair itself with a powerful efficiency. An ever increasing number of studies have supported the notion that neuronal fibers grow and form new terminals in response to nearby damage and have analyzed the myriad of factors (pre- and post-injury experiences, kind of lesion, drugs, hormones, neurotrophic factors, aging, diet, stress, state of immune system,...) influencing recovery or sparing of function following brain damage.

Today, it is fully rigorous studying brain recovery by addressing the double neurobiological and psychological component of the functional recovery that follows brain injuries. In fact, the effects of a brain damage are extremely widespread, provoking physiological, cognitive, emotional, and behavioral changes and impacting all areas of a person’s life. It is now clear that a combination of factors will be required to make as full a recovery as possible. Psychological factors can inhibit recovery of brain function. In many cases, these aspects of the injury may be the most difficult for the affected person to deal with, due to personality changes and social difficulties faced trying to cope with the change. For example, depression and sleep disturbances not only may cause troubles in concentration, memory, and mood but also may cause a negative outlook on life. Current theories suggest that neurobiological and psychological aspects of brain recovery are reciprocally related, so that the psychological aspects may be due to, or a cause of, diminished brain plasticity.
Despite the huge bulk of clinical and experimental data on brain recovery processes, the functional recovery of each subject can hardly be predicted due to the complex variety of mechanisms involved and the possibility to continue recovery over time. No two brain injuries are alike and the course and the degree of functional recovery are different for each subject. So, individuals progress through brain recovery phases at their own pace. There is a first period of spontaneous recovery that takes weeks or months beyond the date of the injury, when the brain attempts to recover the damaged neurons and to redesign the networks controlling communication among neurons. In this first phase, the rehabilitative focus has to be on maximizing the spontaneous recovery processes and improving function within the areas of the specific deficits. Subsequently, the focus may shift toward retraining skills and functions that have been lost and learning adaptive strategies. In neurobiological terms, during the unique window of opportunity in which neural plasticity peaks (one to three months after injury), neurorehabilitative strategies are most effective. However, significant improvements can occur even at later stages, especially when the rehabilitative strategies combine task-specific training with therapies that activate neural plasticity.

In the attempt to work around the damage, the brain uses its greater abilities to adapt function rather than to regenerate structure and this significant adaptive aptitude is based on widespread plastic properties. By understanding molecular and neuronal substrates and interactions that lead to injury-dependent plasticity, we may be able to devise more effective treatment strategies for repair. However, it has to be taken into account that while the synergistic combination (in the correct temporal order) of interventions on sensory or sensory-motor input with molecular interventions may enhance plasticity and thereby promote recovery of function after injury, unfortunately in other cases the combination of drug treatments with behavioral training may reduce the effectiveness of either treatment alone. Thus, it is crucial to elucidate the substrates of injury-induced neuronal plasticity and to understand how the complex interactions between structure (molecules, synapses, neuronal networks) and function (activity, learning, injury) affect persistent changes in the brain. Plasticity substrates likely overlay across developmental processes, adult tissue homeostasis, various types of brain injury, and activity paradigms. How the plastic changes interact with, are influenced by, and directly affect the injury has profound effects on the development of efficient strategies for recovery. One critical feature of brain recovery is that activity facilitates appropriate rewiring after injury and may prevent maladaptive patterns of connections. Activity shapes connections by inducing the expression of promoting or inhibiting growth factors, neutralizing or enhancing either subset to drive structural changes.

The mechanisms of functional recovery can be grouped into two general classes, restitution, and substitution of function. While restitution of function suggests that neuronal pathways are reactivated and functions are restored, substitution of function refers to transfer or reorganization of functions from the damaged tissue to healthy areas. Both kinds of mechanisms are related to synaptic plasticity that modulates density and efficiency of neurotransmitter receptors through protein phosphorylation and regulation of gene expression. Although the changes mediated
by protein phosphorylation have rapid onset and short duration compared with plastic phenomena mediated by gene expression, both plastic processes alter function and efficacy of synapses and finely tune the functional state of neurons in response to varied synaptic inputs. It is known that post-synaptic neurons, once deprived of their characteristic synaptic inputs, develop increased sensitivity to neurotransmitter via the emergence of new receptors and larger surface areas, thus facilitating post-lesional activation of pathways and restitution of function.

The most recent approaches to the “neuroscience of rehabilitation” try to understand the links between motor activity, cognitive functions, and emotional behavior with dendritic structure and cytoskeletal dynamics to reveal the molecular and cellular bases of recovery following brain diseases. Therefore, comprehension of neural architecture appears to be a necessary step toward greater understanding of computation in the nervous system. A strong correlation exists between dendritic abnormalities and motor, cognitive, and emotional behaviors in a variety of neurological diseases including Down, Rett, and Fragile-X syndromes; Autism; Schizophrenia; Alzheimer’s, Parkinson’s, and Huntington’s diseases; and Duchenne/Becker muscular dystrophies. Thus, elucidating the molecular genetic mechanisms by which multiple local interactions of cytoskeleton elements direct the growth of dendrite arbors has direct clinical relevance. Learning to manipulate arbor growth mechanisms appears to be important to develop efficient neuroregenerative strategies. Chapter 1 of the present book (Structural Plasticity in Dendrites: Developmental Neurogenetics, Morphological Reconstructions, and Computational Modeling by Nanda, Das, Cox, and Ascoli) reviews the significance of proper dendritic development and neurogenetic mechanisms governing the brain plasticity with an emphasis on transcriptional-mediated cytoskeletal regulation; then it discusses the techniques used to identify the molecules and pathways responsible for arbor development; next, it describes the digital reconstructions of neuronal morphology; it discusses the modeling methodologies to generate artificial neurons in simulations; and finally, it proposes how novel advances on these three fronts can achieve the objective of a systems level understanding of dendritic growth.

Among the processes that maintain cellular metabolism and homeostasis and guarantee regulation of protein synthesis and organelle, the turnover autophagic activation plays important roles in neuronal survival and function under both physiological and pathological conditions. Although autophagic dysfunction has been implicated in the pathogenesis of several neurological pathologies, the cellular factors underlying homeostatic versus pathogenic activation of autophagy have not yet been identified, nor is it fully clarified how the loss of basal autophagy or imbalance of autophagic flux leads to neuronal death and why autophagosomes accumulate abnormally in damaged neurons in the presence of several neurodegenerative diseases as well as brain and spinal cord trauma. Chapter 2 (Autophagy Mechanisms for Brain Recovery. Keep It Clean, Keep It Alive by Viscomi, D’Amelio, Nobili, Cavallucci, Latini, Biscchica, Sasso, and Molinari) reports the current state of knowledge on the relationship between autophagy impairment and pathophysiological mechanisms in several neurological pathologies; describes how molecular mechanisms of autophagy affect neuronal function and contribute to
neurodegeneration in chronic and acute brain pathologies; and discusses the implications of autophagy processes in neurological recovery.

A bulk of research has evidenced the dramatic effects of experience on structure and function of the brain and it is well known that all activity-dependent processes are based on plastic synaptic rearrangements maintaining and refining active synapses and discarding inefficient connections. Multisensory, cognitive, and social stimulations are the crucial incentives that shape the brain patterns to adapt to environment both in physiological and pathological conditions. The importance of environmental stimuli in shaping brain structure and function has led to the development of an experimental setting in which the housing condition is improved to provide a combination of cognitive, motor and social stimulation relative to standard housing, the environmental enrichment. Such a model has a protective and therapeutic potential in reducing symptoms of a variety of neurological diseases with reduced side effects. The structural modifications induced by the prolonged exposure to an enriched environment have been described in widespread brain areas, from neocortical to cerebellar cortical regions. Since the most remarkable functional outcomes of enrichment are related to learning and memory functions, many researches have been focused on the hippocampus. Chapter 3 (Environmental Enrichment Repairs Structural and Functional Plasticity in the Hippocampus by Ghiglieri and Calabresi) provides the most recent evidence that environmental enrichment stimulates the endogenous potential of hippocampus for plasticity and repair. It examines the role of environmental enrichment as a trigger for intrinsic resilience mechanisms that preserve synaptic integrity in distinct hippocampal neuronal populations against aging and neurodegeneration. According to the potential of the environmental enrichment to boost proliferation and survival of neuronal and glial cells, the analysis of how the environmental enrichment may reopen the critical periods in which the potential to tune a brain function is maximal, can provide support for efficient therapeutic interventions on loss of function due to a variety of neurological diseases.

The experience-dependent increase in neuronal connectivity may represent the neurobiological substrate not only for the efficient therapeutic interventions but also for the neuroprotective capability to cope with an injury as proposed in the theory of “brain and cognitive reserve.” In fact, some individuals maintain high levels of cognitive function despite having pathophysiological signs of what would otherwise be considered debilitating brain damage. The theory of the brain and cognitive reserve has been advanced to provide a mechanistic framework that explains such a discrepancy between behavior and pathology. As described in Chap. 4 (Translatable Models of Brain and Cognitive Reserve by Zeleznikow-Johnston, Burrows, Renoir and Hannan), the theory on brain and cognitive reserve posits that individuals have a certain level of capacity for a certain amount of damage they can sustain before showing behavioral symptoms (brain reserve) and that they differ in the ability to maintain cognitive performance despite equivalent levels of neurological insult (cognitive reserve). The individual differences in reserves may be due to different levels of neuronal redundancy for performing a particular task, ability to recruit brain regions to compensate for the loss of others, and ability to adopt
alternative cognitive strategies for performing the same task. The reserve construct emerged by epidemiological evidence showing that environmental factors, such as education, mental and physical activity, and social engagement, are associated with different rate of cognitive decline and onset of dementia suggesting that reserves are not static and could be influenced by environmental and genetic factors. Enhancers of reserves, such as environmental enrichment, voluntary exercise, and antioxidants, have powerful neuroprotective and pro-cognitive effects in experimental models of traumatic brain injury, aging, and neurodegenerative diseases. These improvements appear to be mediated by a combination of factors such as increased synaptogenesis; synaptic plasticity and hippocampal neurogenesis; glial and vascular modifications; elevated neurotrophic factors; and enhanced molecular regulators related to gene expression, epigenetic, and chromatin changes. The full understanding of the molecular mechanisms through which the reserves confer their beneficial effects allowed developing novel drugs called “enviromimetics” capable of acting upon molecular targets, in that they mimic or augment the natural enhancement that comes from positive environmental influences.

The model of brain and cognitive reserve is closely related to the concept of brain plasticity, the ability of the brain to rearrange structure and neural connections, allowing it to change with learning, to repair, and to compensate. Neural plasticity is central to memory and learning processes because it provides the brain with a lifelong possibility to change and adjust when facing environmental demands and stimuli. The focus on lifelong development is important for the cognitive reserve construct in particular, which, as an active process, is likely formed as a combination of baseline capacity, which is then subjected to modulation by multiple experiences and exposures throughout the entire life course. Through this process, an individual develops a level of cognitive reserve which in turn can mitigate the effects of pathology on the clinical diagnosis later in life. Chapter 5 (Cognitive Reserve: A Life-Course Perspective by Dekhtyar and Wang) discusses the contributors to cognitive reserve from various stages of the life course, including childhood, early adulthood, middle age, and late life and explores the life-course perspective to cognitive reserve in the framework of cognitive decline and dementia. Baseline cognitive abilities lay the foundation of reserve formation, which is subsequently enhanced by intellectual stimulation provided by educational attainment. By exerting continued demands on the brain, occupational tasks preserve this acquired buffer, much the same way as late-life social engagement and rewarding leisure activities. Ultimately, cognitive reserve can be conceived as a sum of mental, physical, and lifestyle inputs over the entire life course, and the brain ability to withstand the changes associated with aging will to a large extent reflect the gradual accumulation of these inputs. Although most of the neuropathology most likely applies to the older ages, the factors allowing the toleration of this pathology occur throughout the entire life course. In conclusion, it is becoming increasingly clear that variations among individuals in their ability to withstand age-related brain changes are ultimately dependent on their lifetime accumulation of mental, physical, and lifestyle inputs into cognitive reserve.
Chapter 6 (Neural Correlates of Brain Reserve: The Neuroimaging Perspective by Serra and Bozzali) describes how environmental factors have significant effects on brain resilience. Years of formal education, occupational attainment, cognitive, social, and physical activities, intelligence quotient, and memory performance represent the most relevant proxies of brain and cognitive reserves so that lifestyle acts on brain plasticity modulating the impact of neurological insults. Although interactive or additive effects among levels of reserves, biomarkers of neurodegeneration, and the risk to develop the clinical symptoms of neurodegenerative disease have been shown, investigating the relationship between cognitive enrichment and brain resilience may provide new therapeutic insights and interventions to prevent the evolution of or treat neurodegenerative disorders.

Studies of brain and cognitive reserves have also addressed the critical issue of the clinical implications of this approach and have indicated the extensibility of the reserve theory to a number of neurological conditions. A critical aspect pertains to the possibility to employ therapeutic interventions aimed at improving reserve in neurological patients as disease progresses. Interestingly, the use of complementary and alternative medicine approaches (from dietary natural products, such as herbal supplements and probiotics, to integrative nondietary approaches, such as meditation and music therapy) is rising among persons with different types of cognitive and health problems who desire a holistic, whole-person-based approach to treatment and a self-control over disease management. Chapter 7 (Non-pharmacological Approaches Based on Mind-Body Medicine to Enhancement of Cognitive and Brain Reserve in Humans by Crescentini, Fabbro and Aglioti) discusses the implications of reserves for clinical interventions based on the most practiced form of complementary and alternative medicine, the mind–body medicine. One of the most prevalent practices of mind–body medicine which integrates the mind, brain, body, and behavior with the intent to use the mind to positively influence psychophysical functioning and well-being is mindfulness meditation. Mindfulness is an attribute of consciousness that consists of being aware of and attentive to what is occurring in the present moment with a non-judgmental attitude of openness and receptivity. Mindfulness skills can be developed effectively through the practice in which any arising feeling, thought, emotion, and sensation is not attempted to be changed by the perceiver but is instead observed and accepted. The studies reported in Chap. 7 evidence that meditation may have neuroprotective effects, slow age-related brain degeneration, increase gray matter, and thus improve cognitive performance in healthy older meditators. On such a basis, mindfulness may be now considered an effective mental training strategy able to potentiate individuals’ reserve in the conditions of healthy aging and progressive neurological conditions, such as Alzheimer’s disease, multiple sclerosis, and Parkinson’s disease.

Synaptic plasticity and new synapse formation are assumed to be the basic mechanisms underlying the recovery of sensory-motor function affected by brain damage. Chapter 8 (Roles of Synaptic Plasticity in Functional Recovery After Brain Injury by Nagao and Ito) specifically elucidates the roles of synaptic plasticity, i.e., long-term potentiation and depression of synaptic transmission, in brain recovery
addressing at first the characteristics of synaptic plasticity in the intact hippocampus, cerebellum, and red nucleus. Then, in an experimental model of recovery of motor function it addresses the spinal, cerebellar, and cerebral mechanisms underlying the recovery of grasping movement after unilateral spinal cord injury. Furthermore, in an experimental model of recovery of sensory function, Chap. 8 describes the neural mechanisms underlying the recovery of somatosensory and vestibular functions after injury of their central or peripheral pathways. Finally, the chapter by Nagao and Ito reviews the recent progress in neurorehabilitative techniques, emphasizing the following items: The rehabilitation training should start as soon as possible after brain injury to facilitate neural plasticity and to avoid disuse muscular atrophy; the activation of large brain areas should be stimulated, and in addition to the conventional training of locomotion and postural control the training of skilled movement of daily life, as well as the mental (imaginary) training of movements and actions should be promoted; repetition of training with rests of an appropriate duration between training sessions should be performed; and new techniques, such as noninvasive transcranial brain stimulation techniques, neuroprosthesis, and regenerative medicine, including the induced pluripotent stem cell technology, should be developed to provide new tools for neurorehabilitation.

In fact, in the last years the transcranial magnetic stimulation (TMS and rTMS) and transcranial direct current stimulation (tDCS) have shown their ability to modulate brain activity in a noninvasive manner, as described in Chap. 9 (Integrated Methods of Neuromodulation for Guiding Recovery Following Stroke by Di Lorenzo and Koch). According to the stimulation parameters it is possible to facilitate or suppress brain activity with variable behavioral effects. While rTMS can create strong currents capable to depolarize neurons, tDCS modulates neuronal activity by weaker electric currents by influencing ion channels and gradients and hereafter the resting membrane potential. In particular, anodal tDCS leads to brain depolarization (facilitation) whereas cathodal tDCS results in brain hyperpolarization (inhibition). Notably, rTMS is employed for therapeutic purposes or as part of a neurorehabilitative strategy for stroke recovery. Cortical stimulation in stroke is meant either to correct maladaptive brain plasticity induced by the cerebrovascular accident or to enhance adaptive brain plasticity during rehabilitation. This aim may be realized by modifying locally cortical excitability or by changing connectivity in neuronal networks. Three types of post-stroke disorders (motor deficit, aphasia, and hemineglect) appear to benefit from cortical stimulation techniques. The therapeutic trials in these three conditions are commonly aimed at rebalancing interhemispheric dynamics, directly increasing the excitability of the ipsilesional hemisphere or decreasing the excitability of the contralesional hemisphere, which results in a reduction of its inhibitory influence onto the lesioned hemisphere (model of interhemispheric competition). The application of an excitability-decreasing paradigm to the contralesional left posterior parietal cortex may represent a putative therapeutic intervention in the treatment of post-stroke neglect in the post-acute phase.

How do brain networks anticipate, endure, respond, and adapt to limit the consequences of a stroke? As described in Chap. 10 (Resilience of Brain Networks
After Stroke by Dirren and Carrera), a focal brain lesion triggers widespread alterations of connectivity between even distant brain regions. The study of changes in connectivity distant to the lesion has led to a new approach in the understanding of the neural correlates of brain function and recovery. In fact, brain connectivity architecture is organized not only to maximize functional performances but also to limit the potential consequences of a lesion. In healthy subjects brain networks are built with a limited number of highly connected nodes to promote an optimal balance between specialization and integration of information. Prelesional organization may limit the impact of a lesion on brain networks and its subsequent clinical consequences, and genetic and environmental factors modulate the organization of the human connectome and possibly its resilience to focal insults. Indeed, the anatomical location and the position of strategic nodes in the connectome prevent major neurological deficits, even when these hubs suffer from a targeted attack. Widespread changes in the organization of brain networks are triggered by the lesion and modulation of network organization is clinically relevant during the whole process of recovery, reflecting the mechanisms of plasticity and repair. This effect can be understood as “connectional” diachisis or “connectomal” diachisis defined, respectively, as the changes in coupling between the two nodes of a specific network or in the totality of brain connections. Clinically, the reduction in interhemispheric coupling after stroke seems to be particularly relevant. Furthermore, the process of resilience includes response and adaptation to the lesion. Although recent evidence points to the importance of changes in networks configuration during recovery, the changes of brain networks in a reorganized architecture not necessarily will lead to favorable outcome. Understanding how these changes are associated with restoration of function may open new therapeutic options to improve clinical outcome.

Depression has a multifactorial etiology arising from environmental, psychological, genetic, and biological factors. In recent decades, the advent of computerization and the progressive urbanization have led to a strong reduction of the levels of physical activity so that the consequent sedentary lifestyle combined with other factors such as alterations of the sleep/wake cycle, abuse of psychotropic substances, and chronic stress favors the onset of mood disorders and depressive syndromes. Furthermore, even a poor diet impacting on brain plasticity may be a risk factor for the onset of depression. Research has indicated that the main factors involved in the pathogenesis of depression may be neurotransmitter and neurotrophic factors imbalance, hypothalamic-pituitary-adrenal axis disturbance, deregulated inflammatory pathways, increased oxidative damage, neurogenesis dysfunction, and mitochondrial disturbance. In addressing the several hypotheses on the pathogenetic mechanisms of depression, Chap. 11 (Functional Role of Physical Exercise and Omega-3 Fatty Acids on Depression and Mood Disorders by Farioli-Vecchioli and Cutuli) focuses the effects of physical exercise and nutritional factors, such as omega-3 fatty acids, on neurotransmitters, neurotrophins, hippocampal neurogenesis, and neuroinflammation. The reported findings emphasize the capacity of omega-3 fatty acids and exercise to elevate the capacity of the adult brain for axonal growth, BDNF-related synaptic plasticity, neurogenesis, and
cognitive functions either under normal and challenging conditions. Given the noninvasiveness and safety of diet and exercise, nutritional and physical interventions represent treatments of choice to enhance cognition, mood, and brain plasticity in depressed patients.

Interestingly, a large number of neurological functions are deeply influenced by the gonadal hormones. Sex steroids prime irreversible life long-lasting changes, referred as “brain masculinization” of specific hypothalamic nuclei, which directly regulate sex functions, and other brain areas involved in the regulation of the endocrine system but also in behavior and, possibly, selected cognitive capacities. In the course of life, the changes in circulating or locally produced sex steroids connote reversible neurophysiological functions and contribute to sex differences in behavior, endocrine functions, and responses to pathological events. In fact, the manifestation of several neurological disorders correlates with specific changes of the synthesis of estrogens. These changes impact the metabolism of neurotransmitters, regulate the activity of glial cells, modulate microglia immune functions, and are involved in brain ability to recover after insult. Chapter 12 (Estrogen Neuroprotective Activity After Stroke and Spinal Cord Injury by Maggi) reviews the current knowledge on the mechanistic determinants of the brain sexual dimorphism and on the role of sex hormones in the modulation of brain recovery after stroke and injury in which the sex determines a differential incidence. Chapter 12 describes the well-established relationship between endogenous and exogenous sex hormones and many neurological disorders, such as epilepsy, chorea, and neurodegenerative diseases and emphasizes that the appreciation of the sexually dimorphic functions of the brain may facilitate the development of efficacious therapies for sex-prevalent disorders. Furthermore, a better understanding of the molecular mechanisms involved in female resistance to injury may shed novel light on their etiology and provide new avenues for more targeted therapeutic interventions.

In addition to biomedical factors, more recent research has begun to examine psychosocial influences and processes promoting functional recovery. Among the most important psychosocial aspects of functional recovery are the meanings that people with brain injuries assign to their situation (e.g., the injury, what recovery requires, what they are capable of) and the coping strategies they use in their efforts to achieve functional recovery. Chapter 13 (Appraisals of and Coping with Acquired Brain Injury: Resources for Functional Recovery by Park) presents a model of meaning and coping that highlights the centrality of meaning appraisal and coping in adjusting to adverse events. This model is then applied to adaptation to acquired brain injury by drawing on relevant theories and empirical findings. It is increasingly evident that how individuals manage brain injury and its sequelae influences functional recovery and determines the effects of the injury on productivity, social activity, emotional stability, and quality of life. Chapter 13 addresses the roles of appraised meanings, violations, coping, and meanings made in promoting functional recovery. Coping is driven by efforts to alleviate distress that are produced by violations between survivors’ global meaning and their appraisal of the brain injury and its implications. Interestingly, long-term well-being following brain injury may require a mix of restoration-oriented coping, loss-oriented coping,
and meaning-focused coping so that both emotion-focused coping and problem-focused coping may be needed simultaneously to deal with different aspects of recovery. Even the reduction in violations between one’s life goals and one’s actual performance is central to functional recovery. This reduction in violations will largely be accomplished by letting go of goals that are no longer tenable and developing new, more realistic goals in light of the brain injury. The creation of a new identity and development of new perspective about life and expectations about the future are the basis of long-term adjustment following brain injury. Given the pervasive roles played by meaning making throughout the recovery process, increased efforts should be made to incorporate this perspective into existing rehabilitation programs.

Despite the conceptual utility and broad application, brain and cognitive reserve construct does not account for the variability in traumatic brain injury or stroke outcomes. In the clinical practice, it is assumed that the behavioral changes after traumatic events are most often related to cognitive function impairments due to brain damage, but increasingly data report that these manifestations are underpinned by a variety of mechanisms partially related to personality traits and environmental context of the patient. New multidimensional approaches take into account the complexity and dynamic relation between reserve construct and post-damage neurobehavioral changes associated to negative consequences of neurological damage on functional outcomes, caregiver distress, and social reintegration following the traumatic event. The depression often reported after brain damage has been associated with emotional and cognitive disabilities, reduction of quality of life, time of hospitalization, nonadherence to treatment schedules, repeated use of community health services, and increase of mortality. The attempts made to identify the predictors of depressive symptoms after brain damage have revealed the role of physical disability, damage severity, and cognitive decline. The few studies focusing on psychological aspects as predictors of depressive symptoms have indicated that premorbid personality dimensions may be associated with the development of post-damage depression. In this framework, environmental factors, emotional aspects, and premorbid personality traits have increasingly been considered as potential moderators of traumatic brain damage. Chapter 14 (Premorbid Personality Traits and Brain Recovery: Another Aspect of Resilience by Laricchiuta, Markett, Reuter, and Montag) defines the role of premorbid personality and attachment style within the context of resilience against the detrimental effects of traumatic brain damage on adaptive functioning. Premorbid personality features appear to be a relevant factor of resilience that predicts brain recovery efficiency or predisposes to neurodegenerative diseases. The biopsychosocial approach should be encouraged in the management of neurobehavioral difficulties, since this approach apprehends behavioral changes as a result of complex and dynamic interactions among neurobiological (type and severity of injury, time post-injury), social (psychosocial history, family context), personal (medical history, personality traits, and pre- and post-morbid coping strategies), and environmental factors (problematic and anxiogenic situations related to brain injury).
As repeatedly described, the often co-present motor, cognitive and affective symptoms dramatically affect the patient’s quality of life and the project for self-realization. Changes may occur suddenly, such as after stroke or traumatic brain injury, representing a highly stressful condition for the affected individuals and their family. Indeed, brain injury may provoke a kind of discontinuity in the feeling of self and requires cognitive reintegration to regain a unitary image of body and of the self. The psychopathological symptoms and maladaptive coping styles frequently observed after brain injuries negatively affect the therapeutic outcome. Psychological reactive mechanisms and premorbid cognitive–affective coping style are reported to play a significant role in the patient’s recovery processes. Among the various factors potentially modulating the effects of therapeutic interventions, the study of the possible role of psychic defense mechanisms aroused poor interest.

Within the traditional psychoanalytic framework, defense mechanisms are described as psychic operations adopted to keep far from consciousness aspects of emotional experience that may cause severe anxiety and mental sufferance. After brain injury, patients may present stable responses conditioned by the adoption of particular defense mechanisms (regression to maladaptive behavioral schema, repression of emotions, denial…) that significantly affect compliance with treatment. In psychological terms, the tendency to use a particular defense mechanism may rely upon the premorbid cognitive/affective style of functioning. Mechanisms of psychological defense, such as repression/denial, may be active in patients who after brain injury show emotion/affective dysregulation and tend to use poorly efficient coping strategies. Moreover, repression/denial can influence the patient’s ability to correctly acknowledge the illness and its consequences, in so way hampering the productive participation to the rehabilitative program and social reintegration.

Chapter 15 (Psychodynamic Factors of Recovery After Brain Injury: A Role for Defense Mechanisms? by Costa, Gullo and Caltagirone) discusses the possible role of psychodynamic mechanisms in the recovery after brain injury and provides some clues for the purpose of the clinical intervention. To improve the clinical approach to brain-injured patients, the psychological and psychodynamic processes potentially implied in the adjustment response should be correctly recognized and adequately treated, even if valid and reliable tools to assess defense mechanisms in brain-injured individuals are still scarce.

Even a cursory glance at the chapters makes it obvious that there are different views and approaches to the same topic, an aspect of book with which I’m very pleased. The pluralism linked to multifaceted approaches is a good route to take, given the state of the art, and serious attention needs to be paid to all of these approaches (from the most molecular to the most psychodynamic). The fact that there are so many overlapping themes among very different approaches speaks to the interdisciplinary nature of the studies on brain recovery. I hope that this book might stimulate all of us to be more interdisciplinary and to rise to challenge to try to answer difficult questions. All things considered, it is important to emphasize
how the most effective strategies for brain recovery should combine multifactorial (from molecular to psychological) manipulations to drive the most robust and functionally appropriate plasticity.

I would like to thank all the contributors and Springer Press for working with enthusiasm and dedication on this project. Identifying the most prominent factors that contribute to successful injury-induced neuronal plasticity is the current challenge that has to inform future studies on brain recovery to expose new avenues for neuronal repair strategies.

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