Books Received


Books Reviewed


Every four years I offer a graduate level course on neural plasticity. I select a recently published book to serve as a common foundation for the neuroscience students to read before they dive off into the empirical literature, current reviews and more recently CD ROMs and web-based material. I have used edited books in the past because they can offer a breadth of material and perspective. In contrast a single authored book promises a coherent and comprehensive vision and for that reason I was positively predisposed towards Aage Moller's "Neural Plasticity and Disorders of the Nervous System".

Scientist and author of eight previous books, Moller has a great legacy in auditory research. Notably, he pioneered cochlear nucleus research in the late 60's with techniques like reverse correlation that much later became commonplace. In the 1980's, he started to work on intra-operative monitoring and he is considered the expert in that field. His related work identifying the generators of the auditory
brainstem response is also well known. About a decade ago his attention shifted to tinnitus, hyperacusis, and chronic pain. Recently his work has become more clinical in orientation, generally less mechanistic, and useful at the level of undergraduate or early graduate teaching.

This book is organized into six chapters; an opening chapter on neural plasticity followed by a chapter on the anatomy, physiology, pathology and pathophysiology of nerves. The remaining four chapters cover sensory systems, pain, movement disorders and cranial nerves. Each of those chapters deals with the anatomy, disorders and pathophysiology for each of the chapter topics. There are several important features in the book such as the functional role of non-classical pathways, the fact that descending pathways can modulate the neural traffic in ascending pathways, inclusion of recent progress using transcranial magnetic stimulation, electrical stimulation, and microvascular decompression operations. A central theme to the book is that it is difficult to diagnose certain neuropathological disorders because the anatomical location of the physiological abnormality that causes the symptoms is often different from the anatomical location to which the symptoms are referred, and that hyperactive disorders such as tinnitus, paresthesia, vertigo as well as other disorders are caused by neural plastic changes to the system rather than the direct effects of an insult.

The use of the term neural plasticity is problematic because it has become like the term addiction, a term so blurred, its scientific value is nullified. People speak of addiction to drugs, addiction to sex, addiction to work, addiction to food and so on. These different examples of repetitive behaviour may have some mechanisms in common, but they may not. Hence use of the common term is potentially very misleading. Neural plasticity has become a short form for any and all changes in brain function including those mediated by non-neuronal glial cells. The current use of the term neural plasticity encompasses developmental trajectories, those processes associated with learning, memory, and experience, including drug taking, neuropathological states themselves and the consequences of damage. Neural plastic mechanisms include, but are not limited to, neurogenesis, dendritic hyper- and hypo-trophy, synaptic growth and regression, the unmasking of dormant synapses, axonal sprouting, the myriad ways to alter neurotransmitter and hormone levels, receptor modulation, channel insertion or inactivation, phosphorylation of intracellular messengers and intracellular cascades, gene transcription, protein synthesis, post-translational modifications, and apoptosis. So when someone says neural plasticity is involved they might as well say the brain is changing. This says nothing of value. Those of us who study behaviour understand that one or two word terms like innate, inborn, learned, or neural plasticity cannot be used as explanations for anything, and this is perhaps the biggest weakness of the book.

The book also contains an annoying amount of repetition; for instance in at least four separate locations in the book the author tells us that the distinction between pyramidal and extrapyramidal motor systems is no longer valid. While true, we only need to be told this once, maybe twice. Perhaps the most egregious error that I encountered is found on page 314. The author states that tolerance develops to deep brain stimulation and that “Deep brain stimulation...causes the expression of neural plasticity or the kindling phenomenon as it has been called”. The kindling phenomenon is an example of brain sensitization that specifically refers to the progressive intensification of epileptiform activity and/or seizure activity. Deep brain stimulation does not normally result in seizure activity and to the extent that tolerance to DBS occurs, it has nothing whatsoever to do with kindling. Unfortunately this book does not advance our understanding of the brain or its function but rather offers a superficial overview of neural plasticity and disorders of the nervous system. I will be choosing a different book for my graduate course.

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This book is the second volume in the “Progress in Epileptic Disorders” book series, edited in collaboration with the journal “Epileptic Disorders” and published by John Libbey Eurotext. The book is the fruit of a workshop, designed as a discussion forum, with the participation of experts from all over the world. The book is divided into seven sections with a total of 18 chapters. The first section talks about potential definitions of generalized epilepsy. The second section offers a clinical and a physiopathological description of tonic seizures. The third section approaches absence seizures, describing mainly the physiopathological mechanisms. Section four describes some models of myoclonic epilepsy, physiopathology of these types of seizures and an interesting chapter regarding primary reading epilepsy. Section five reviews clinical and physiopathological aspects of primary versus secondary tonic-clonic seizures. Section six contains only one chapter and reviews the cortical and centrencephalic theories. Section seven has an interesting chapter about why some antiepileptic drugs control certain types of seizures and aggravate others. Finally section eight has a chapter criticizing the available systems of seizures classification.

This is not the first book regarding primary generalized epilepsy; I think that the main purpose of the book was to create controversy about the current evidence regarding this type of seizure disorder. The purpose of the panel was to review the current evidence on primary generalized epilepsy emphasizing the new advances including clinical, EEG and imaging aspects. Secondarily the book creates controversy about a very well known dogma in epilepsy. In recent years we have increasing evidence that some of the syndromes that classically have been classified as generalized epilepsy may have focal expression. One of the chapters that exemplify very well this controversy is the chapter regarding primary reading epilepsy. This rare type of epilepsy has been recognized for years as a generalized type of epilepsy, although the clinical expression and the advances in the imaging techniques shows that it may show focal expression. This chapter is very interesting and highly recommended for readers. Another useful chapter is the one that talks about the worsening of some type of seizures with specific medications. This is an important topic in epilepsy and is well reviewed in the book. Section two on tonic seizures is also very valuable. The three chapters in this section review different aspects of tonic seizures and represent a good section in the book. The book’s final chapter is a good review of the