

may not be the case for all of us. The roles of neuronal dendrites and glial cells are little discussed. It is very strong in cellular electrophysiology and in neuromodulatory mechanisms.

Overall I consider this to be an outstanding, easily readable, and quite up-to-date overview of fundamental neurobiology. This book is quite useful for the clinician who wishes to have a digestible presentation of basic neuroscience as a prelude to understanding neurological disease.

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NEUROPROTECTION IN CNS DISEASES. 1997. Edited by P.R. Bar and M. Flint Beal. Published by Marcel Dekker Publishers. 585 pages. \$C241.00

Neuroprotection has come of age. The 585 pages of this volume attest to this. The first part deals with the basic components of the nervous system, including neurons, glia and the brain blood barrier, key substances and in vitro and in vivo models of neuroprotection. The second part deals with acute neurological diseases and the third with chronic neurological diseases.

In addition to the specific information in each chapter, several themes emerge: 1) Most substances essential for life also have potential for damage. Calcium ions, neurotransmitters and cytokines fuel normal function, but released from tight control, harm the very environment from whence they come. 2) Glia are much more than "glue" for the nervous system. They guide the development of neurons, maintain their function and help their repair. Additionally, glia and neurons may communicate, neurons responding to intracellular calcium waves travelling through astroglia. 3) A large gap remains between the laboratory and the bedside. In stroke, over 100 compounds reduce ischemic damage experimentally, but none has yet been proven effective in randomized clinical trials; but the time is nearing.

In a book of 29 chapters one can expect uneven quality, and one finds it. However, a common feature is succinctness and emphasis on basic principles. Thus, one finds gems such as Detsai et al.'s chapter (16) on glia, Marshall and Kiebertz chapter (19) on the design and interpretation of clinical trials in neuroprotection and Vanderworp et al.'s chapter (20) on the medical treatment of acute ischemic stroke.

Overall the book is too bulky, specialized and expensive to recommend to the practising neurologist, but it can be recommended for the neurological library as a good introduction and inventory of a fast-moving field.

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NEUROINFLAMMATION. 1998. Edited by Paul L. Wood. Published by Humana Press. 375 pages. \$C188.50

Most dogmas are wrong. Until recently, the brain was considered an "immunologically privileged" organ. This book shows that it is not, and that neuroinflammation may mediate the damage in a host of neurological disorders, from multiple sclerosis to Alzheimer's disease. The volume includes five parts: 1) Microglia 2) Acute phase proteins 3) Cytokines 4) Free radicals 5) Miscellaneous mediators, including adhesion cell molecules, nitric oxide and cyclo-oxygenase.

We learn that microglia are critical in the defence and clean-up of the brain, but if chronically activated, may lead to neurodegeneration. Interestingly, microglia can be activated remotely in areas devoid of neurodegeneration, also, microglial activation precedes neuronal loss in Down's syndrome.

Contrary to received wisdom, all components of both the classical and alternative complement pathways are found in the brain and cerebral spinal fluid. Sustained complement activation may be key process in myasthenia gravis, multiple sclerosis and Alzheimer's disease. Nitric oxide is the Dr. Jekyll and Mr. Hyde of the brain, essential physiological modulator in health and deadly neurotoxin in disease.

The utility of this volume lies in bringing together research dispersed in a myriad of specialized journals and focusing it on neurodegeneration. While some details may escape a neurologist's comprehension, the main message is clear enough: neuroinflammation is important in brain injury and neurodegeneration and we must think more about common mechanisms and less about distinct diseases.

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NEONATAL CEREBRAL ULTRASOUND. 1997. By Janet M. Rennie. Published by Cambridge University Press. 248 pages. \$C117.00

This book has 12 chapters covering the physical principles of sonography and doppler ultrasound; the choice, maintenance and use of equipment; the normal and pathological appearance of the brain; and appearance of both the term and the immature brain. Additionally there is an attempt to correlate ultrasound features and prognosis. A glossary and an index are also included.

The first chapter contains adequate information and is easy to read. With the current rapid development of sonography, some of the information on equipment in Chapter 2 is bound to be somewhat out-dated in a few years, but the basic principles, particularly those dealing with quality control and artifacts, will continue to be of value for a longer period of time. I was surprised however, to see in the photos under the headline "Safe use of ultrasound equipment", that the examiner holds the transducer to the fontanelle of the infant without a supporting finger on the adjacent bone. A sudden movement of the infant's head or of the transducer may result in unnecessary pressure on the fontanelle.

In Chapter 3 "Normal appearance", unfortunately only the conventional planes acquired through the anterior fontanelle are described. Other acoustic windows, such as the posterior fontanelle for additional coronal views, the sphenoidal fontanelle and the squamosal suture for axial views of the mesencephalon and third ventricle, and the mastoid fontanelle for coronal and axial views of the posterior fossa contents are needed to evaluate the complex anatomy and the pathology of the brain. These added views are obviously easier in the very young infant due to the size of the fontanelles and state of the sutures.

Chapter 4 gives a good overview of the immature brain, which is important to the sonographer, and emphasizes the fact that gestational age is of paramount importance to the interpretation of the ultrasonogram. For example, a smooth cortex may indicate lissencephaly in the full term infant, but may reflect a normal developmental stage in the very premature infant.

Chapter 6 gives a thorough introduction to the complex issue of