are not discussed. In addition, the Parkinson Foundation of Canada's address is not correct.

My review of this book is therefore mixed. The objective of the book is partially met with good information about Parkinson's disease and its treatment. Unfortunately the style of writing, the inappropriate focus on the potential genetic etiology of this disorder and the lack of "Canadian content" take away from its strengths.

> Mark Guttman, Toronto, Ontario

DIGITAL EEG IN CLINICAL PRACTICE. 1996. By Peter K.H. Wong. Published by Lippincott-Raven Publishers. 296 pages. \$C111.00

I can think of no-one whose background, interests and capabilities would better equip him or her to write a book on digital EEG than those of Dr. Wong. Therefore, this work represents an entirely appropriate match of ability and orientation with subject matter. Indeed, the illustrations are surprisingly clear given the quality of many printers attached to digital EEG apparati.

A future edition might benefit from co-authorship with an electroencephalographer who is less familiar with the technical aspects of digital EEG than is Dr. Wong. This would create a more "user friendly" introduction which might benefit from instructive diagrams and a more practical "how to do it" approach. Prominent in such an introduction might be guidelines as to how a digital EEG product should be evaluated by the potential buyer. This applies not only to a demonstration of the full capability of the setup, but also a complete detailing of its limitations.

One such example is the annoying vertical lines which makers and vendors of such printers feel all electroencephalographers treasure. Dr. Wong had no option but to include several examples of such print-outs.

The "clinical examples" could be subdivided by subject and so labelled. Most legends do not appear on the same page as the figures, requiring the reader to flip back and forth. This difficulty is compounded by designating montages as runs 1, 2, 3 and thus requiring the reader to memorize these after they are indicated on page 23.

Although one may quibble with some of the legends, the illustrations are generally well chosen and do illustrate how appearances may be altered by montage, sensitivity (not gain), filtering and paper speed.

How digital data are handled by his own department (Chapter 3) is a valuable inclusion.

This book will be of interest to those neurologists with prior knowledge of electroencephalography and will be a useful edition to the libraries of departments with digital components.

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PATHOGENESIS AND THERAPY OF AMYOTROPHIC LATERAL SCLEROSIS. Advances in Neurology, Volume 68. 1995. Edited by Georges T. Serratrice, Theodore L. Munsat. Published by Lippincott - Raven. 312 pages. \$C150.00

This text represents a compilation of papers presented at a conference held on October 28-29, 1994, in Marseille, France addressing the issues of pathogenesis and therapy in amyotrophic lateral sclerosis (ALS). It is remarkable in the rate of publication following such a conference and hence still remains quite current. As such, it is a text that will find a place in the libraries of clinicians interested in the treatment of ALS, clinician/scientists attempting to frame concepts of etiopathogenesis, and to basic scientists attempting to understand the clinical relevance of studying ALS.

The text, on the whole, is well-written, topical and adequately referenced. There are several chapters that are outstanding and present excellent reviews. The chapter by Munsat on trial designs is a good, balanced overview and presents a historical perspective of drug trials in ALS. This chapter should be read in the company of those by Brooks et al. and by Meisinger et al. on attempts at quantitation of disease progression and regional onset in ALS. The chapter by Pouget et al. on the diagnosis of ALS is perhaps the best to date that I have reviewed. My only concern is the inadequacy of the discussion on primary lateral sclerosis (PLS), and the omission of key references by Pringle et al. (Brain, 1992; Canadian Journal of Neurological Sciences, 1990) and Hudson et al. (Brain Research Bulletin, 1993). These three references delineated the clinical, pathological, and diagnostic criteria for PLS, and yet are not mentioned at all in this chapter. Rowland's paper provides some useful insight into the diagnostic difficulties that arise in the finding of a paraproteinemia in a patient with motor neuron disease. My only concern with the chapter was a paragraph on page 97 on trangenic models of neurofilament expression. While this is included in a section on anti-neurofilament antibodies, it seemed out of place and was not brought into the relevance of the overall chapter. The chapter by Rothstein on the excitotoxic mechanisms of neuron death in ALS, and particularly the glutamate-induced neurotoxicity, is well-written, clear and concise.

These positive features are off-set by a number of minor annoyances within the text itself. The organization of the text is somewhat unusual. Clinically-relevant material is left to the latter half of the text. While useful for researchers this may not be so useful for the mainstream neurologist who wishes to pick up the text and have an initial overview of the diagnostic difficulties and the classification of ALS prior to reading about pathogenesis. The paraproteinemia and immune-based chapters are scattered in that three are grouped (Appel, Drachman and Jeagar) and then 3 chapters later appears the chapter of Rowland. While the chapter by Mitsumoto and Pioro discussing animal models spends considerable time discussing the Wobbler mouse, the editors have stated in the preface "the Wobbler model has been extensively investigated, but its relevance to ALS is a concern". Indeed, it is a useful model for understanding pathogenesis of motor neuron dysfunction, and one of the most useful models to date for therapeutic trials. It is disconcerting from my point of view to find that the aluminum neurotoxicity models are scarcely mentioned, and when discussed, inaccurately. As stated in the chapter, "chronic encephalopathic signs" were not described in the model and hence this section is inaccurate. Similarly, the equine model was