Contemporary Clinical Neurophysiology (Supplement No. 34 to Electroencephalography and Clinical Neurophysiology) Edited by W. A. Cobb and H. Van Duijn. (Pp. 577; illustrated; price not stated.) Elsevier Scientific Publishing: Amsterdam. 1978.

This book comprises the invited lectures which formed the basis for individual contributions to the Round Tables and Symposia at the Ninth International Congress of Electroencephalography and Clinical Neurophysiology in Amsterdam in 1977. Three main topics are covered — Electroencephalography, a short section on Therapeutic Electrical Stimulation, and Electromyography. The former topic incorporates data processing and automatic reporting in clinical EEG; behavioural correlates of evoked potentials and slow potentials, attentiveness and consciousness in epilepsy and experimental models of epilepsy. The section on electromyography covers motor control, structure and function, central EMG, neuromuscular physiology and pathophysiology. The papers are in general short, informative, and clearly illustrated. In summary, the book is an excellent source of information on clinical neurophysiology.

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leaves the cerebellum by the interpositus nucleus to red nucleus and reticular formation, possibly playing a role in holding a position against loads and in stopping a movement. Kornhuber has already suggested basal ganglia and cerebellar differences between ramp and ballistic movements. Some of the reports in this book reach contradictory conclusions. It is not feasible to abstract the different views but the interested reader should study the book carefully as it gathers together 22 excellent papers from the main workers in the field. Neurologists trained 30 years ago may have a sense of déjà vu with regard to many of the concepts, re-emphasised rather than newly discovered, and will see little merit in abandoning clinical terms for those of servo engineering, as suggested in one chapter, but the opportunity to time delays of responses traversing cortex and cerebellum promises new techniques for clinical neurophysiology and for the study of drug actions on the intact nervous system. Professor Desmedt is to be congratulated for this valuable compendium.

J. A. SIMPSON

Pathophysiology of Spinal Cord Trauma By J. L. Osterholm. (Pp. 212; illustrated; \$19.50.) Charles C. Thomas: Springfield, Illinois. 1978.

This is a monograph in the Bannerstone Division of American Lecture Series in Neurosurgery on the studies carried out since 1968 by Professor J. L. Osterholm and his colleagues at the Thomas Jefferson University, Philadelphia.

The spinal cord is very sensitive to mechanical trauma, not because its tissues are especially fragile since it is not common for even a major fracture dislocation to lacerate the cord, but because of a process termed necrotising haemorrhagic necrosis that starts in grey matter, and later spreads to white matter, and eventually to the entire cord.

The response of the cord to injury varies with time, force, the particular segment of cord injured, and the species of experimental animal used. This has inevitably produced conflicting reports in the literature. An experimental model that produces a standardised injury—morphological and biochemical—to the spinal cord was, therefore, required and subsequently established by

the author and his team.

There is evidence that blunt injury to the cord induces increased activity in the noradrenaline bulbospinal pathway and at the same time releases sufficient local noradrenaline to cause toxic effects, possibly mediated through the microcirculation. Current evidence, therefore, strongly suggests that cord damage is transmitter-mediated and raises the hope that the lesions may be either prevented or at least minimised by drug treatment.

The most disappointing aspect of this book is the relative lack of information about the physiology (function, metabolism, and blood flow) of the spinal cord. Considerable detail is given about the response of the cord to mechanical trauma and yet basic information about the response of the normal microcirculation to changes in blood gases, blood pressure, and various pharmacological agents in the preparations used by the authors is scanty. This inevitably means that some of the conclusions reached by the author are rather speculative. In spite of this the monograph is informative, and should be of interest to anyone concerned with injury to the spinal cord.

D. I. GRAHAM

The Pathogenesis of Hypertensive Encephalopathy By L. M. August. (Pp. x+111; illustrated; DM68.00.) Acta Neurochirurgica, Supplement 2. Springer-Verlag: Vienna, New York. 1978.

Variation in the calibre of the pial vessels of animals with accelerated phase hypertension was first described in the mid 1950s. It was found that when the blood pressure became excessively high a pattern of alternate constriction and dilatation sometimes developed in the pial arterioles—the so-called “sausage-string” appearance. Further studies suggested that the narrowed segments were in “vasospasm” which in turn caused a reduction in the local cerebral blood flow and ultimately caused the symptoms and signs of hypertensive encephalopathy. It was the use of different methods by different investigators that led to the alternative hypothesis, namely that a high intraluminal pressure is the most likely cause of the encephalopathy.

This monograph describes a series of experiments using a closed window technique to study the reactivity of

Cerebral Motor Control in Man: Long Loop Mechanisms Progress in Neurophysiology, Volume 4. Edited by John E. Desmedt. (Pp. 392; illustrated; Sw fr 118.) S. Karger: Basel. 1978.

Starting with the observations of P. H. Hammond in 1964, there have been a number of papers on muscular responses to stretch or relaxation which indicate that the monosynaptic spinal stretch reflex is followed by at least two responses with latencies suggesting that they are also reflex, though it is difficult to dissociate the later responses from voluntary reactions. The timing of these responses, and their modification by local anatomical lesions, indicate that they are reflexes mediated by “long loops” traversing supraspinal parts of the nervous system. Correlation of the human studies with experimental work on primates suggests that one passes via parietal association cortex to pontine nuclei and thence to cerebellum, leaving by the dentate nucleus and so to the major motor outflow; another, ascending by spinocerebellar fibres and from sensory cortex to pontine nuclei,