highly regular over consecutive recording periods. Indeed, movements made by patients at maximum frequencies were qualitatively similar to movements made at the same frequencies by control subjects.

Most striking was the inability of all patients to produce smoothly alternating movements at or below frequencies of about 1.0 Hz (fig 1, middle, lower sets of records). At these frequencies, movements became typically irregular with abrupt changes in velocity throughout the movement. The frequency at which this breakdown in movement performance occurred varied across patients, ranging from 1.0 to 0.6 Hz. At 0.5 Hz movements became considerably irregular in all patients with prolonged periods of rapid velocity transients.

In three patients, the relation between movement performance and visual feedback was examined. Fig 2 shows velocity and acceleration recordings for "eyes open" and "eyes closed" conditions. As described above, movements became irregular at approximately 0.5–0.75 Hz with periods of abrupt transients, particularly noticeable in the acceleration records. In both patients shown in fig 2 (Cb1, Cb2), removal of visual feedback during slow alternating movements (0.5, 0.75 Hz) did not improve movement performance.

One of the cardinal signs of cerebellar dysfunction is an impairment in the ability to produce rapid, alternating movements about a given joint. Movement trajectories are, however, relatively smooth despite a reduction in maximum alternating frequency. The results presented here demonstrate that, despite bradydiadochokinesia, cerebellar patients have more difficulty producing smooth alternating movements when made at low frequencies compared with movements made at higher frequencies. This supports recent findings showing that patients with mild upper limb ataxia have difficulty in performing long duration (> 600 ms), discrete movements.^{3,4}

The highly irregular movements at frequencies under 1.0 Hz are similar to those observed during sine wave tracking following dentate cooling in non-human primates⁵ and during slow ramp tracking in cerebellar patients.⁴ In these studies pursuit tracking tasks were used. In our study, target zones remained fixed and thus the task did not require moment to moment matching of the handle cursor with a continuously moving target. Since, in addition, removal of visual feedback did not abolish movement irregularities, it is difficult to attribute the observed breakdown in motor performance to visually mediated corrections arising from a mismatch between actual and intended arm positions. It has been shown that, in cerebellar patients, removal of visual feedback can lead to an improvement in pursuit tracking performance.⁶ The results presented here in the "eyes closed" condition, however, suggest that this is not the case for alternating movements made between stationary targets. This would support the view originally put forward by Holmes⁷ and later confirmed by others⁸ that cerebellar dysmetria is not dependent upon visual feedback.

In conclusion, these findings underline the importance of clinical evaluation of both slow and rapid movements during routine neurological assessment. In particular, testing of slow alternating movements such as elbow pronation and supination may reveal

significant upper limb impairment in patients who may otherwise show only minimal cerebellar signs.

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BOOK REVIEWS

Recent Advances in Clinical Neurology 7. (Series: Recent Advances). Edited by CHRISTOPHER KENNARD. (Pp 245 Illustrated; Price £34.95). 1992. Edinburgh, Churchill Livingstone. ISBN 0-443-04570-4.

This volume continues the tradition of excellence which has accrued from the impact of previous editions in this series. The aim of this series is to provide a collection of succinct and timely reviews of particular growing points in clinical neurology written by leading authorities in the relevant subject areas. This aim has always been admirably achieved and the current volume, reviewed here, carries this forward. All the chapters have many strengths and while I feel it would be invidious to single out particular contributions to any great extent I feel compelled to make the following specific comments.

There is something in this book for everybody ranging from junior staff just beginning their training in neurology to the established consultant. From my own point of view, I was particularly pleased to see

clear descriptions of the syndromes of chronic paroxysmal hemicrania and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). In my view, both these conditions are still seriously under recognised, and in view of the important therapeutic implications of both disorders, should be more widely known. Indeed, considering the paucity of neurological services in the UK, I could not help feeling that the chapter on inflammatory demyelinating neuropathy should be widely read by general physicians.

The expanding areas of the general role of magnetic resonance in neurological practice and prion related disease are presented in a balanced way, and it is useful to have a current review of expanding impact of mitochondrial pathology in neurological disease. The mechanisms subserving the control of eye movements are notoriously complex, but the editor has found two authors who have been able to give an admirably lucid summary of this difficult area.

All in all, this volume is highly recommended. A copy should be available for day to day reference in all departments of neurology. It will also find a worthy place in the libraries of district general hospitals lacking on-site neurological units and I would, perhaps provocatively, suggest that before being deposited in such libraries, the volume might first be circulated among the general physicians to read the chapter on inflammatory demyelinating neuropathy!

J D MITCHELL

Duchenne Muscular Dystrophy: Animal Models and Genetic Manipulation. Edited by B A KAKULAS, J MCC. HOWELL and A D ROSES (Pp 308; Price: \$112.50.) 1992. New York, Raven Press. ISBN 88167-938-0.

Once again Byron Kakulas' Co-editors and Publishers are to be congratulated on the timely publication of what is now the second in a series of monographs devoted to Duchenne and Becker Muscular Dystrophy. The format is similar to that of their earlier volume (Pathogenesis and Therapy of Duchenne and Becker Muscular Dystrophy; 1990) and consists of a series of lectures given by experts in the field followed by, what appears to be, a verbatim transcription of the discussion which followed each paper.

In Part 1, the localisation, distribution and function of dystrophin is reviewed in the light of developments from their earlier volume. In Part 2, the potential of myoblast transfer as a method for introducing the missing dystrophin into muscle cells is discussed at length and the limitations of such a strategy clearly identified. The third part is concerned with gene therapy which, at present, is still in its infancy but the possibilities in that field are clearly reviewed.

Once again, following each section, there is a general discussion which provides much of interest to the research worker in the field irrespective of whether he is involved in clinical or basic research. The papers themselves provide a very balanced overview of the situation in respect to the rapidly advancing field of molecular biology up to the time of the meeting.

Roses introduces a measure of clinical realism when he says that, "The scientific excitement and experimentation concerning

the biology of dystrophin . . . has obscured a focused, scientific commitment to the needs of the patient . . . The main aim point of our research is not the making of dystrophin, or even restoring normal muscular histology; is the successful treatment of skeletal and cardiac muscle affected boys hoping they will lead normal lives".

This sets a sense of urgency which is clear throughout the monograph.

This book can be recommended to every Neurologist and Scientist working in the field of Duchenne/Becker Muscular Dystrophy for their personal shelves and should be in the Library of all Departments who are charged with the care of these boys.

It is to be hoped that this series will continue.

WJK CUMMING

Stress, The Aging Brain, and the Mechanisms of Neuron Death. By ROBERT M SAPOLSKY (Pp 429; Price: £49.50). 1992. London, The MIT Press. ISBN 0-262-19320-5.

This book is a comprehensive account of the role of glucocorticoids in cellular aging and neuronal death. The author has made many important contributions in this area. Inevitably the book is angled from the author's own perspective. The book is well structured and the text written in entertaining and engaging fashion. Dr Sapolski uses his imagination to help the reader understand certain concepts; for instance I found his parallel between neurons destined for programmed cell death and the trappings of a construction site such as scaffolding and porto-toilets and the work sheds interesting, if not slightly surprising given the oligophrenia that usually characterises such sites.

There are several useful "summaries of the book so far". This allows the reader to enjoy the "story" even though he may have missed previous episodes. The chapter on programmed cell death (apoptosis) was particularly well written and relatively up to date. The role of glucocorticoids and stress particularly with relevance to lymphocyte function and immune competence was particularly interesting.

The cause of aging and the biochemical changes that accompany it are the subject of increasing attention in the neurosciences. This is particularly so because of the relevance of these processes to neurodegeneration and selective neuronal death in the CNS. Does the aging process play any part in accelerating this phenomenon and if so, can these diseases (and presumably the aging process itself) be retarded? The suggestion that excessive exposure to glucocorticoids over lifetime can accelerate brain aging may be relevant in this respect. A feature of this theory is the self-regenerating cycle of hippocampal degeneration caused by glucocorticoid excess and glucocorticoid hypersecretion caused by hippocampal degeneration. It is proposed that degeneration of the hippocampal neurons is consequent upon the action of glucocorticoids in impairing the capacity of these neurons to "survive coincident neurological insults". These include hypoxia and ischaemic reperfusion damage, seizure activity and oxidative stress. It will come as no surprise to the reader that these activities are associated with the accumulation of excitatory amino acids.

Overall I believe this book will find interested readers amongst the steroid community and perhaps a small section of the neuroscience community but I doubt whether the bulk of neuroscientists involved in neurodegenerative research will find anything but certain sections of this book of value.

AHV SCHAPIRA

Epidemiology and Control of Neural Tube Defects. By JM ELWOOD, J LITTLE and JH ELWOOD (Pp 926; Price: £85.00). 1992. Oxford University Press. ISBN 0-19-261884-9.

This volume sets out to be a new edition of "Epidemiology of Anencephalus and Spina Bifida" by Elwood and Elwood (1980), but events overtook it. In addition to updating the original ground, including substantial advances in pre-natal diagnosis, it became necessary to review developments in the field of primary prevention, and the ethical and legal issues associated with neural tube defects (NTD).

Most of the book (more than twice as large as its predecessor) discusses the classical approaches to the epidemiology of NTD, each chapter reviewing the relevant literature *in extenso* and ending with a summary and comment. The generous use of Figures and Tables helps the reader to grasp the tremendous amount of information presented.

In the field of primary prevention, the results of the MRC vitamin supplementation trial were published as the book approached completion. The authors have, however, included a comprehensive account of its confirmation of the protective effect of high dose folic acid against recurrence of NTD. They could not wait for the outcome of the Hungarian study showing prevention of first occurrence of NTD by low dose folic acid & multivitamins (Czeizel and Dudas, *N Eng J Med* 1992;327:1832-5).

The ethical and legal issues relating to prenatal diagnosis, pregnancy termination, clinical trials and post-natal treatment (or non-treatment) of babies with spina bifida are thoroughly and thoughtfully reviewed by Professor Alastair Campbell. I wish Chapter 15 had been entitled "Nutrition" rather than "Diet", for there is more to the vitamin story than what people eat. Otherwise there is little to criticise. The book is beautifully produced and is a superb work of reference.

DICK SMITHELLS

Behavioral Endocrinology. Edited by J B BECKER, M BREEDLOVE and D CREWS (Pp 574; Price: £31.50 (P/bk), £58.50 (cloth)). 1992. London, The MIT Press. ISBN 0-262-52171-7 (P/bk).

Behavioural endocrinology is written as an undergraduate text book which reviews in a clear and interesting manner current knowledge of the effect of hormones on behaviour and of behaviour on the release of hormones. The main areas covered are sexual behaviour, parental behaviour, aggressive behaviour, the stress response, ingestive behaviours and biological rhythms. The main species covered are rodents but a wide number of other species are covered from invertebrates, to which a whole chapter is

devoted, to frogs, snakes, lizards, birds and hyenas. The comparative approach makes for enjoyable reading and indicates how the influence of hormones on behaviour is inversely related to the development of the cerebral cortex. So what then does the book have to say about human behavioural endocrinology?

In the area of clinical psychoendocrinology some parts are disappointing. It is true that there are few findings that would merit detailed discussion but the endocrinology of depression and the behavioural consequences of Cushing's Syndrome would have merited a chapter in their own right but yet are hardly mentioned. There are also areas which provide useful "bridges" between basic and clinical behavioural endocrinology. Thus the development of learned helplessness—an animal model for depression—is dependent upon the activation of central glucocorticoid receptors. If undergraduates reading such a textbook do not know of the existence of such bridges they will never cross them.

For someone wanting an introduction to the effects of hormones on behaviour in the animal kingdom this is an excellent buy, and a very enjoyable read. It is not an ideal text for trainee psychiatrists or neurologists: but then it was not written for them.

S CHECKLEY

Handbook of Cerebellar Diseases (Neurological Disease and Therapy Series 16). Edited by RICHARD LECHTENBERG. (Pp 592 Illustrated; Price: \$185.00). 1993. New York, Marcel Dekker, Inc. ISBN 0-8247-8776-5.

This multi-authored volume provides extensive coverage of cerebellar disease under major sections such as structural, neoplastic, vascular, degenerative, infectious, demyelinating, and metabolic categories. There are many strong contributions including chapters on embryology, anatomy and imaging diagnosis. Tumours, von Hippel-Lindau disease, radiation therapy and paraneoplastic disease are all covered well; for example I found it particularly helpful to see an account of the autoimmune aspects of paraneoplasia, and tabulation of the different cerebellar, antibodies. For the vascular specialist four chapters on cerebellar infarctions, haemorrhage, venous disease and transcranial Doppler seem the most impressive and reflect much of the recent interest in this area. The section on degenerative disease was clearly the most challenging to organise for it consists of assorted subject matter, often in brief contributions, including three chapters on Machado-Joseph disease, seven on Friedreich's ataxia and many contributions appropriately reflecting local experience. Nevertheless the range of authors and experience will be of particular interest to specialists. The chapter on alcohol, including considerable detail of animal studies of foetal toxicity, struck me as being appropriate justification for the US Surgeon General's warning to expectant women.

There are relatively few publications dedicated to cerebellar disease, and although this book does not include comprehensive coverage of the degenerations, it does contain some fascinating contributions and a feast for the hungry mind of the generalist. Specialists should also see it.

WRG GIBB