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