

Department of Neurophysiology
S RENOWDEN
Department of Neuroradiology, Radcliffe Infirmary,
Oxford, UK
A SELLAR
DNA Laboratory, Churchill Hospital, Oxford, UK

Correspondence to: Dr MG Hanna, Department of Clinical Neurology, Institute of Neurology, Queen Square, London WC1N 3BG, UK.

- Hammans SR, Sweeney MG, Hanna MG, Brockington M, Morgan-Hughes JA, Harding AE. The mitochondrial DNA transfer RNA^{Leu(UUR)} A->G⁽³²⁴³⁾ mutation: a clinical and genetic study. *Brain* 1995;118:721-34.
- Ciafaloni E, Ricci E, Shanske S, et al. MELAS: clinical features, biochemistry, and molecular genetics. *Ann Neurol* 1992;31:391-398.
- Moraes CT, Ciacci F, Silvestri G, et al. Atypical clinical presentations associated with the MELAS mutation at position 3243 of human mitochondrial DNA. *Neuromusc Disord* 1993;3:43-50.
- Morgan-Hughes JA. Mitochondrial diseases. In: Engel AG, Franzini-Armstrong C, eds. *Myology*. New York: McGraw Hill 1995: 1610-60.
- Goto Y, Nonaka I, Horai S. A mutation in the tRNA Leu(UUR) gene associated with the MELAS subgroup of mitochondrial encephalomyopathies. *Nature* 1990;348: 651-3.
- Hirano M, Ricci E, Koenigsberger MR, et al. MELAS: an original case and clinical criteria for diagnosis. *Neuromusc Disord* 1992;2: 125-35.
- Rosen I, Phillips S, Enzmann D. Magnetic resonance imaging in MELAS syndrome. *Neuroradiology* 1990;32:168-71.
- Jackson MJ, Schaefer JA, Johnson MA, et al. Presentation and clinical investigation of mitochondrial respiratory chain disease: A study of 51 patients. *Brain* 1995;118:339-57.

MATTERS ARISING

Brain and spinal cord MRI in motor neuron disease

We read with interest the article by Thorpe *et al*¹ raising the question of the importance of MRI alterations as prognostic factors in motor neuron disease. In a retrospective study at our institute, we also found signal abnormalities in MRI along the pyramidal tracts in 11 of 16 patients affected by amyotrophic lateral sclerosis.² In all of them, symmetric hyperintensity in T2 weighted images were recognisable at the level of the corona radiata, in the internal capsule and the cerebral peduncles; in two of these cases signal changes were also present in the pons. When the patients with and without MRI abnormalities were compared, we failed to find prognostic factors. Age, spinal or bulbar onset, disease severity as assessed by the Norris scale,³ time interval between onset of disease and MRI investigation, duration of disease (time interval between onset and death or study closure) did not differ between the two groups of patients. Conversely, MRI signal abnormalities were associated with more pronounced upper motor neuron signs. Thus in our experience, alterations in MRI do not have a prognostic value but they simply reflect the severity of pyramidal tract degeneration. As Thorpe *et al* suggested,¹ abnormalities of MRI may be helpful in early diagnosis of motor neuron disease. However, this has to be confirmed in further studies, as neuropathological examinations have shown great variability in

the expression of the involvement of the pyramidal tracts even in the typical cases.⁴

D TESTA
F CARELLA
Division of Neurology,
National Neurological Institute "C Besta",
Milan, Italy

Correspondence to: Dr D Testa, Divisione di Neurologia, Istituto Nazionale Neurologico "C Besta", V Celoria 11, 20133, Milano, Italy.

- Thorpe JW, Moseley IF, Hawkes CH, MacManus DG, McDonald WI, Miller DH. Brain and spinal cord MRI in motor neuron disease. *J Neurol Neurosurg Psychiatry* 1996; 6:314-7.
- Carella F, Grisoli M, Savoirdo M, Testa D. Magnetic resonance signal abnormalities along the pyramidal tracts in amyotrophic lateral sclerosis. *Ital J Neurol Sci* 1995;16: 511-5.
- Norris FH Jr, Calanchini PR, Fallat RJ, Panchari S, Jewett B. The administration of guanidine in amyotrophic lateral sclerosis. *Neurology* 1974;24:721-8.
- Bronwell B, Oppenheimer DR, Hughes JT. The central nervous system in motor neuron disease. *J Neurol Neurosurg Psychiatry* 1970; 33:338-57.

NOTICE

Announcement from the British Neuropsychiatry Association: 1997 summer meeting

The 1997 summer meeting of the BNPA will be held jointly with the American Neuropsychiatry Association on 20-22 July at Robinson College, Cambridge, UK. It will include half day sessions on frontosubcortical circuits and emotion/reward/violence, and the presentation of short scientific papers, posters, and single case videos by members. The winner of the 1997 BNPA Prize will be announced. Two prizes of £200 each will be given to the best paper/poster presentations by junior members. The AGM of the BNPA will be held on 21 July.

For further details of this meeting please contact Suzanne Miller, 44 Roan Street, London SE10 9JT. Telephone 0181 858 2699; fax 0181 853 4416; e-mail wight@compuserve.com.

For details of membership of the BNPA, which is open to psychiatrists, psychologists, neurologists, and those in related fields, please contact Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol BS16 1QT.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made

by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and your full name.

Pituitary Adenomas. Edited by A M LANDOLT, M L VANCE and P L REILLY. (Pp 656; £125.00.) Published by Churchill Livingstone, Edinburgh. 1996. ISBN 0443051348.

I very much enjoyed reviewing this book which has been edited by three neurosurgeons, from Europe, the United States and Australia. It is a very complete text which covers everything that you wanted to know about pituitary adenomas and would previously have had to search widely for contemporary answers. The editors have succeeded in commissioning a wide range of expert opinion covering all related specialist areas. The chapters have been logically arranged and there are many high quality illustrations including colour plates of histological slides. This is essential to achieve adequate clarity for interpretation by the non-expert reader. Inevitably there is some repetition, but in some ways this is no bad thing as the chapters can be read in isolation.

As a surgeon I was particularly interested in the chapters covering radiology, micro surgical anatomy, the history of pituitary surgery and current surgical techniques. The contemporary surgeon largely operates via the transnasal transphenoidal approach with minimal morbidity and high rates of cure. These remarkable achievements rely very much on sophisticated pre and per operative imaging techniques and the use of the operating microscope. Following an excellent and detailed account of the trans-sphenoidal approach there are further chapters on surgical results and prognosis for the various pituitary adenomas.

Following on from surgical considerations are excellent and very readable chapters covering the medical treatment of the different endocrinopathies associated with pituitary tumours. I particularly enjoyed the chapters on the different radiotherapeutic approaches to pituitary tumours. I have never been certain about which patients can safely be spared the potential long term morbidity of this treatment. Although optic neuropathy, brain necrosis and vascular injury are the most feared complications, hypopituitarism requiring hormonal replacement therapy eventually develops in a large proportion of patients after fractionated radiotherapy. It seems increasingly clear that despite full hormone replacement therapy many patients with iatrogenic hypopituitarism have a reduced quality of life.

The clinical features, diagnosis, and management of pituitary apoplexy are covered in a single chapter. This gives sound advice to the non-specialist neurosurgeon who may be called upon to deal with these cases when they present as emergencies. I would concur with the view that the beneficial effects on neurological, visual, and endocrine functions are sound reasons for early surgical intervention by the transphenoidal approach. The improved prognosis of pituitary apoplexy that has been achieved by contemporary management is amply illustrated by the fact that all 12 patients in the first published series of apoplexy were diagnosed at necropsy.

Overall I think that this is an excellent