

Water deprivation test

Time	U osmolality mmol/kg	S osmolality mmol/kg	U volume (ml)	Treatment
9.00 am	280	281	165	—
10.00 am	291	—	140	—
11.00 am	308	—	142	—
12.00 am	320	288	126	2 µg ADH
13.00 am	445	—	52	—

S = serum; U = urinary.

against herpes simplex virus. Antibodies against HIV and CSF cultures were negative. Three weeks later, she developed polydipsia and polyuria with a daily urinary output of up to 7 l. Anterior pituitary hormone function was normal. A water deprivation test by the method of Miller and colleagues¹ was performed and the table shows the results. Intravenous infusion of hypertonic saline (5% saline for two hours at a rate of 0.06 ml/kg/min) was performed. Plasma vasopressin concentrations were 3.0, 3.5, 3.3, 3.7, and 3.9 pg/ml at 0, 30, 60, 90, and 120 minutes respectively, in relation to a progressive increment in serum osmolality of 277, 289, 302, 308, and 315 mmol/kg. A diagnosis of partial central diabetes insipidus was made and she was given a course of subcutaneous 1-diamino-8-D-arginine vasopressin (DDAVP). Brain MRI showed the absence of the posterior pituitary "bright spot" in sagittal T1 weighted images. Six months later there was still considerable polyuria, which improved with nasal DDAVP treatment.

Hypothalamic-pituitary dysfunction after acute meningoencephalitis is very rare. A case of central diabetes insipidus was described as a complication of herpes simplex encephalitis in a patient with AIDS.² We report here the first case of herpes simplex encephalitis associated with diabetes insipidus in a previously healthy subject. Polyuria and polydipsia can also result from

nephrogenic diabetes insipidus with insensitivity to the antidiuretic actions of vasopressin, which can be caused by several drugs such as aminoglycosides. Although our patient had been treated with amikacin, the persistence of the clinical manifestations after drug withdrawal makes this an unlikely cause. Furthermore, both the water deprivation and hypertonic saline infusion tests clearly indicated a partial central diabetes insipidus. The absence of the posterior pituitary "bright spot" on T1 weighted MRI images, although reported in neurogenic diabetes insipidus, occurs in a large proportion of healthy people.

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- 1 Miller M, Dalakos T, Moses AM, *et al.* Recognition of partial defects in antidiuretic hormone secretion. *Ann Intern Med* 1970;73: 721-9.
- 2 Zuhayr T, Madhoun MD, Dwight B. Dubois, *et al.* Central diabetes insipidus: a complication of herpes simplex type 2 encephalitis in a patient with AIDS. *Am J Med* 1991;90: 658-9.

BOOK REVIEWS

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The Management of Pituitary Tumours. Edited by MICHAEL POWELL and STAFFORD L LIGHTMAN. (Pp 238; £49.50.) Published by Churchill Livingstone, Edinburgh 1996. ISBN 0-4430-5214-X.

This is a concise guide to the diagnosis and treatment of pituitary tumours aimed at neurosurgeons, endocrinologists, neurolo-

gists, and ophthalmologists. It aims to give the clinician an overview of pituitary tumour management and achieves this in a compact and well presented style.

The overall approach is that of the joint pituitary clinic at the National Hospital, Queen Square. Following an introduction, separate chapters cover the pathophysiology, medical management, visual manifestations, radiology, surgery, anaesthesia and radiotherapy of pituitary tumours. However, the book emphasises a joint specialist approach which is described in the second chapter.

Of the individual chapters, that on pathophysiology and pathogenesis comprehensively covers the theories of pathogenesis of pituitary adenomas, but does not clearly describe the classifications used in clinical practice. The chapter on medical management is comprehensive and includes protocol for pituitary function tests. The imaging chapter is well illustrated with clear reproductions of Magnetic Resonance and Computed Tomography Scans. The surgery of pituitary tumours has sections on surgical anatomy, classifications, pre-operative work-up, transsphenoidal hypophysectomy and transcranial surgery. It mentions the four main transsphenoidal approaches to the pituitary and then concentrates on the septal

endomucosal approach, which is clearly described. Post-operative management does not advocate neurological observations for all patients undergoing transsphenoidal surgery, which differs from the practice of other units. The penultimate chapter gives an excellent description of other parasellar lesions and the final chapter by the editors concentrates on controversial issues, including a balanced opinion of the treatment of craniopharyngiomas.

My only major criticism is that in a text of handbook style, summaries and key points would have been a useful addition for each chapter. Overall, I think that the book is a useful and readable guide to the management of pituitary tumours and I would recommend it to trainees and consultants, particularly in neurosurgery and endocrinology.

PETER HUTCHINSON

Pathogenesis and Therapy of Amyotrophic Lateral Sclerosis. (Advances in Neurology, Volume 68). Edited by GEORGES T SERRATRICE and THEODORE L MUNSAT. (Pp 312; £88.00.) Published by Lippincott-Raven Publishers, Philadelphia, PA 1995. ISBN 0-7817-0333-6.

Although Charcot described amyotrophic lateral sclerosis almost 130 years ago, we have been little the wiser as to its aetiology until very recently. This book, which accompanied the 1994 Marseilles neuromuscular conference, exudes the optimism which followed the discovery of superoxide dismutase mutations in familial ALS, and the results of the Riluzole study.

The book opens with 16 chapters addressing pathogenesis, with a stress on excitotoxic mechanisms, in particular the role of glutamate. SOD1 defects have only been detected in 20% of familial ALS cases, suggesting that glutamate may only play a part in a cascade of processes leading to cell death. The evidence for glutamate transporters, post synaptic receptor changes, and autoimmunity against calcium channels or gangliosides is discussed, together with the evidence from animal models implicating abnormalities of neurofilament genes, and neurotrophins.

The issues of causation having been set out, the niceties of differential diagnosis and natural history are discussed in relation to clinical trial design. In the final section, various therapeutic modalities are discussed, including neurotrophins, glutamate receptor antagonists, and mechanisms of inhibiting excitotoxins. Inevitably, many of the management approaches are based on animal models of diseases which do differ from ALS/MND, and which are reminiscent of the logic by which selegiline was considered a possible neuroprotective agent in idiopathic Parkinson's disease. Nevertheless, the biological principles which are being uncovered may have a role in neurodegenerative diseases other than ALS.

The many short chapters (34 in 277 pages) offer a "bite sized" approach to the subject matter, allowing an easy acquaintance with many of the issues. Whereas some of the chapters are extremely informative, the overall effect of the text is reminiscent of a bowl of Shreddies placed before a Shredded Wheat eater—arguably everything appears to be present, yet something is still missing.

JON SUSSMAN