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was fully alert. Serum and urinary osmolality became normal, urinary specific gravity was 1005-1025. She recovered from ovarian hyperstimulation syndrome and laparotomy during the next month.

Brain MRI and CT performed during the next five years were normal, as were repeated neurological and psychiatric examinations. The patient's IQ was 126-130.

Severe seoriasis with ascites and hydrothorax due to ovarian hyperstimulation syndrome and haemoperitoneum due to tubal pregnancy, with hypovolaemia, anaemia, and hyposomolar serum concentration masked an SIADH that was heralded by seizures, followed by a prolonged leu­kog­tic state. Collateral evidence of SIADH was obtained by normal creatinine clearance with urine hyperconcentration. The symp­toms of CNS water intoxication, as usual, appeared during a sudden decrease in Na+ serum concentration, and were treated slowly to avoid central pontine myelinolysis. During SIADH, CT showed several patchy areas of hypolucency, resembling severe lesions of acute hypoxic-ischaemic encephalo­pathy with brain oedema. Hypoxic-ischaemic encephalopathy is associated with several neu¬rological or psychiatric alterations, and CT and MRI did not show residual neurological or psychiatric alterations, and CT and MRI did not show residual brain oedema.1 5 Hypoxic-ischaemic lesions are, however, usually caused by residual neurological or psychiatric deficits, and CT shows evolution of lesions, with ventricular enlargement and leucoma­phy in this patient the hypolucencies disappeared, the patient had no neurological or psychiatric alterations, and later CT and MRI did not show residual areas of altered signal corresponding to early hypolucencies. Furthermore, unlike the situation in hypoxic-ischaemic lesions, the basal ganglia did not seem to be involved, and the ventricular system was not narrowed as in severe brain oedema. We concluded therefore that water intoxication induced CT images of patchy hypolucencies rather than the expected homogeneous hypolucency.1 5

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Thyrotoxic Hashimoto's encephalopa­thy

Thyrotoxic Hashimoto's encephalopathy is a distinct clinical disorder with high anti-thyroid antibody titers-reported in 2 cases. Prog Brain Res 1986;64:279–85. Thyroid hormone therapy in compensated Hashimoto's thyroiditis: a clinical expression of autoimmune cerebrum vasculitis. Brain 1986;6:80-4.

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Cerebral salt wasting syndrome

Excessive natriuresis, resulting in hypona­emia and polycythaemia, is an often recognised complication after subarachnoid haemor­rhage. Initially this was attributed to inappropriate antidiuretic hormone (ADH) secretion resulting in water retention, but
Letters to the Editor

blood in the suprasellar cisterns and the left

drome is accompanied by hypovolaemia.

examination was unremarkable. Brain CT

changes in urine production during surgery.

However, from day 12 onwards she again

and fluid loss were fully compensated by

plasma sodium range was between 128 and

sciousness and she gradually recovered from

development of a progressive polyuria of up to

21 200 ml per day (on day 22) and a 24

hour urine production rose to preoperative values.

vasospasm and therefore angiography was

left middle cerebral artery was disclosed,

which was successfully clipped on day 24.

Two days after surgery diuresis decreased

after the neurosurgical procedure was

urine production rose to preoperative values.

protein in CSF (and maybe other humoral

tant. Because an increase of atrial natriuretic

CSF through the open dura may have

lowering the intracranial pres­

cially in more severe cases.

A patient with cerebral autosomal dom­
inant arteriopathy with subcortical infarcts and leuconecephalopathy (Cadasil) confirmed by sur­

cerebral artery was admitted with severe headache and vomiting. Physical examination was unremarkable. Brain CT

showed a subarachnoid haemorrhage with blood in the suprasciatal cisterns and the left Sylvian fissure. Two days later she devel­

ed acute ischaemic deficits during surgery, and salt and fluid loss were fully compensated by 0-9% NaCl infusion. On day 9 she

was found unconscious with respiratory failure and bradycardia and CT disclosed a recur­

rent subarachnoid haemorrhage in the left striatum. The patient regained consci­

sciousness and she gradually recovered from a mild aphasia and right facial weakness.

However, from day 12 onwards she again

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