The Management of Postoperative Diabetes Insipidus

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The usual response to a major surgical procedure is temporary oliguria which persists for several days.1,3,12,17 The secretion of antidiuretic hormone (ADH) that produces this oliguria is, in a sense, an "inappropriate" secretion, in that it is not controlled by osmotic stimuli.

After surgical manipulation in the sellar and suprasellar areas, however, the supraoptic-hypophysial system may be temporarily or permanently damaged, so that a transient or permanent diabetes insipidus results. The coexistence of uncontrolled polyuria with postoperative sodium retention, or with possible excessive sodium loss in patients with prolonged hypopituitarism, may make the management of these patients very difficult. Two cases will be described to illustrate some of these problems.

In the charts illustrating these cases, "crude" water balance indicates only fluid intake and urine output. Insensible fluid loss, water of oxidation, and water in the food are not indicated. However, when this balance is considered along with changes in body weight, a good first approximation of the actual water balance can be made.

Corrected serum sodium concentration, or osmolality, indicates the ratio between body water and body electrolytes and tends to vary directly with changes of exchangeable electrolytes and inversely with changes of total body water.

Case Reports

Case 1. In November, 1955, a chromophobe adenoma of the pituitary was partially removed from a 30-year-old man with hypopituitarism. This procedure was followed by radiation therapy. The patient was placed on a maintenance dose of thyroid hormone and cortisone. In June, 1965, at the age of 40, he reported progressive loss of vision.

On September 30, 1965, under general anesthesia, a recurrent chromophobe adenoma of the pituitary was partially removed and the optic nerves and chiasm decompressed. The immediate postoperative course was uncomplicated except for marked polyuria and polydipsia. The maintenance dose of thyroid hormone and cortisone was continued. Initially, no specific treatment was given for the diabetes insipidus, and water balance was maintained by increasing the intake. A slight deficiency soon developed, however, as can be seen in Fig. 1. Treatment with pituitary "snuff" was started on October 6 and urinary volume decreased slightly. Intermittent injections of Pitressin® tannate in oil were begun on October 9 and resulted in definite decrease in urinary volume and slight weight gain.

The patient had been alert and ambulatory until the evening of October 11 when he suddenly became drowsy and incoherent. Serum sodium concentration that morning had been 132 mEq./L; at the time of this sudden drowsiness it was 114 mEq./L. Therapy during the night consisted of 2 liters of normal saline. On the following morning, 500 cc. of 3% sodium chloride were given with definite improvement of the patient's condition and an increase in his serum sodium concentration to 158 mEq./L. by noon of October 12. Lumbar puncture at that time revealed a slightly xanthochromic fluid under a pressure of 175 mm. of water. Also on October 12, there was definite negative water balance and subsequent weight loss. Serum sodium concentration rose to 145 mEq./L. on the morning of October 13. Despite these changes, the patient's alertness and orientation did not return to the prehypotensive state for several days. Beginning October 12, 9-alpha-fluoro-hydrocortisone was administered daily in addition to hydrocortisone. Intermittent injections of Pitressin® tannate in oil were given (Fig. 1). Radiation therapy was started on October 22.

Comment. In this case, symptomatic hypotonicity was precipitated by the postoperative treatment. The patient stayed in reasonably good water balance with a maximum daily fluid flux of 9.5 liters. Treatment was started with pituitary "snuff" followed by Pitressin® tannate in oil, mainly because of the inconvenience of the large fluid flux. Water retention and weight gain occurred, culminating in stupor and a serum sodium concentration of 114 mEq./L.

Two factors appear to have caused the
hyponatremia in this case, namely excessive postoperative sodium loss and positive water balance induced by administration of ADH. The 24-hour urinary sodium excretion at the time of the symptomatic hyponatremia was approximately 300 mEq. This patient had longstanding hypopituitarism. While the control of aldosterone secretion and sodium metabolism are partially independent of control by the pituitary, and while aldosterone output and sodium conservation are generally intact in association with pituitary lesions, the aldosterone may be decreased and the ability to conserve sodium may be impaired in patients with prolonged hypopituitarism.

Thus, a combination of negative sodium balance over several days followed by a shorter period of water retention (dilution) precipitated acute symptomatic hypotonicity (hyponatremia). Treatment with 0.9% saline was relatively ineffective, but administration of 3% saline promptly restored body fluid tonicity to normal levels and improved the patient's condition. The synthetic salt-retaining hormone, 9-alpha-fluoro-hydrocortisone, was then added to the clinical regimen.

Case 2. A 25-year-old man who had had a craniotomy in 1954 for a craniopharyngioma, reported an increase in the number and severity of his headaches, progressive visual loss, and convulsive seizures. Since his craniotomy he had had panhypopituitarism and had been receiving testosterone, cortisone and thyroid extract. Pneumoencephalography revealed a preoptic mass as well as a suprasellar mass that indented the 3rd ventricle.

On November 17, 1965, a right frontal craniotomy was performed and a craniopharyngioma was partially removed. Replacement therapy included hydrocortisone, 100 mg, prior to the operation and 50 mg. every 6 hours postoperatively. The patient did not regain full consciousness after operation.

Several hours after operation, diabetes insipidus was recognized and careful monitoring of