DISORDERS OF ELECTROLYTE AND WATER METABOLISM FOLLOWING BRAIN SURGERY

IRVING S. COOPER, M.D.
Department of Neurologic Surgery, New York University-Bellevue Medical Center, New York City

(Received for publication February 4, 1953)

Despite the acknowledged importance of maintaining water and electrolyte balance in surgical patients during the postoperative period, and the many contributions to the subject in the literature pertaining to general surgery, reports concerned with water and electrolyte metabolism in neurosurgical patients have been relatively few. It is the purpose of this report to discuss briefly specific syndromes of disturbance of water and electrolyte balance that are related not only to the physiologic reaction to a neurosurgical procedure, but also, and apparently more specifically, to lesions of the central nervous system that affect neurogenic factors in control of water and electrolyte metabolism. The more general principles of fluid balance have been adequately described elsewhere and will not be discussed in this report.

Modern physiologic studies of the role of the brain in water balance originated with the production of urinary diuresis by an experimental lesion of the 4th ventricle. The elucidation of the role of lesions of the supra-optico-hypophyseal system in the syndrome of diabetes insipidus established the physiologic relationship of the brain to control of water metabolism. Although neurogenic control of electrolyte metabolism is less well understood, certain facts have been established. One controlling factor in electrolyte balance is the thirst mechanism, which is apparently governed by a physiologic apparatus within the hypothalamus and posterior half of the frontal lobes. Moreover, Verney has demonstrated the presence of an osmo-receptor in the forebrain which regulates secretion of antidiuretic hormone by responding to the osmolarity of circulating electrolytes. Electrolyte abnormalities secondary to experimental hypothalamic lesions in animals have been recorded by several independent investigators.

Several clinical reports have appeared that seem to indicate that electrolyte disturbances may, in certain instances, be secondary to brain lesions. A syndrome of extreme retention of sodium and chloride combined with decreased urinary excretion of these ions associated with cerebral lesions has been independently reported by several investigators. All reported cases of this syndrome of neurogenic hypernatremia have recently been reviewed by the author. A syndrome that seems to be the exact opposite of neurogenic hypernatremia, that is, excessive salt loss associated with cerebral disease, has been described by Peters et al and confirmed by
The recent reports of these two syndromes have re-awakened interest in the role of the central nervous system in the control of electrolyte metabolism. Welt et al.\textsuperscript{32} have recently stated that “an awareness of the potentialities for disorders in metabolism and excretion of electrolytes and water in association with a variety of primary intracranial abnormalities will increase the frequency of their recognition and provide insights into the mechanisms involved. This must ultimately promote better care for the patient.” Since one might justifiably add parenthetically—particularly for the neurosurgical patient—it is the purpose of this paper to review briefly those electrolyte abnormalities most commonly encountered following brain surgery.

**NEUROGENIC HYPERNATREMIA**

**ILLUSTRATIVE CASE REPORTS**

*Case 1.* A 58-year-old housewife, who had had a ventriculocisternal shunt (Torkildsen operation) performed elsewhere 3 years previously, was admitted because of confusion, headaches and blindness.

**Examination.** Positive neurological findings were complete amaurosis and optic atrophy with superimposed papilledema. Ventriculography demonstrated massive dilatation of both lateral ventricles and the anterior portion of the 3rd ventricle with a round filling defect in the posterior half of the 3rd ventricle.

**1st Operation.** A suboccipital craniotomy and second ventriculocisternostomy were performed.

**Course.** The Torkildsen tube functioned only temporarily, and signs of increased intracranial pressure recurred within 2 months.

**Metabolic Studies.** Throughout this period, from the time of admission, the levels of serum sodium and potassium, plasma chlorides, and carbon-dioxide combining power of the blood were obtained 3 times weekly. All findings were within normal limits.

**2nd Operation.** Eight weeks after the second ventriculocisternostomy, a right frontal craniotomy was performed, and a transventricular removal of a large brownish, vascular meningioma was accomplished.

**Postoperative Course.** The patient remained stuporous and became increasingly comatose. The somatic musculature was flaccid. There was marked hypotension (60/40) and hyperthermia (104–105\degree F.). She died in respiratory failure 1 week following surgery.

**Metabolic Studies.** After the 2nd operation the serum sodium, which had not risen above 141 mEq/l. in the preceding 8 weeks, rose to 164 mEq/l. by the 3rd postoperative day, and to 169 mEq/l. on the 6th postoperative day, while the plasma chlorides rose to 132 mEq/l. The fluid intake was maintained by gavage feedings at 2500 cc. per day. Urine excretion remained adequate and never fell below 800 cc. in 24 hours. The urine was dilute (sp. gr. 1.004) and acid in reaction during the week prior to death. Urinary excretion of chlorides determined 2 days before death was 80 mEq/l.

**Comment.** This patient with a known 3rd ventricle tumor was subjected to a control metabolic study for 8 weeks prior to removal of the tumor. All electrolyte studies during this period were within normal limits. Follow-