Clinical Implications of Adipsia

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In the usual case of diabetes insipidus, the polyuria is associated with a polydipsia. The failure of an individual to increase his intake under these conditions can be expected to result in a hypernatremia. However, in previously reported instances hypernatremia associated with lesions of the central nervous system has been imputed to a number of causes.

Allott reported several cases of hypernatremia associated with lesions of the central nervous system of varying types and location. Too little information was given to be certain about the status of the patients' fluid balance. Sweet et al. reported on a patient with gastrointestinal hemorrhage, hyperglycemia, azotemia and hypernatremia and hyperchloremia following prefrontal lobotomy. In their discussion, they postulated that the azotemia and hypernatremia might be ascribed to the cerebral lesion.

Peters presented the case of a young woman in whom hypernatremia developed following aspiration and removal of a cyst in the third ventricle. She exhibited marked polyuria immediately after operation; however, since adequate records of output of urine were not possible because of incontinence, the possibility exists that the patient was not in proper fluid balance.

MacCarty and Cooper reported a case of hypernatremia and hyperchloremia following ligation of both anterior cerebral arteries. Subsequently, Cooper reported 2 more cases with similar findings following operation in the region of the third ventricle. None of the patients had diabetes insipidus and it was postulated that the hyperosmolarity was caused by disturbance of a mechanism of the central nervous system regulating electrolyte metabolism.

The authors of the cases cited thus far were concerned primarily with the condition of hyperosmolarity and provided insufficient evidence to draw any conclusions regarding the possible role of disturbance of a primary thirst mechanism to account for their findings. However, reports of 3 other patients have been found in which the possibility for some form of disturbance of the thirst mechanism exists.

Engstrom and Liebman reported on a 28-year-old woman who had onset of polyuria and polydipsia 3 years prior to admission. These subsided after 5 months, except for occasional 2- to 3-day bouts of excessive thirst. Studies established the diagnosis of diabetes insipidus and some dysfunction of the anterior pituitary lobe. On numerous occasions when not on Pitressin therapy, the patient had serum-sodium levels of 150 to 165 mEq./l. Throughout her hospital course she maintained a relatively constant voluntary intake of fluid irrespective of her level of sodium or apparent state of hydration. Although the patient failed to vary her intake of fluid in a manner which would have been expected from her serum osmolarity, she never refused water or fluids.

Subsequently, Avioli et al. reported on a 12-year-old girl with Schüller-Christian's disease who apparently had diabetes insipidus 4 years prior to admission. This was controlled by vasopressin. After 3 years her need for this drug decreased and it was discontinued about 4 months prior to her admission without subsequent polyuria or polydipsia. Studies in the hospital revealed a partial diabetes insipidus. The patient maintained a hypernatremia of 155 to 165 mEq./l., but she never complained of thirst. On an unrestricted intake of fluid she did not have progressive dehydration and her body weight and serum sodium, although high, remained constant over long periods of time. One of the theories offered by the authors to ac-
count for their findings postulated a derangement of "centers" responsible for the perception of thirst in such a manner that thirst was only appreciated at levels of serum osmolality in excess of the normal range.

Persistent and excessive high levels of sodium and chloride serum in an 8-year-old girl were reported by Droese et al. This patient was retarded mentally and underdeveloped physically. Contrast studies showed a Cyclopean ventricular system. Extensive studies of renal and endocrinologic function were carried out. The authors concluded that there was a defect in the thirst mechanism secondary to maldevelopment of the hypophysial-midbrain region. They also postulated that the excessive hyperosmolarity subsequently damaged osmosensitive cells, making the lack of thirst even more marked. While it was noted that the sensation of thirst was always absent in their patient, there was no mention of active avoidance of water nor was there evidence that fluids had to be forced on the patient.

Although evidence of some form of disturbance of the thirst mechanism exists in the preceding cases, none (with the possible exception of the last patient cited) refused water nor had to be forced to drink. An adipsia or hypodipsia apparently did not exist. The present authors define adipsia as the failure of the alert patient to voluntarily ingest water. The subject of the case to be presented refused water and would not maintain an adequate intake of fluid except on a regimen in which fluids were urged upon him.

Case Report

#59-12684. E.A., an 11-year-old Negro boy, was admitted to the Pediatric Service on Sept. 21, 1959 with a chief complaint of "spells." The first had occurred 2 years previously and all had been of similar nature. The boy would become lethargic, complain of headaches and often vomit. This would be followed by a 2- to 3-minute period of hyperextension of the back and stiffening of the extremities, after which he would sleep for several hours. Between the attacks he was quite well.

There was no past history available as the child had not been living with his parents.

Examination. The initial findings were within normal limits with these exceptions: the boy appeared to be younger than his stated age and there was blurring of the nasal margins of the optic discs without definite elevation.

Laboratory studies revealed normal counts of blood, hemoglobin and urine. Roentgenograms of the right wrist and skull showed a bone age of 6 to 7 years, separation of the cranial sutures and enlargement of the sella turcica with flecks of calcium above it. An electroencephalogram revealed recurrent theta activity in the right occipital, right posterior temporal and right parietal areas. There was a left inferior quadratic defect of the visual fields.

A presumptive diagnosis was made of craniopharyngioma.

The specific gravities of the urine ranged from 1.009 to 1.018, the daily intake of oral fluids from 600 to 1600 ml., and the daily volume of urine from 450 to 1400 ml. The patient's level of protein-bound iodine was 6.9 µg per cent and his level of 17-hydroxycorticosteroid was 4.0 mg per 24 hours. The adult normal range in this laboratory is from 1.1 to 10.7 mg per 24 hours.

Course. The patient was placed on cortisone prior to operation on Oct. 6, 1959. Pneumoencephalography was attempted after placement of bilateral frontal burr holes. Spinal-fluid pressure in the upright position was 150 mm. above the level of the foramen magnum. No air passed into the ventricular system. A needle was introduced subsequently into the region of the suprasellar calcification via the right burr hole, and 10 ml. of yellow fluid, containing crystals, was obtained. The boy reported immediate improvement in his headache.

Operation. A right frontal craniotomy was performed and the optic chiasm was found to be tightly stretched over a posteriorly situated mass. Since it was felt that any attempt at removal of the tumor would necessitate cutting either one optic nerve or the chiasm, the operation was terminated so that the situation could be discussed fully with the boy's parents.

Course. The patient's condition was unchanged following the surgical exploration.

2nd Operation. This was carried out under hypothermia on Oct. 20, 1959. The optic chiasm was split longitudinally to expose the tumor. The mass stretched the chiasm downward and forward and extended caudally beneath the optic tracts. The pituitary stalk was divided during total removal of all visible tumor.

Microscopic diagnosis was craniopharyngioma.

Course. The patient was kept in a hypothermic state for 2 days. He responded adequately and the only changes on neurologic examination were bilateral absence of the pupillary light response, dysconjugate movement of the eyes and apparent absence of vision in the right eye. The patient was