Restricted fluid intake

Rational management of the neurosurgical patient

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Water balance studies in postcraniotomy patients indicate that restriction of fluid intake to 1 liter daily maintains the patient in homeostatic balance. A larger fluid intake will expand the extracellular space and presumably unfavorably influence cerebral edema. Daily observation of serum sodium and osmolarity and blood urea nitrogen, and preserving their normalcy, is a rational way of regulating fluid intake of the brain-injured patient. Fluid restriction should be used with caution if hyperosmolar agents, diuretics, or dexamethasone are also administered.

KEY WORDS ▪ water balance ▪ brain edema ▪ craniotomy ▪ head injuries

The amount of fluid recommended for administration to the postcraniotomy or brain-injured patient has varied from 1200 ml to 3000 ml daily, generally including 4.5 to 9 gm of sodium chloride.9,10,11 Only Fox, et al.,8 are specific about the basis for day-to-day variations in recommended fluid intake. They propose that the serum osmolarity level and serum sodium content be the criteria by which fluid administration is regulated. They arrived at this conclusion from the frequency with which the syndrome of inappropriate increase in antidiuretic hormone (ADH) secretion, or a tendency in this direction, was encountered in neurosurgical patients. We, too, have reported that patients with brain tumor who are maintained on 2 liters of fluid containing 9 gm/day of salt during the postoperative period retained water, and, in particular, had an expansion of the extracellular space.8

It is agreed that some cerebral edema follows brain injury, and its prevention or control by avoidance of overhydration, or even the use of dehydrating agents, is widely recommended. If expansion of the extracellular space does take place with the amounts of fluid presently recommended, brain edema will be adversely affected. It is therefore possible that the water intake requirements for maintaining homeostasis of the body water compartments in brain-injured patients are not as great as heretofore presumed. We have demonstrated that dexamethasone in pharmacological doses prevented this retention of water and expansion of the extracellular space in postcraniotomy patients by an increased urinary output.9 In other words, dexamethasone acted as a diuretic and a dehydrating agent.

Clinical Material and Methods

In a previous publication8 we have reported on the size of the body water compartments, serum osmolarity, and serum sodium con-
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 Patients among other parameters in a consecutive series of 10 patients (Series 1) with hemispheric brain tumors before and after surgery over a period of 6 days. These patients were maintained on an average daily intake of 1995 ml of fluid consisting of 0.45% saline in 2.5% glucose solution. Later we reported similarly on a comparable series of patients (Series 2) treated with 24 mg of dexamethasone daily with an average fluid intake of 1802 ml/day.

This report concerns a third series of 10 similar patients (Series 3), maintained on an average of 1055 ml of 0.45% salt in 2.5% glucose solution every 24 hours. Unfortunately, curtailment of laboratory facilities prevented measuring the body water compartments directly. However, daily observations of serum osmolarity and sodium, blood urea nitrogen (BUN), and weights of these patients, when compared with similar parameters of the previous series of patients whose body water compartments were measured, permit reasonable assumptions as to the effect of the reduced fluid intake on their body water distribution and economy.

Results

From the day of operation through the sixth postoperative day, the series of patients who were limited to an intake of 1 liter/day accumulated an average total of 1265 ml of fluid intake in excess of urinary output (Fig. 1). On the operative day, we were not able to control the intake as rigidly as during the postoperative period. Nevertheless, the highest fluid intake was 1850 ml and the lowest 700 ml with an average of 1237 ml for the day of surgery. From this point we

Fig. 1. Graphs showing fluid intake in relation to urine output (upper), serum osmolarity (center) and serum sodium (lower) in three series of patients. Broken line = Series 1, average daily intake 1995 ml; dotted line = Series 2, average daily intake 1802 ml, with dexamethasone; continuous line = present series, average daily intake 1055 ml. PO = postoperative days.

Fig. 2. Changes in average body weight in the three series, with 10 patients in each group. For key see Fig. 1.
attempted to regulate fluid intake to average 1 liter/day. The actual average daily intake was 1055 ml, with the second and third postoperative day averaging 893 and 841 ml respectively. In Series 1, maintained on 1995 ml, the average total excess of intake over urinary output in a similar period of time was 4498 ml, whereas the average total excess was only 429 ml in the dexamethasone group (Series 2), whose intake averaged 1802 ml daily. Serum osmolarity in patients on 1 liter/day throughout the week of observation remained within 1.5% of their average preoperative level of 290 mOsm/liter and this change was always in the direction of an increased concentration. This compares with an average decrease in osmolarity of 5% over the same period of time in Series 1, with patients averaging 1995 ml daily intake, and an average decrease of 3.6% in Series 2, in which patients had an average daily intake of 1802 ml and were treated with dexamethasone (Fig. 1). Serum sodium concentration likewise changed insignificantly in Series 3 patients, averaging 1055 ml daily intake. It increased an average of 0.8% (from an initial average concentration of 141.1 mg/l) through the sixth postoperative day. This is to be compared with a 2.2% average decrease (from an initial 143 mg) in Series 1 patients, whose fluid intake averaged 1995 ml daily, and 3.8% average decrease (from an initial 153 mg) in Series 2 patients averaging a daily intake of 1802 ml and treated with dexamethasone. It should be repeated here that we had previously demonstrated that dexamethasone increased sodium excretion, which accounts for the relative decrease in serum osmolarity and sodium content despite the fact that patients on this drug had the largest urinary outputs and the smallest excesses of water intake over output of all patients studied.

Changes in body weights in the three series of patients under consideration generally confirm the data given above (Fig. 2). The greatest weight loss was noted in the patients treated with dexamethasone ranging, on the average, up to 5% over the period of observation. This was to be expected since it was these patients who retained the least amount of fluid. Series 1 patients averaging 1995 ml of daily intake and not receiving dexamethasone lost 3% of weight over the 6 days of observation, but averaged no weight loss whatsoever (most patients gained weight) on the second postoperative day, the day of greatest water retention. Patients in the present series, limited to an average of 1055 ml daily intake, all consistently lost weight in amounts between the preceding two series, averaging 4% of their preoperative weights.

Changes in BUN (Fig. 3) and hematocrit (Fig. 4) in the three series of patients confirmed the inferences drawn from the above
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observation on the relative sizes of their intra-vascular spaces. These changes, although small on the average, revealed that the hematocrit was reduced least in the Series 2 patients given dexamethasone; the BUN tended to fall over the observed postoperative period in Series 1 patients given 1995 ml of fluid daily and tended to increase, even though minimally, in the other two groups.

Discussion

The data presented indicate that postoperative brain-tumor patients maintained on just over 1 liter of fluid daily are maintained in homeostatic balance with regard to their serum osmolarity and electrolytes. Inferentially, from reference to an analogous group of patients (Series 1, given almost 2 liters daily) whose body water compartments were measured,8 the extracellular spaces of patients restricted to 1 liter a day were prevented from expanding. Despite this relative restriction of fluid intake no evidence for over-dehydration occurred. This can be explained by the water retentive action of the antidiuretic hormone (ADH) known to be increased in the operative and postoperative state, and by the fact that craniotomy does not produce a "third space" of great consequence in terms of size.

The falling hematocrit in the postoperative period was observed in the three groups, and an explanation is not readily available. Moore5 has noted this trend in the general surgical patient and suggested it could be due to a delayed plasma volume refilling. Perhaps total body hematocrit determinations would offer the best insight as to the cause of this observation.

A further conclusion from Series 2 is that dexamethasone in pharmacological doses accomplished the same result, prevention of extracellular space expansion, by causing a diuresis that prevented accumulation of fluid in patients given an average of almost 1802 ml fluid intake per day.9 If this is one of the mechanisms by which dexamethasone prevents excessive cerebral edema then restriction of fluid intake in postcraniotomy patients could accomplish the same result without exposing patients to the side effects of corticosteroids. This principle of treatment can be applied to brain-injured patients generally. However, if multiple injuries are present, then corrections must be made for fluids lost into other injured tissues. This can best be judged by following the serum osmolarity and serum electrolytes on a daily, or even more frequent, basis. This should always be accompanied by strict attention to the level of the BUN. This approach was advocated by Fox, et al.,2 but their concentration on the syndrome of inappropriate ADH secretion in brain-injured patients tended to obscure this recommendation for the control of fluid intake to neurosurgical patients generally.

Several words of caution are necessary when recommending fluid intake restriction. If osmolar agents, diuretics, or pharmacological doses of dexamethasone are used, fluid restriction can result in severe dehydration and often its undesirable consequence will be abrupt and possibly irreversible renal failure. Hypertensive patients who have been treated with diuretics over a period of time may, to some extent, be salt-depleted; in this case their serum sodium content may be lowered and their extracellular space relatively contracted, and further water restriction would produce an undesirable degree of dehydration. Monitoring of the BUN in all such circumstances will help prevent such an error. In the patient with multiple injuries and large blood losses, an isometric concentration of the intravascular space can occur with a normal serum osmolarity and sodium and urea content. However, the weakness of the circulation would be obvious and no restriction of fluid intake would be recommended. By the same token, the multiple-injured patient could have a severe loss of isotonic fluid into a large "third space," and, if one relied only on the serum osmolarity and the serum sodium level, the contraction of the extracellular space would not be detected and fluid restriction would be harmful. In such cases, the probable weakness of the circulation and elevated BUN would indicate the need for increased, rather than restricted, fluid intake.

Despite postoperative fluid restriction, we have not infrequently noticed a lowering of the serum osmolarity and sodium content in the postoperative period. Examination of the situation revealed two unexpected causes. In some instances, a patient had an excessive amount of fluid administered during the operative period and this produced an iatrogenic simulation of the syndrome of in-
appropriate ADH secretion requiring several days of extreme fluid restriction (400/600 ml/day) to overcome. Nevertheless, on occasion the cerebral edema was irreversibly aggravated. Second, we have noticed that if a patient is administered humidified air, either by tube or mask, sufficient water will be absorbed through the lungs to expand the intravascular space, and by inference, the entire extracellular space, even though the patient is otherwise restricted to 1 liter/day of fluid intake. It has been determined that up to 500 ml/day of water can be absorbed in this manner by the lungs. Authors have discussed the quantity of sodium chloride that should be given with the daily fluid allotment to the postoperative or brain-injured patient. On the one hand, the withholding of salt is feared, since it might contribute to the postoperative hyponatremia noted by others, and documented by us when 2 liters of fluid are given daily. On the other hand, it has been feared that saline solutions given postoperatively might aggravate the results of the salt-retaining mechanisms usually brought into play in the posttraumatic or postoperative period and be a further cause of water retention. General surgeons, fearful of dehydration and electrolyte depletion produced by extrarenal losses as well as shifts into the considerable "third space" encountered in their patients, advocate administration of much larger quantities of electrolyte solution to their patients than are necessary for the neurosurgical patient. Most neurosurgical authors conclude that it is proper to replace the estimated daily normal salt loss, that is, 5 to 10 gm daily, and we have no reason to question this recommendation as long as serum electrolyte contents are followed closely. A serum sodium level below 135 mg/l is an ominous sign, and in the ordinary neurosurgical patient, generally reflects water retention, with an expanded extracellular space. Since the brain shares in this expansion, a consequent deleterious effect on intracranial pressure could be expected. Occasionally, it can reflect a depleted body sodium with a reduced body water. This is especially seen in patients who have been on prolonged antihypertensive medication (principally diuretics), in old, debilitated patients, or in patients with adrenal or renal disease. Lately we have seen salt and water depletion in brain-injured patients transferred to our care who have been treated over several days with dexamethasone together with repeated doses of hyperosmolar solutions. Finally, it should be recognized that dexamethasone does not hold back sodium excretion, but is indeed a natriuretic and therefore its use should not inhibit the administration of salt solutions to the patient.

The clinical course of these patients was characterized by the lack of facial swelling as has been noted in the dexamethasone group. The small number of patients in each series precludes any further meaningful comment on their respective postoperative courses since the location, size, and type of pathological lesions were variable.

References


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J. Neurosurg. / Volume 45 / October, 1976