Trauma and Water Requirements

THE EARLY convalescent period after trauma or operation is complicated by an intolerance for sodium and water, and, in the past, the amount and character of fluids administered parenterally to patients has been determined rather arbitrarily. It is well recognized that water is essential for the maintenance of normal physiology; but, it must be present in proper volume, distribution, and concentration. Inexplicable disturbances in water metabolism, without consistent alterations in electrolyte metabolism, have been observed in many diseases; and there are many evidences for similar disturbances after trauma or operation that are best attributed to some poorly delineated alteration in water metabolism itself. These alterations usually last about three days.

Several instances of altered water metabolism related to trauma are worth mentioning. During the postoperative period, expansion of the interstitial space in patients with complicated clinical courses has been observed, even with degree of water retention leading to convulsions. In patients free of endocrinologic or renal tubular disease, large volumes of urine with low specific gravity are not excreted in the immediate posttraumatic or postoperative period. Instances of weight gain postoperatively can be shown to result from primary water retention with lowered serum osmolarity and serum sodium concentration.

The action of the anesthetic agent or agents must be considered in this alteration in the metabolism of water during operation. In animals a depression in renal function during anesthesia is characterized by decreases in urine flow, glomerular filtration, renal blood flow, and tubular reabsorption. The changes are directly related to the depth of anesthesia and are promptly reversed by discontinuation or lightening of the anesthesia. Investigations carried out in the same manner during operative procedures on man disclose the temporary nature of the change in renal function and attribute it principally to intramural vascular changes and afferent impulses arising from the operative site inhibiting water diuresis. Since there appears to be unanimity among investigators that the change in renal function which does occur under these circumstances is temporary, it would seem improbable to ascribe to it alterations in water metabolism enduring for several days.

The patient who is so ill that his water and electrolyte requirements must be met by parenteral administration is entirely at the mercy of his physician. Uninformed decisions determining the quality and quantity of fluids to be given during anesthesia and the postoperative period can jeopardize an otherwise successful treatment of disease. The remarkably wide limits of tolerance for ingested water by normal man on an unrestricted diet (fig. 1) have misled physicians to believe that

such is also true when homeostatic mechanisms have been altered by trauma. The illustration shows that on an average diet under normal circumstances the urine output of solute is between 600 and 800 milliosmoles per square meter for twenty-four hours. At this level of urinary output of solute, it is evident that the tolerance for administration or acceptance of water is in the wide range of about 800 ml. to 8 liters

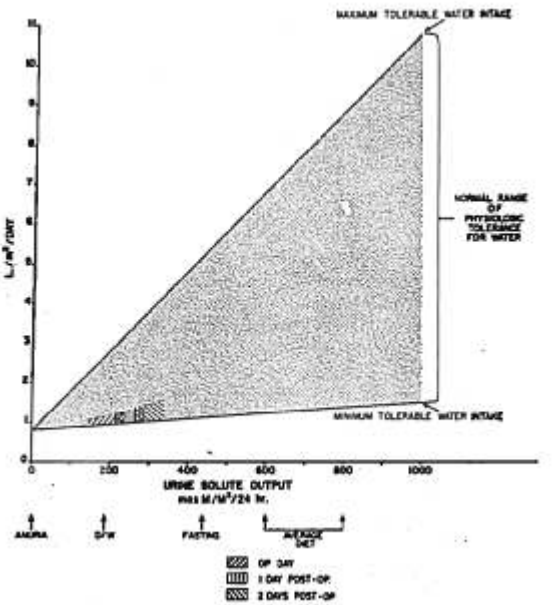

FIG. 1. The physiologic limits of water tolerance in normal individuals as determined by antidiuretic hormone activity (modified and reprinted with permission from Kerrigan *et al.*, J. Clin. Endocrinol. 15: 265, 1955). Superimposed on this basic diagram are the limits of water tolerance on the operative day and the first two postoperative days as limited by antidiuretic hormone activity and available solute in patients receiving glucose, 5 per cent in distilled water.

per square meter per twenty-four hours. When trauma and, with it, anesthesia are superimposed on the normal antidiuretic hormone-renal mechanism for the metabolism of water, quite another situation exists.

If patients are to receive only 5.0 per cent dextrose, in distilled water during operation and for the first two days postoperatively to maintain a fluid balance for insensible loss and renal requirements, it is essential to know, with confidence, within what limits hormonal (anti-

diuretic) reabsorption of water is occurring and what is the osmolar excretory load during that period.

It is known that the limits of *water concentration in the urine* are 0.7 ml. per milliosmole as a minimum and 10 ml. per milliosmole as a maximum. Coincident with trauma, an antidiuretic mechanism is active almost at the minimal limits of tolerance for urine water concentration. Prior to operation with free access to water the mean of urinary water concentration varies in normals from about two to four milliliters per milliosmole, the central area of physiologic tolerances as shown in figure 1. On the day of operation, however, in patients who receive more water than is required, urinary water concentration decreases and is 1.2 ± 0.5 ml. per milliosmole; on the first postoperative day it is 1.8 ± 0.3 ml. per milliosmole; and on the second postoperative day it is 1.6 ± 0.5 ml. per milliosmole. Knowing the urinary concentration of water for these important three days and the osmolar excretory load for each day, it is possible to superimpose figures relating to urinary requirements for water as effected by trauma on the physiologic tolerance limits when there is no disturbance of water metabolism, as in figure 1. Such calculations (using the mean value of each determination for each day) indicate the following amounts of parenteral glucose, 5.0 per cent, in distilled water to be given to provide adequate urinary function without excessive water retention: for the day of operation, 250 ml. per square meter per twenty-four hours; for the first postoperative day, 450 ml. per square meter per twenty-four hours; and for the second postoperative day, 480 ml. per square meter per twenty-four hours.

Though biological identification of antidiuretic hormone is lacking, the experimental results make it nearly mandatory to accept an antidiuretic mechanism as being active after trauma. It seems logical to accept the neurogenic release of antidiuretic hormone from the posterior pituitary as *the* mechanism involved in posttraumatic water retention. Since the water retention occurs in the presence of progressive and often alarming decrease in serum osmolarity (a mechanism normally inhibiting antidiuretic hormone release), it would be justifiable to assume that it is "centrally driven," that is, forcibly discharged.

Another source of miscalculation in determining basic water requirements for operative and postoperative patients is the evaluation of insensible loss. From very careful measurement of rates of weight loss under given metabolic states in a constant temperature-constant humidity room, it is evident that moderately covered normal and postoperative individuals display a relatively constant rate of insensible weight loss over the customary ranges of environmental temperature and humidity. The rate of insensible loss in afebrile individuals, if lightly covered, is 40.9 ± 5.1 Gm. per square meter per hour over the ranges of temperature 20 C. to 25 C. and humidity of 20 per cent to 60 per cent. This amounts to 984 ml. per square meter per twenty-four hours insen-

sible water loss. Since estimates give 197 ml. per square meter per twenty-four hours of water which is available from endogenous metabolism, the amount to be supplied is 787 ml. per square meter per twenty-four hours, an amount in close agreement with those obtained from previously published metabolic data by recalculation. A temperature correction for insensible water loss alone of 7.0 per cent for each degree that the rectal temperature is above 99.6 F. must be made. If this

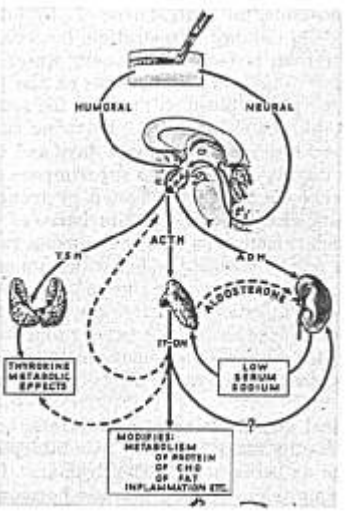

FIG. 2. A concept attempting to correlate the known and suggested information available concerning the neuroendocrine response to trauma. The interrupted lines indicate antagonism resulting from trauma. Neural influences from the site of trauma act via the neurohypophyseal system to release antidiuretic hormone. Antidiuretic hormone, producing primary water retention, and the 17-hydroxycorticoids, possibly producing increased urinary excretion of sodium, both tend toward lowered serum sodium levels. The low serum sodium acts through the adrenal cortex to release aldosterone, independent of the pituitary, effecting maximum renal conservation of sodium. (Reprinted with permission, Hayes *et al.* Surgery, 41: 353, 1957.)

amount is added to the values recommended for obligatory urinary function, the total requirements, excluding abnormal losses by drainages, total 1,000 ml. per square meter per twenty-four hours for the operative day, adding 250 ml. per square meter each of the two successive days. The calculated amount theoretically is to be administered over the entire twenty-four hour period; thus supplying water as it is metabolized, since there is no storage in the body for water. For the operative day, the theoretically "normal" man (1.73 sq. meters) would

receive 72 ml. per hour, the rate at which he is losing the water. If it is given faster, temporary water retention will occur with temporary falls in serum sodium and serum osmolarity.

Briefly (fig. 2), it is evident that trauma influences hypothalamic centers through the media of various neural effectors and possible humoral mediators. By disturbing the usual homeostatic mechanisms operative in the hypothalamus, its effect on the pituitary gland is altered; by neural corrections, the posterior pituitary (the neurohypophyseal system) discharges antidiuretic hormone; and by probable humoral influences, the anterior pituitary (the adenohypophyseal system) discharges at least thyrotrophic and adrenocorticotrophic hormones, each acting on its specific target gland.

Among the other metabolic effects of the increased release of adrenocortical steroids which are controlled by the anterior pituitary is a possible increase in renal excretion of sodium, tending to a lowered serum sodium concentration. On the other hand, the increased antidiuretic activity when water is supplied too rapidly, or in amounts beyond metabolic requirements, results in primary water retention and serum dilution producing a lowered serum concentration of this ion. The lowered serum sodium level, with an inadequate sodium intake, acts independently of any known controlling mechanism to increase adrenocortical release of aldosterone. This steroid effects maximal renal conservation of sodium.

Practical implications would caution against the administration of amounts of water exceeding metabolic requirements or exceeding rates of metabolic utilization. If the state of sodium metabolism is not known with absolute certainty prior to trauma, it probably would be unwise to give extra sodium during trauma or convalescence. If sodium restriction or depletion has not antedated the traumatic episode, normal daily requirements for sodium can be given throughout operation and convalescence without fear of excessive retention and edema.

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