Objective: To review the physiological changes in fluid and electrolytes that occur in aging.

Data Sources: Data collected for this review were identified from a MEDLINE database search of the English-language literature. The indexing terms were fluids, intravenous fluids, fluid resuscitation, fluid management, perioperative, electrolytes, aged, elderly, hemodynamics, hyponatremia, hypernatremia, hypocalcemia, hypercalcemia, hypomagnesemia, hypermagnesemia, hypophosphatemia, hypokalemia, and hyperkalemia. Relevant references from articles obtained by means of the above search terms were also used.

Study Selection: All pertinent studies were included. Only articles that were case presentations or did not specifically address the topic were excluded.

Data Synthesis: The fastest-growing segment of the population in the United States is individuals 65 years or older. It is imperative that health care professionals review the physiological changes that manifest during the aging process. Fluids and electrolytes are important perioperative factors that undergo age-related changes. These changes include impaired thirst perception; decreased glomerular filtration rate; alterations in hormone levels, including antidiuretic hormone, atrial natriuretic peptide, and aldosterone; decreased urinary concentrating ability; and limitations in excretion of water, sodium, potassium, and acid.

Conclusions: There are age-related alterations in the homeostatic mechanisms used to maintain electrolyte and water balance. Health care providers must familiarize themselves with these alterations to guide treatment of this growing population.

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As with other organ systems, there is a progressive decrease in the baseline function of the kidney after young adulthood. Longitudinal studies have shown significant variation in the rates of change in renal function among otherwise similar individuals, so it remains uncertain whether these common changes reflect subclinical disease or normal aging. In most individuals between the ages of 30 and 85 years, there is a 20% to 25% loss of renal mass, most of which is cortex. The aging kidney also exhibits hyalinization of blood vessel walls and a decrease in the number of glomeruli. This process progresses to hyalinizing arteriosclerosis and scattered arteriolar obliteration with a resultant loss of nephrons secondary to ischemia. A well-known comorbidity is that elevated blood pressure will advance the decline of renal function.

Functional changes parallel anatomic changes in the kidney. The kidneys exhibit an impaired concentrating capacity over time and a 10% decline in renal blood flow per decade after young adulthood. Functionally, the most studied change in the aged kidney is the decline in the GFR.

Rowe et al in 1976 first showed a sequential fall in standardized GFR in an aging population. Subsequent results obtained from the Baltimore Longitudinal Study of Aging demonstrated that a declining GFR is not inevitable. In a healthy cohort, examined regularly for 20 to 30 years, most individuals demonstrated a fall in GFR at an average rate of about 10 mL/min per decade. However, 30% of healthy aging individuals showed no decrease in GFR. Without more rigorous clinical testing for a more precise estimation of GFR (eg, 24-hour creatinine clearance), it would be difficult to select the 30% of aged patients who maintain a GFR comparable to that of their younger years; therefore, it is clinically sound to assume that the older patient has a reduced GFR.

Excluding individuals with inherent renal disease, it is generally accepted that there is a 50% to 63% decline in GFR from the ages of 30 to 80 years. Despite this decrease, the serum creatinine concentration remains within normal limits in the healthy aged. This paradox is due to the decrease in muscle mass (the principal determinant of creatinine production) that accompanies the aging process. The decrease in GFR parallels the decrease in muscle mass; hence, serum creatinine level remains constant with age. Notably, an increased serum creatinine in an elderly patient implies a much lower GFR than in a younger patient with a similar creatinine level (Figure). An elevated creatinine level in an elderly patient implies a GFR low enough that any small physiological stress could result in frank symptoms of uremia. Hence, the elderly surgical patient is at increased risk for virtually every cause of acute renal failure, an outcome associated with a mortality of greater than 50%.

The normal renal response to anesthesia and surgery does not appear to differ significantly between older and younger patients. Although GFR is directly depressed by inhalation anesthetics, the more deleterious effects on GFR and renal blood flow arise from a decrease in cardiac output. Hypothermia and/or intravascular loss will impact systemic blood pressure and cardiac output and result in a concomitant reduction in renal blood flow. Under elective surgical conditions, none of the above factors should result in clinically significant alteration of the GFR. Most patients, however, are relatively oliguric during surgery and in the immediate postoperative period. Urine tends to be concentrated, principally because of the stimulation of ADH as a direct consequence of the procedure and the anesthetic agents used.

Body fluid homeostasis can usually be maintained under normal circumstances. Problems occur when the older patient is stressed by illness confounded with fluid deprivation and/or iatrogenic insult. The most important principle in the elderly surgical patient is to maintain normal intravascular volume.

**FLUIDS**

Total body water decreases with age. In a younger man near his ideal body weight, total body water composes 60% to 65% of his body mass. By age 80 years, this contribution is reduced to 50%. Less arterial distensibility, decreased baroceptor reflexes, and sluggish homeostatic mechanisms result in increased susceptibility to hemodynamic changes in the elderly. After ingestion of water (via enteral or parenteral routes), the body fluid compartments are diluted.

With less than a 1% decrease in osmolality, the hypothalamus–posterior pituitary axis responds by inhibiting ADH release. In the absence of ADH, the kidney excretes a dilute urine, hence the efficacious excretion of the water load. Even with GFR rates as low as 30 to 50 mL/min, typical water loads can be excreted effectively. However, in the presence of inappropriately elevated ADH levels or significant extracellular volume depletion, a relative water excess can result in hyponatremia.

**Defect in Urinary Concentrating Ability**

The elderly are limited in their ability to tolerate water deprivation and, contrarily, to tolerate water boluses. A
decrease in maximal urinary concentration has been recognized for decades and is a partial explanation of nocturia in the elderly. Antidiuretic hormone levels are not suppressed in the elderly; therefore, failure of ADH secretion cannot account for the age-associated urinary concentrating defect. Rather, a failure of normal renal responsiveness to ADH appears to mediate this concentrating defect. It is not clear whether this failure results from a decreased medullary solute gradient or a decreased tubular response to ADH at the receptor level.

The hormonal regulation of fluid and electrolyte balance requires an intricate interaction between aldosterone, ADH, and ANP. Alterations in the levels of these hormones are partly responsible for changes in fluid balance associated with aging.

The peptide hormone, ADH or vasopressin, is produced by the supraoptic and paraventricular nuclei of the hypothalamus. It is well known that ADH is involved in the regulation of body water. To date, no study has documented age-related degeneration of the paraventricular or supraoptic nuclei. Furthermore, there are no age-related changes in ADH in the cerebrospinal fluid. In younger individuals, ADH release exhibits diurnal variation, with increased secretion at night. This diurnal variation is absent in the elderly. Plasma osmolality constitutes the major physiological stimulus for ADH secretion; however, changes in blood volume and blood pressure also stimulate its release.

Although debate exists, it is generally accepted that vasopressin levels are increased for any given plasma osmolality, when compared with values in younger individuals, with the notable exception of elderly patients with Alzheimer dementia, who exhibit decreased ADH secretion. Osmoreceptor hypersensitivity is the proposed mechanism of exaggerated ADH response. There may also be age-related changes in volume- and pressure-mediated ADH release. These alterations can make the elderly more susceptible to hyponatremia by interfering with the body’s ability to purge itself of excess water.

Atrial natriuretic peptide is produced and secreted by the cardiac atria. The concentration of ANP is increased 5-fold over basal levels in the aged. In addition, the elderly exhibit an exaggerated increase in ANP in response to sodium chloride infusions. The natriuretic response to exogenous ANP is exaggerated as well, as is its ability to suppress aldosterone. Increased ANP levels presumably cause direct suppression of renin, with a secondary decrease in angiotensin II and in aldosterone culminating in the renal loss of sodium associated with aging. This may help protect the elderly from volume expansion. Interestingly, with superimposed hypovolemia, ANP is not completely inhibited.

The secretion of aldosterone is also altered in the elderly. Adaptation to salt and extracellular fluid volume depletion is narrowed in older individuals. Epstein and Hollenberg showed that, given a low-sodium diet, the older subject required 2 to 3 times as long to bring sodium excretion into balance. A proposed mechanism for the delay is that renin secretion is blunted in the elderly. Weidmann et al found that the elderly exhibit a blunted renin response to a low-sodium diet. This results in a decreased angiotensin II and aldosterone response with subsequent sodium loss. The clinical consequence of these changes is “salt wasting” and is exemplified by situations that result in a paradoxical exaggeration of clinical hypovolemia despite the body’s need for maximal sodium conservation, such as in gastrointestinal losses.

It is also notable that the direct aldosterone response to hyperkalemia is diminished in elderly patients, and the tubular responsiveness to aldosterone appears to be less vigorous than in younger individuals.

Defect in Thirst

The elderly have an intrinsic defect in the thirst mechanism resulting in decreased fluid intake despite increases in osmolality and serum sodium level. A landmark study by Phillips et al illustrated that, in the setting of water deprivation, older individuals consistently described less thirst, despite their greater level of dehydration (as evidenced by decreased body mass and increased plasma osmolality). In addition, once granted free access to water, plasma osmolarities in younger subjects normalized within the first hour, whereas plasma osmolarities in older subjects failed to normalize after several hours. The decrease in thirst has been attributed in part to a defect in an opioid-mediated thirst center in the central nervous system.

Further evidence of a decreased thirst mechanism was provided by Zappe et al, who concluded that the inability of the elderly to increase their plasma volume after repeated days of exercise is not primarily related to an impaired renal fluid and sodium conservation ability, despite a reduced urine-concentrating ability during exercise, but to other factors (eg, fluid intake and total circulating protein) that appear necessary for a hypertensive response.

Free-Water Response

Finally, the ability to excrete a water load is impaired in older individuals. Free-water clearance during maximal water diuresis is significantly impaired in older patients, especially in the presence of thiazide diuretics. Although hyponatremia in the older patient is often multifactorial, the sluggish free-water response demonstrated in healthy elderly persons is exacerbated in aged hospitalized patients.

ELECTROLYTES

Sodium

Sodium is the principal cation of the extracellular fluid; the total sodium content determines the size of the extracellular volume. The age-related alterations in hormonal control of salt and water homeostasis described earlier predispose the aged to hyponatremia and hypernatremia.

Younger patients have no difficulty excreting sodium boluses. The ability of the elderly patient to deal with these extremes has not been well studied or defined. Since a prerequisite mechanism for acute salt excretion is a rapid increase in GFR, it is intuitive that the
elderly patient (with an intrinsically lower GFR) would have a limited capacity for sodium loads and would be more prone to overexpansion of the extracellular fluid compartment.

Under conditions of sodium deprivation (or of extrarenal salt losses), it is clear that the renal response is sluggish in the elderly population. There is a delay in the ability of the aging kidney to lower sodium excretion to minimal values. Hence, the elderly patient is more likely to develop extracellular fluid volume depletion than is his or her younger counterpart under similar conditions.

The most common manifestation of sodium imbalance is neurologic and ranges from mild confusion to seizures and coma. These alterations in neurologic function are due to changes in cell volume, with hyponatremia and hypernatremia resulting in brain swelling and brain shrinkage, respectively. Particularly in the older patient, improvement in the neurologic dysfunction may lag days behind correction of the electrolyte abnormality.

Hyponatremia is a common disorder in the aged and more common than hypernatremia in the hospitalized elderly patient population. Studies indicate an age-related increase in plasma sodium levels by approximately 1 mEq/L per decade from a mean ± SD value of 141 ± 4 mEq/L in young subjects. It is estimated that 7% of elderly outpatients and 11.3% of geriatric inpatients are hyponatremic. Seventy-three percent of inpatient cases were iatrogenic in origin and attributed to intravenous fluid therapy and diuretic use. Sunderland and Mankikar concluded that a diagnosis of hyponatremia was associated with a 2-fold increase in mortality over age-matched control subjects.

Hyponatremia may result from hormonal imbalance as well. The syndrome of inappropriate ADH secretion is characterized by isovolemic hyponatremia and elevated ADH levels despite subnormal plasma osmolality levels. There are numerous conditions associated with the syndrome of inappropriate secretion of ADH, such as central nervous system disorders, infection, pharmacologic agents, and malignancies. However, there have been several case reports and a prospective cohort of this syndrome occurring in elderly patients without an identifiable cause. The findings of these reports suggest that the syndrome of inappropriate ADH secretion in elderly patients may be secondary to the age-related physiological changes in salt and water balance.

A variety of drugs taken by elderly persons can also induce hyponatremia; these include fluoxetine hydrochloride, amitriptyline hydrochloride, vincristine sulfate, vinblastine sulfate, and cyclophosphamide. Specifically, hyponatremia induced by chlorpropamide is most commonly seen in elderly patients.

A 1% incidence of hypernatremia (mean serum sodium concentration of 154 mEq/L) was reported in a retrospective analysis of 15187 hospitalized geriatric patients. Of note, 57% of these patients did not exhibit this electrolyte abnormality on admission. The etiologic factors provided were surgery as the No. 1 cause (21%) followed by febrile illness (20%). Hypernatremia is associated with depressed sensorium and increased mortality (7 times that of age-matched controls, although not correlated with the severity of hypernatremia).

The homeostatic limits of potassium excretion have not been defined in older individuals but are almost certainly narrowed with respect to those of younger individuals. As a consequence of these decreased homeostatic reserves, the older surgical patient is more likely to develop acute drug-induced hyperkalemia. Drugs that can cause hyperkalemia include potassium supplements, salt substitutes, potassium-sparing diuretics, nonsteroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors, β-blockers, heparin, digoxin overdose, and trimethoprim sulfate. The Boston Collaborative Drug Study demonstrated a progressive age-related increase in hyperkalemia in patients prescribed oral potassium supplementation. Dietary practices may play a role as well. A significant portion of the elderly population is placed on a sodium-restricted diet, which is inherently high in potassium.

Such patients may exhibit high-normal serum potassium concentrations on admission but remain asymptomatic overall. If surgery, trauma, or tissue breakdown occurs, or if other potassium-containing medications are administered, frank hyperkalemia may result, which would be compounded by any concomitant acute renal insufficiency. Hyperkalemia is particularly likely to occur in the elderly diabetic patient who usually has concomitant hypoadosteroneism. As stated previously, the response of aldosterone to an elevated serum potassium level is blunted in the elderly patient. As the prevalence of hypertension and edematous disorders increases with age, older patients are more likely to have been taking diuretics at the onset of surgical intervention. On the other hand, hypervigilance for hypokalemia is warranted in elderly patients. Potassium depletion predisposes to serious tachyarrhythmias, particularly in patients taking digitalis preparations, and in those undergoing a catechol-releasing stress such as surgery. Furthermore, gastrointestinal losses may result in large deficits in total-body potassium. Therefore, potassium deficits should be recognized and corrected before elective operations.

The ability for acid excretion has been studied in healthy elderly individuals and is dramatically impaired as compared with their younger counterparts. The elderly patient is quite susceptible to the development of acute metabolic acidosis. As the aging kidney becomes less effective at excreting a hydrogen ion load rapidly, the severity of the metabolic acidosis may be exaggerated, particularly if concomitant pulmonary disease limits compensatory hyperventilation.

Calcium, Magnesium, and Phosphorus

The effect of aging on the relationship between the concentrations of blood ionized calcium and of serum parathyroid hormone (PTH) have been explored. Serum concentrations of PTH in elderly men were twice those of younger men, whereas blood ionized calcium did not dif-

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fer between the 2 groups. With intravenous infusion of calcium gluconate, the minimum PTH concentration was 2-fold to 3-fold higher in elderly men. These findings suggest that the relationship between calcium and PTH is altered with aging, such that at any given level of calcium, the concentration of PTH is higher. Optimal oral calcium intake of 1500 mg/d has been proposed for the elderly. However, high-calcium diets may reduce net zinc absorption and balance and ultimately increase zinc requirements.

The aging critically ill patient is also susceptible to alterations in magnesium and phosphorus homeostasis. Renal insufficiency predisposes to elevated concentrations of these electrolytes. Contrarily, poor dietary intake, use of various medications, and underlying disease can lead to deficiency states. Hypocalcemia cannot be normalized without correcting hypomagnesemia. Magnesium secondary to catecholamine shower, intestinal losses, various medications, and the unreliability of serum concentrations (even ionized) contribute to the true difficulty in defining magnesium deficiency. Levels of these ions should be vigilantly monitored in the critically ill geriatric patient.

CLINICAL APPLICATION

The most effective approach to fluid and electrolyte management in the elderly surgical patient is prevention. Knowledge of altered homeostatic mechanisms and side effects of polypharmacy in this population should guide clinicians' care of this population. The prevalence of clinically inapparent fluid and electrolyte disorders in hospitalized elderly patients warrants measurement of electrolytes, serum urea nitrogen, and creatinine and performance of a urinalysis in every patient.

Azotemia should prompt the clinician to identify the cause and correct or mitigate prerenal or postrenal factors before surgery. It cannot be overemphasized that the most important risk factor for perioperative acute tubular necrosis is hypovolemia.

Patients with chronic salt-wasting disorders, such as enterocutaneous fistulas or chronic diarrhea, may be electrolyte and volume depleted, with markedly impaired renal perfusion, and not exhibit classic signs of hypovolemia such as postural hypotension and tachycardia. Meticulous attention to volume replacement should be paid to all surgical patients, but particularly to trauma patients and to those subjected to extensive blood loss or fluid shifts common to biliary tract, cardiac, or aortic surgeries. In such patients, central hemodynamic monitoring should be used to guide fluid therapy and optimize renal perfusion. Despite these efforts, the treatment of these patients is challenging. Shoemaker et al concluded that vital signs, urine output, pH, and PO2 were not prognostic of patient survival in a population of high-risk surgical patients.

Studies using the APACHE (Acute Physiology and Chronic Health Evaluation) system have demonstrated that when severity of illness is controlled for, differences in survival between age groups disappear. In fact, physiological measures were the only predictors of short-term mortality.

Older patients may demonstrate deceptively normal vital signs despite a low cardiac output and mixed venous oxygen saturation. Scalea and colleagues demonstrated an improved survival rate of 53% in patients older than 65 years with diffuse blunt trauma with the use of early (<2.2 hours) hemodynamic monitoring and augmentation of cardiac output. Historical controls averaged 5.5 hours before the initiation of invasive monitoring and treatment, with a 7% survival rate.

Blood transfusions should be administered on an individual basis, depending on the patient's age, underlying cardiopulmonary disease, symptoms, life expectancy, and expected blood loss. Hemoglobin is an important component in the oxygen delivery equation. It is estimated that for every 1-g decrease in hemoglobin, cardiac output must increase 9% to maintain the same oxygen delivery. Older patients unable to augment their cardiac output and oxygen delivery in response to increased tissue demands may be at risk for myocardial damage and multisystem organ failure. A retrospective study of trauma victims older than 60 years demonstrated that survivors had higher hemoglobin levels (12.1 g/dL) and higher oxygen delivery than did nonsurvivors.

For the elderly patient who has undergone uncomplicated surgery, early resumption of oral intake is the best approach to maintain fluid and electrolyte balance. During the immediate postoperative period, ongoing fluid losses from all sites, including insensible losses, should be replaced meticulously. Adequacy of free-water replacement should be guided by the serum sodium concentration. Care to avoid nephrotoxic drugs is imperative.

CONCLUSIONS

The contribution of age in predicting survival is relatively small compared with the contribution of acute physiology or diagnosis, although it is well documented that mortality and complication rates from major surgical procedures increase with greater age. Although the general definition of a geriatric population begins at age 65 years, the physiological changes associated with aging are a gradual and individual process. To best serve these patients, the health care provider must understand the homeostatic changes that occur in elderly persons and avoid the pitfalls of ageism, where elderly patients' problems are inappropriately attributed to the "natural processes" of aging.

Normal physiological changes of aging increase the likelihood of fluid-electrolyte disorders in the elderly surgical patient. The aged kidney undergoes interstitial fibrosis as well as widespread sclerosis of glomeruli and of afferent arterioles. The most crucial functional changes are a decrease in GFR, decreased urinary concentrating ability, and narrowed limits for the excretion of water, sodium, potassium, and hydrogen ion. Despite these changes, body fluid homeostasis is effectively maintained under normal day-to-day circumstances. Problems may arise when the older patient is placed in a state of fluid deprivation or iatrogenic insult. Attention to age-related limitations of fluid homeostasis can help the physician prevent clinical complications such as hypotonicity and hyperkalemia, and volume depletion. Meticulous detail must be paid to salt and water balance and to drug dosing and...
choices. As the size of this population increases, randomized clinical trials regarding fluid management and invasive hemodynamic measures in the critically ill elderly patient may be warranted.

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