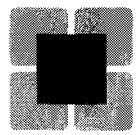

Aging and risks for dehydration

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■ The elderly are at increased risk for dehydration and associated fluid and electrolyte imbalances. Changes in functional and mental status, medication effects, and changes in the aging renal system all may be factors. Furthermore, hypodipsia, or insensibility to thirst, may be a physiologic process of aging. These and other risk factors are presented, along with a guide to the management of dehydration in the elderly.

□INDEX TERMS: DEHYDRATION; THIRST □CLEVE CLIN J MED 1990; 57:341-344

DEHYDRATION is the most common cause of fluid and electrolyte imbalance in elderly patients,[1] who are at risk because of physiologic as well as environmental factors. The risk of dehydration is significant in nursing home patients, where the prevalence may be as high as 35%.[2] And dehydration plays a significant role in the newly appreciated syndrome of failure to thrive in the elderly.[3]

A stable *milieu interior* depends on mechanisms that regulate water and electrolytes. The elderly are particularly prone to homeostatic imbalance because of physiologic changes in the renal system, reduced sensitivity to thirst, various disease states, and medication effects. The result is often dehydration with subsequent morbidity and mortality.

CLINICAL FEATURES

A useful classification for dehydration is based on sodium status,[4] or hypo- and hypernatremic dehydration (*Table 1*). These metabolic abnormalities should be included in the differential diagnosis whenever a geriatric patient presents with a change in mental or functional status—for example, a previously ambulatory patient who is now chair- or bedbound.

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Hyponatremic dehydration

Hyponatremic dehydration results from loss of both sodium and water, with the effect of sodium loss predominating. In the elderly, the primary cause is extracellular fluid depletion, often related to overuse of loop diuretics. Extracellular fluid depletion can also occur because of vomiting, diarrhea, osmotic diuresis of glucose, salt-wasting nephritis, third spacing (such as in cirrhosis), adrenal insufficiency, and decreased intake of both salt and water.[5]

The clinical manifestations are those usually associated with volume depletion and include orthostatic changes, dry mouth and mucous membranes, decreased skin turgor, weakness, anorexia, and confusion.

These signs and symptoms can be confusing because other entities can present similarly. For example, orthostasis (manifested primarily by orthostatic hypotension) can be due to autonomic dysfunction, which is relatively common in the elderly; dry mouth and mucous membranes can be due to medication side effects, such as the anticholinergic effect of antidepressants, or by mouth breathing; and a decrease in skin tur-

TABLE 1
TYPES OF DEHYDRATION

	Hypernatremic dehydration	Hyponatremic dehydration
Causes	Rapid free water loss Fluid deprivation Insensibility to thirst Infection	Extracellular fluid depletion
Predominant symptoms	Change in mental status Diminished thirst Weakness	Postural changes Weakness
Treatment	Hypotonic saline	Isotonic saline

TABLE 2
RISK FACTORS FOR DEHYDRATION IN THE ELDERLY

Unable to eat or drink without assistance
Enteral support without added free water
Depression
Loss of interest in self-care
Loss of memory, communication skills
Apraxia
Multiple chronic diseases
Acute infection
Polyuria related to diabetes insipidus or poorly controlled diabetes mellitus
Vomiting or diarrhea
Use of medications such as diuretics, laxatives, and sedatives
Neglect
Insensibility to thirst
Physiologic renal changes

gor can be caused by elastic changes in the skin that accompany aging.

Laboratory findings include a decreased sodium level; an increase in the blood urea nitrogen to creatinine ratio (BUN/Cr) to greater than 10:1; a variable serum uric acid level; and, depending on the cause, variable urinary osmolality and sodium level. Once the cause has been determined, most cases of hyponatremia are treated by administration of isotonic saline.[6]

Hypernatremic dehydration

In hypernatremic dehydration, water loss is greater than sodium loss. The causes include rapid free water loss, the inability to obtain free water, and a diminished thirst response. The primary clinical presentation is diminished mental status. The "classical" signs, such as orthostatic hypotension, are not generally present.[2] Weakness is a common symptoms. Diminished thirst may or may not be present.

Many of these patients may have an occult infection at the time of hospitalization for an acute episode of dehydration. Mahowald and Himmelstein[7] noted a mortality rate of 48% in patients presenting with hypernatremic dehydration; they related the mortality not to the degree of hypernatremia but to acute bacterial infections unrecognized at the time of presentation.

The treatment of hypernatremic dehydration is usually free water or hypotonic saline.

RISK FACTORS

Table 2 lists the risk factors for dehydration in the elderly. It can and does occur simply because fluids are out of reach of wheelchair- or bedbound patients. Many patients in long-term care facilities require assistance in feeding and fluid intake and depend on an often-overworked staff to meet their needs. Frequently, these patients are not assisted. Some advocate that dehydration be included as an indicator of neglect.[8]

Patients receiving enteral nutrition are at risk. With many products, a hyperosmolar state can be induced unless water is given in conjunction. The associated diarrhea promotes water loss, but it can be prevented with iso-osmolar, lactose-free supplements.[9]

Altered mental status increases risk. The depressed patient may lose interest in self-care and fail to maintain adequate fluid intake. A patient with dementia may forget to drink or be unable to communicate thirst; those with apraxia may be unable to perform the movements necessary to drink.

The risk of dehydration increases with the number of chronic diseases that affect the individual.[2] Acute infections, particularly of the urinary tract or lungs,[8] increase risk, as do vomiting, diarrhea, and polyuria.

Medications may be implicated. Diuretics, particularly furosemide, lead the list. Sedative hypnotics, because they decrease mental status, and laxatives, which increase malabsorption, also may be causative factors.[2]

AGE-RELATED DECREASE IN THIRST

Water intake depends on environmental, psychological, behavioral, and physiologic mechanisms. The evidence to date suggests that the ability to detect thirst decreases with "normal" aging. It is likely that the responsible mechanisms involve a combination of various peptides and neurotransmitters.

The brain mechanisms that play a role in thirst were first recognized in the 1950s, leading Epstein[10] to develop the double depletion hypothesis: that both cellular dehydration and extracellular volume loss could separately arouse thirst. Since then, other mechanisms contributing to drinking behavior have been proposed, including oropharyngeal cues,[11] vagally mediated stimuli

with food ingestion,[12] anticipation of fluid intake,[13] subjective sensations,[14] cortical lesions,[15] and alterations in thirst with aging.[11]

Numerous chemicals modulate drinking.[16] Those that increase drinking include angiotensin II, beta adrenergic agonists, serotonin, histamine, neurotensin, neuropeptide Y, and prostaglandins. Substances that decrease drinking include alpha adrenergic agonists, antihistamines, and substance P.

Opioid peptides

Several studies[17–21]—particularly those involving the opioid antagonist naloxone—support the role of endogenous opioid peptides in the control of fluid ingestion. Possibly, these substances block the effects of angiotensin II, which stimulates drinking, and blunt the sense of satiety that normally results from drinking water.[22,23] The opioid-induced drive to ingest food and drink may decrease with age.[24,25]

Preliminary data from my laboratory have shown that, in both mice and humans, older subjects were less sensitive to the suppressive effects of naloxone on fluid intake. These findings do not appear to be secondary to age-related differences in tissue distribution or half-life of the various agonists and antagonists.[24] Rather, actual decreases in brain levels of neuropeptides, including endorphins, or endogenous opioid peptides,[26] and opioid receptors[27] have been reported.

Altered thirst threshold

Several studies have suggested that the thirst threshold is altered with aging. In the report of Miller and associates,[15] elderly patients who were admitted repeatedly for hypernatremic dehydration all lacked thirst and had a history of stroke. Miller concluded that cortical lesions caused the thirst deficit. Phillips and co-workers[11] found that, unlike younger individuals, elderly subjects who were deprived of water and then given free access to water did not drink enough to return their plasma osmolality to pre-deprivation levels; furthermore, the elderly subjects noted no significant difference in thirst before and after deprivation.

The younger group consumed more water during rehydration, even though older subjects had higher serum sodium and osmolality levels. The investigators concluded that health elderly men have thirst deficits.

AGE-RELATED RENAL CHANGES

Fluid homeostasis depends not only on an intact thirst, but also on the ability of the kidney to excrete or retain fluids. Numerous anatomic and physiologic changes in the renal system that occur with aging can affect fluid and electrolyte balance.[28]

Decreased renal mass

Anatomically, renal mass—primarily the cortex—decreases by about 20%. This leads to a decreased number of glomeruli. The number of sclerotic glomeruli increases with aging to 30% of the total. Vascular changes also occur and resemble those caused by hypertension, such as tapering and increased tortuousness of interlobular arteries, and increased shunting of blood away from the arteriolar-glomerular unit. Physiologically, renal blood flow decreases, primarily in the cortex, as does glomerular filtration rate.

Because these changes occur primarily in the cortex, medulla-to-cortex blood flow increases, and the medulla loses the capacity for maximal concentration of concentrate urine. Also diminished is the ability to conserve sodium under sodium-depleting conditions, such as a low-sodium diet, and decreased ability to excrete a salt load, as from a radiologic diagnostic test. Similarly, the aging kidney has less capacity to conserve water during limited fluid intake, which leads to hypernatremia, or to excrete excess water, which result in hyponatremia. Finally, under experimental conditions involving heat exposure and exercise,[29] plasma volume decreases more in the elderly than in younger subjects, suggesting a decreased capacity to maintain fluid within the vascular compartment.

Decreased plasma renin activity

Age-related changes also occur in the renin-angiotensin-aldosterone system,[30] such as decreased plasma renin activity and aldosterone secretion.[31] Plasma renin activity drops 70% after sodium restriction and 4 hours of ambulation; this may be secondary to either a decrease in renin storage or a decrease in the sympathetic nervous system's renin-releasing action.

Elderly subjects on an unrestricted or a low-salt diet had a mean urinary aldosterone excretion rate 30% lower than younger subjects.

A recent study by Yamamoto and colleagues[32] demonstrated impaired angiotensin II production in elderly hypernatremic patients; the authors theorize that this may in turn impair thirst and cause dehydration and suggest that the impaired angiotensin II production is the mechanism for impaired vasopressin release and renal concentrating capacity. Vasopressin release has been shown to decrease with aging in response to volume stimuli, but to increase in response to osmotic stimuli.[28] Alterations

in vasopressin regulation have been reported in Alzheimer's disease,[33,34] placing this group at increased risk for dehydration.

IMPLICATIONS FOR PRACTICE

Among the risk factors for dehydration, environmental causes are the most obvious and often easily rectified. Close observation of patients with diminished functional or mental status can prevent iatrogenic dehydration. Age-related physiologic changes play a significant role in dehydration. For example, the renal system is unable to adjust to changes in fluid or electrolyte balance as quickly as in the past, particularly during times of stress. Even a healthy elderly individual with free access to water risks dehydration, primarily because of hypodipsia, or insensibility to thirst.

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