Contents lists available at ScienceDirect

e-SPEN, the European e-Journal of Clinical Nutrition and Metabolism

journal homepage: http://www.elsevier.com/locate/clnu

Educational Paper

# A review of the literature on dehydration in the institutionalized elderly

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ARTICLE INFO

*Article history:*
Received 23 December 2006
Accepted 23 October 2009

*Keywords:*
Dehydration
Elderly
Hypernatremia
Hyponatremia
Confusion
Disorientation

SUMMARY

*Background & aims:* Dehydration is the most common fluid and electrolyte problem among the elderly. The purpose of this review is to summarize the literature on dehydration in the institutionalized elderly.
*Methods:* To find relevant literature for this narrative review, a computerized search of articles published until June 2009 was performed in three databases: PubMed, Medline, and the Cochrane Library.
*Results:* Dehydration is conceptualized and operationalized in many different ways in the literature. Yet, dehydration is reported to be widely prevalent and costly to individuals and to the health care system. It affects large numbers, contributes to or exacerbates other severe medical conditions, may cause acute confusion and disorientation, and severely impairs the elderly individual's quality of life. Various strategies to detect and address dehydration are reported in the literature and these are primarily based on practice, or small scale research projects.
*Conclusions:* Detection and prevention of dehydration is critically important among the frail, institutionalized elderly. In the future, the efficacy, effectiveness and economics of these strategies need to be further evaluated through research.


## 1. Introduction

Declining birthrates and increased life expectancy have substantially increased the proportion of elderly people in the population and the total number of older people around the world and these trends are expected to continue.[1] In Canada, about 8% of older adults live in long-term care (LTC) facilities.[2] Residents of such facilities generally require chronic care and often present complex clinical profiles including presence of co-morbidities, polypharmacy, functional and cognitive limitations, and nutritional problems.

Dehydration, a well known nutritional problem, can be defined as depletion in total body water content due to pathologic fluid losses, diminished fluid intake, or a combination of both[3] and poses particularly acute dangers for the institutionalized elderly. Hence, dehydration among institutionalized elderly is the focus of this narrative review.

Dehydration has been well examined among athletes, with the focus on optimizing their performance. In the literature, relatively little attention has been paid to dehydration among older adults, although they are particularly vulnerable to it due to age-related changes in total body water, impaired thirst perception, renal concentrating ability, vasopressin effectiveness and medication-related hypodypsia. Functional limitations, infrequent urination, and urinary incontinence common among the elderly may further increase their vulnerability to dehydration.[4,5] The effects of dehydration include confusion, disorientation, weak spells, infection, coronary artery disease, impaired or delayed wound healing, and death.[5–11]

Treating dehydration among the elderly imposes significant costs on the health-care system. Aggregate statistics on the economic burden of dehydration are not available for Canada; but according to U. S. statistics, $1.36 billion was spent in 1996 to treat hospitalized elderly patients with dehydration as their primary diagnosis.[13] The figure will continue to increase given the overall inflation as well as the growing proportion of the elderly. Yet dehydration is largely preventable and treatable. In the present article, we have reviewed the literature on dehydration among institutionalized older adults and provided recommendations for future research. Specifically, the review includes the following sections: "Definition," "Physiology," "Causes," "Prevalence, Consequences and Costs," "Detection, Prevention and Treatment," "Dehydration of the Terminally Ill," and "Conclusions and Recommendations."

## 2. Definition of dehydration

Dehydration is typically defined as depletion in total body water content due to pathologic fluid losses, diminished fluid intake, or

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1751-4991/$36.00 
doi:10.1016/j.eclnm.2009.10.007

a combination of both.[3] However, as Thomas et al.[14] noted in 2003, no absolute definition of dehydration exists. The term is often used as a generic one for any derangement in any fluid compartment.

Clinicians tend to equate dehydration with the depletion of *intravascular* fluid.[6,7] However, as Mentes et al.[78] have pointed out, it is important to understand that such depletion can take three forms. *Hypertonic dehydration* is depletion in total body water (TBW) owing to pathologic fluid losses, diminished water intake, or a combination of both.[2,8–11] This leads to hypernatremia in the extracellular fluid compartment, which then draws water from the intracellular fluids. Since the water loss is shared by all body fluid compartments and leads to comparatively little reduction in extracellular fluids, the individual's circulation is not compromised unless the loss is very great. This is also known as *intracellular* or *hypernatremic dehydration*. But clinicians also speak of *extracellular* or *hypotonic dehydration*, which is a fluid depletion in which more sodium than water is lost and extracellular fluid becomes depleted.[8,10–12] *Isotonic dehydration*, which is a balanced depletion of both water and sodium, also leads to a loss of extracellular fluid. This is also known as *isotonic fluid volume depletion*.[8,10,13] Thomas et al.[14] prefer to call a loss of both intracellular and intravascular water *hypovolemia* and restrict the term "dehydration" to hypertonic dehydration. According to their terminology, dehydration exists where the individual's calculated serum osmolarity exceeds 295 milliosmols. Intravascular volume depletion is marked by a BUN-creatinine ratio above 20 or a level of serum sodium above 145 mg per decalitre, and hypovolemia exists when the individual displays both a serum osmolarity above 295 milliosmols and a BUN-creatinine ratio above 20.[9]

Given the multiple ways of defining dehydration, it is important to understand the specific type of dehydration to identify and address the underlying causes in a timely and appropriate manner. In practice, dehydration has been defined as any severe decrease in total body water, whether it is pathological,[3] or results from failure to increase water intake to compensate adequately for losses, inability to ingest fluids in adequate amounts, or a combination of these factors.[15]

## 3. Physiology of dehydration

The human body is made up mainly of water (45–75%).[16,17] The largest reservoir of this water is the intracellular space, which accounts for about two thirds of the total, with the other third comprising intravascular and interstitial fluid. About a quarter of extracellular water, or 8% of total body water, is confined to the intravascular space. The chief regulator of extracellular water is sodium, which is readily transported into this space. The chief regulator of the intracellular compartment is the effective osmolarity of the extracellular fluid, which produces essentially equal tonicity across both compartments.[14]

Although hydration in the extracellular and intracellular compartments is separately regulated, the human body contains redundant backups that allow for some overlap in function. Extracellular hydration is mainly regulated by blood volume, and changes in blood volume are sensed by baroreceptors located in the aortic arch. When intravascular volume decreases, there is a sympathetic response that results in vasoconstriction in the subcutaneous bed, the gut, and the renal vasculature. In contrast, intracellular hydration is regulated primarily by changes in osmolality and is sensed centrally by osmoreceptors in the hypothalamus.[18] Homeostatic responses of thirst and alterations in salt appetite are associated with both types of dehydration.[19] Both these homeostatic mechanisms and the associated intake of fluids of varying tonicity are affected by psychological and environmental factors.[13] Factors such as exposure to heat, fever, drinking little fluid, and unusual physical activity increase osmolality and decrease plasma volume, which produces hyperosmotic hypovolemia. In normal individuals, this stimulates thirst and increases vasopressin levels.[20]

Normally, a healthy individual loses about 2500 ml of water a day, but the actual amount can vary greatly between individuals and depends on environmental conditions. When the air is dry or the weather is hot, water loss from the skin and lungs increases because of the increased vapour pressure gradient. The amount lost in urine depends greatly on the volume of fluid taken, the total losses by other routes, and also on the solute content of the diet. High intake of salt (sodium chloride) or protein will increase the daily fluid requirement, since the kidneys have a limited capacity to concentrate urine. If their water intake is restricted, individuals will conserve water by producing more concentrated urine. This concentrating ability varies between individuals, but in most people the maximum urine osmolality ranges between 900 and 1200 mosm/kg.[21] Conversely, since the body cannot store excess water, the kidneys get rid of any excess by producing a large volume of dilute urine. Most people take in more fluid each day than their perceived need, and their kidneys maintain water balance by excreting the excess.[22] In extremely hot weather, however, the body cannot lower its temperature by simply transferring heat to the atmosphere and compensates by producing sweat. This causes the body to lose both water and salts (electrolytes).[23]

Dehydration can also lead to decreased elimination of urea. Both creatinine and urea are eliminated when they are filtered by the renal glomerulus, move through the tubular system, and pass out of the body in the urine. Urea differs from creatinine in that about 40% of it is reabsorbed in the tubular system by passive back-diffusion in the proximal tubule, related to the sodium and water absorption occurring there.[24] Thus, when the reabsorption of salt and water is stimulated, urea reabsorption is enhanced and the ratio of BUN/Cr in the serum is increased.[25]

## 4. Causes of dehydration among the elderly

Between age 30 and age 90, human kidneys lose between a third[26] and a half[27] of their nephrons, which significantly impairs their ability to reabsorb solutes and conserve water.[28,29] By age 80, the glomerular filtration rate diminishes to about 300 ml/min and creatinine clearance decline by approximately 30%.[30] Since aging kidneys secrete less renin, the body releases less serum aldosterone, which in turn affects the reabsorption of sodium in the renal tubules.[30] The elderly also have increased plasma vasopressin, while their osmoreceptors become more sensitive to it;[5,31] but aging kidneys respond less well to vasopressin and therefore grow less able to concentrate urine.[30] This is particularly acute in victims of Alzheimer's disease.[32] At the same time, however, the response of aging kidneys to antidiuretic hormone also decreases,[15,30,33–35] while the rate of water excretion and the pattern of the constituent electrolytes also change.[36] Thus, aging kidneys become less able to dilute urine and excrete free water in it.[9] This places the elderly at risk for free-water overload, which causes hyponatremia. The hyponatremia in turn increases the risk of disorientation, stumbling, falling, and, ultimately, seizures, coma, and death.[31,37,38]

Diarrhea, fever, diuretics, cathartics, heat exhaustion, and simple overexposure to sunlight can all cause the body to lose abnormal amounts of fluid, while kidney failure, heart failure, poorly controlled diabetes, inappropriate secretion of antidiuretic hormone, diuretics, and angiotensin-converting enzyme inhibitors can all disrupt the body's ability to balance and manage fluid and electrolytes.[38] Normally, the whole system maintains a fairly constant volume of extracellular fluid despite wide variations in salt intake, as sodium output matches intake. However, in certain