

# Fluid and Electrolyte Disorders in Elderly

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Disorders of fluid and electrolytes are frequent cause of morbidity and mortality among the elderly. The true incidence of these disorders in Indian scenario is limited. Disorders related to fluid intake which are dehydration and hypovolemia are common in elderly. In one study, dehydration occurred in approximately 7% of hospitalized patients older than 65 years of age and was associated with significant morbidity.<sup>1</sup> The mean length of hospital stay among these with a primary diagnosis of dehydration was 14 days.

Derangements in sodium and potassium levels are frequent in clinical settings. Hyponatremia is found in approximately 1% of hospitalized patients over the age of 60 years and in a study comprising of febrile elderly hospitalized patients, the reported incidence of hypernatremia was high.<sup>2</sup> Among those with impaired oral intake, the mortality related to hypernatremia is significant.

Hyponatremia is also a common finding in the hospitalized elderly patients, especially with comorbid conditions like congestive heart failure, nephrotic syndrome, cirrhosis of liver etc. Disorders of other electrolytes like potassium are frequent on account of various physiological changes attributed to ageing and association with co prescribed medications eg diuretics.

## Physiological Considerations

In a normal individual residing in temperate climate, the daily fluid intake comprises of 1-1.5 litre in 24 hours. Water comprises 60% of the body weight in males (50% of body weight in females; 65-75% body weight in infants; 40-45% of total body weight in elderly). In a 60 kg male, total body water comprises 36 liters of which 65% (24 liters) is in the intracellular fluid and remaining 35% (12 liters) is present in extra cellular space. The water in intracellular space is also contributed by the cellular metabolism. The total intake of water is balanced by the inevitable insensible losses from the body which is approximately 500ml/

day; as well as the 180 litres of the blood which is daily filtered in the glomeruli out of which approximately 1.5 liters of urine is produced.<sup>5</sup>

This delicate balance of fluid and that of sodium is maintained by various physiological mechanisms of which the two most important are the (a) Thirst and (b) Antidiuretic hormone (ADH).

Normally, if a person is exercising on a hot day, then the increased insensible losses from the body and decreased fluid intake cause stimulation of thirst mechanism with increased craving for water intake. There is also increase in plasma tonicity with stimulation of osmoreceptors in the hypothalamus leading to release of ADH from posterior pituitary gland which helps in conservation of water with production of concentrated urine. On the other hand if there is liberal fluid intake, the thirst mechanism is suppressed and decreased ADH release causes loss of excess water as dilute urine. (Fig.1, 2, 3)

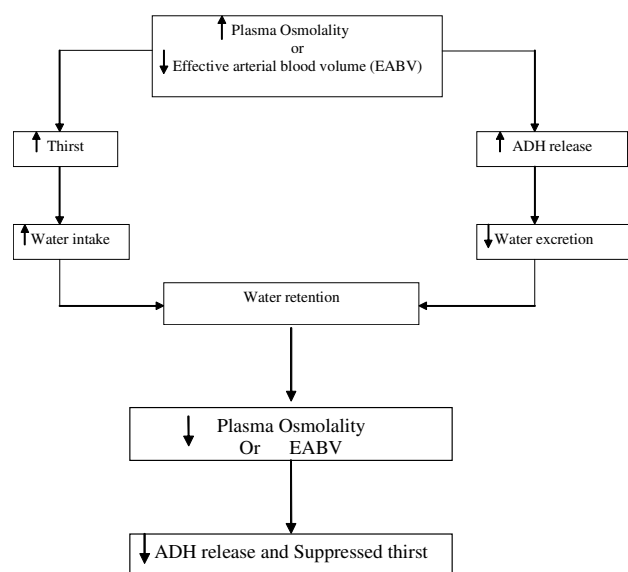


Fig. 1: Physiological responses to increase plasma osmolality.

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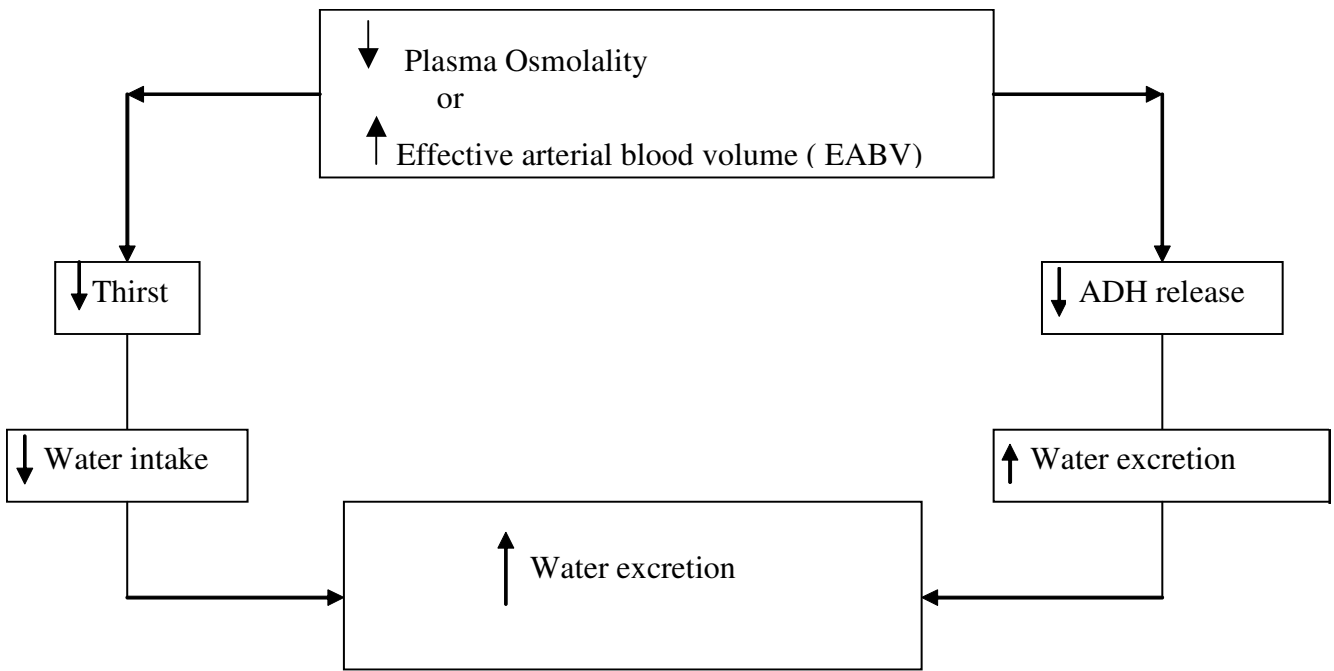


Fig. 2 Physiological responses to decreased plasma osmolality

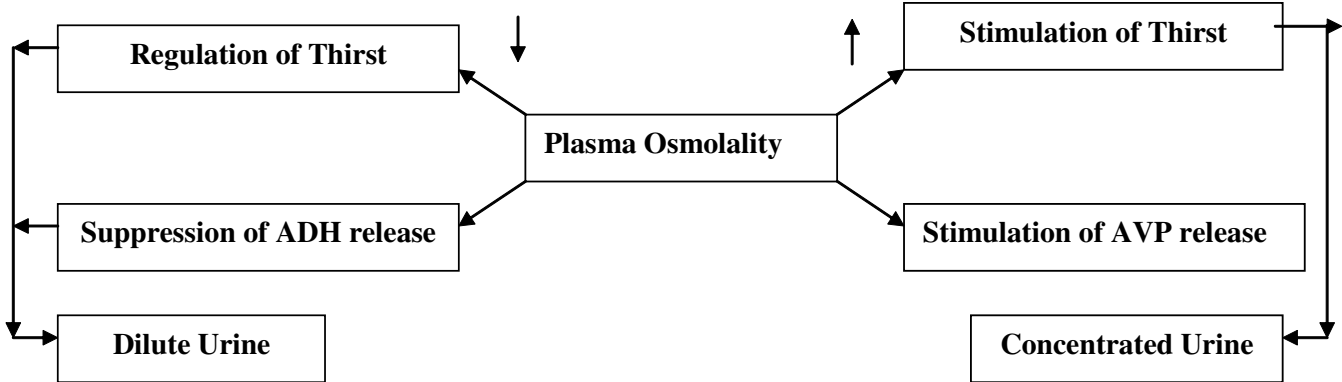


Fig. 3: Integrated relationship between plasma tonicity and related renal response

Hypernatremia reflects a relative deficit of water in relation to solute that is usually produced by lack of replacement of usual or increased water losses. Pure water loss is called as dehydration while hypovolemia is a condition in which extracellular fluid volume is reduced and tissue perfusion may be impaired. It is produced by salt and water loss (as with vomiting, diarrhoea, diuretics) or by water loss alone (i.e. dehydration). Salt and water loss comes primarily from the extracellular fluid where as pure water

loss or dehydration comes from total body water. Thus, dehydration as a cause to induce extracellular volume loss would require greater degree of fluid loss as compared to hypovolemia.<sup>2,6</sup>

Hypernatremia strongly stimulates the sensation of thirst. A free water deficit causing hypernatremia is rare in the patient who is physically and mentally capable of obtaining and drinking water. The associated increase in water lowers the plasma sodium concentration back to normal.

Disorders of volume are associated with abnormalities in sodium balance. Sodium loading tends to produce volume expansion and edema, (if the excess sodium is not excreted) while sodium loss with water can lead to volume depletion (hypovolemia).

**Age related changes**

There is a progressive loss of nephrons with ageing. The kidney tends to loose about 20% of its mass and the involutionary process is represented histologically by a decrease in the renal vasculature (especially in the renal cortex), increase in the number of obsolescent glomeruli, tubular atrophy with interstitial scarring. In spite of compensatory hyperfiltration and hyperfunctioning by the remaining nephronal units, the glomerular filtration rate declines with age beginning at about 35 yrs of age with a steady decline at the rate of 0.8 -1ml/min/year. However, the serum creatinine remains within normal range because of decreased muscle mass with age.<sup>7</sup> The alteration in thirst mechanism, renal function and /or hormonal functions also predispose to fluid and electrolyte abnormalities in the elderly.<sup>8</sup> (Figure 4)

In elderly individuals, the sensation of thirst which is subserved by hypovolemia and hyper osmolality is decreased. Elderly patients when deprived of water or if administered hypertonic saline tend to show diminished response to thirst mechanism to maintain the plasma sodium. This impairment is attributed partly to dysfunction in the drinking drive due to involvement of opiate receptors. Besides this confounding factors like various psychological factors, concurrent food ingestion, temperature of food and liquids and abnormalities in glossopharyngeal reflexes are present which may contribute to decreased fluid intake.

There is a decreased response of anti diuretic hormone in renal tubules with reduction in renal concentrating ability in the elderly. There is also

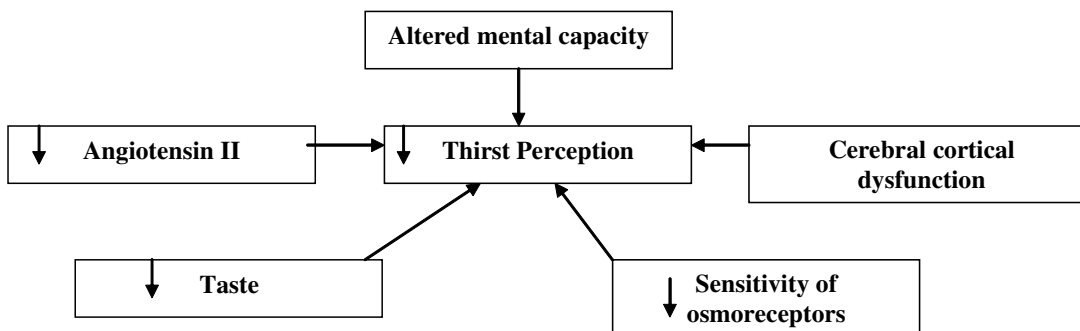
diminished function of the counter current system responsible for concentration of urine.

There is diminution in the serum concentration of renin and aldosterone with increase in atrial natriuretic peptide levels which predispose elderly individuals to greater sodium losses in the urine. Decreased glomerular filtration rate and concurrent comorbid conditions like congestive heart failure also lead to impaired sodium excretion in the elderly. Decreased ADH levels at night time in elderly also predisposes to nocturia.<sup>9</sup> Similarly elderly patients with Alzheimers disease and other neurologic disease have low ADH levels for a given plasma osmolality. Thus, decreased ADH with inability to request and obtain water in such conditions can lead to free water loss and hypernatremia.<sup>10</sup>

**Hypovolemia**

The predisposing factors for hypovolemia in elderly are increased insensible losses of fluids during infections in elderly with altered thirst response, presence of hyperglycemia, hypercalcemia and use of various drugs like diuretics, mannitol, and radiographic contrast material. These agents enhance further fluid loss with concomitant impaired ADH and renal concentration ability leading to fluid depletion. Frequent use of laxatives, bowel cleansing regimes for constipation and infectious diarrhoea also predispose to fluid losses in elderly.

Elderly individuals with limited access to fluids secondary to mobility restriction and poor vision cause inability to maintain proper fluid intake. This may be compounded further by use of physical restraints and aggressive fluids restrictions with associated co-morbid conditions. There is also increased incidence of urinary incontinence and swallowing difficulty which also



**Fig. 4: Factors contributing to impaired thirst perception in elderly.**

consciously reduces the fluid intake in elderly. Dementia and altered sensorium due to various causes like stroke can lead to a high risk of hypernatremia secondary to decreased fluid intake.

The above mentioned factors and lack of functional reserve in kidney predispose to severe fluid losses in the elderly. Of concern are obese elderly, who have greater proportion of fat relative to lean muscle mass since fat has less water reserve than muscles; hence elderly obese individuals are more susceptible to greater water loss.

### Clinical Features

Hypovolemia may lead to acute weight loss of > 3% of body weight. Orthostatic hypotension may be elicited on clinical examination and however it is imperative to consider its normal presence in an elderly due to primary autonomic dysfunction, medications etc. There is dryness of mouth and tongue, difficulty in speech, sunken eyes, confusion, flat JVP, decreased skin turgor and decreased axillary sweating. Oral breathing and anticholinergic drugs may normally lead to dryness of mouth and loss of normal skin laxity in elderly may confound skin turgor estimation in elderly individuals.<sup>11</sup>

Laboratory investigations reveal increase in blood urea nitrogen / s. creatinine ratio (Pre renal ARF) >20:1 with decreased urinary sodium. However, use of diuretic therapy as in congestive heart failure or cirrhosis patients may alter urinary sodium profile.

The following risk factors have been attributed to predispose for increased fluid losses:

1. Female gender.
2. Age > 85 years.
3.  $\geq 4$  co morbid conditions.
4.  $\geq 4$  medications.
5. Bedridden status.
6. Laxative use.
7. Chronic infections.

### Management of Hypovolemia

If patient is conscious, then oral supplementation of water, low sodium rehydration solutions, fruit-juices without added salts are used if there is coexistent hypernatremia. Patients with normal plasma sodium may be treated with water and sodium supplementation or with isotonic rehydration solutions available. Intravenous fluids are indicated in patients with poor oral acceptance or those who cannot take oral fluids.  $\frac{1}{2}$  normal saline or dextrose in water may be given intravenously if hypernatremia is present and isotonic

saline (normal saline) infusion if coexistent hyponatremia is present.

### Hypernatremia

Hypernatremia, a common electrolyte disorder, is defined as rise in serum sodium concentration exceeding 145 meq/L.<sup>6</sup> Sodium is a major extra cellular cation which contributes to tonicity and induces the movement of water across the cell membranes. Hypernatremia invariably denotes hypertonic hyperosmolality and leads to cellular dehydration. The resultant morbidity may be inconsequential or serious and life threatening. It frequently develops in hospitalized patients as an iatrogenic condition and some of its serious complications results not from the disorders but from inappropriate treatment of hypernatremia. Net water loss accounts for majority of cases of hypernatremia leading to relative deficit of water in relation to solute and less frequently from administration of sodium in excess of water. Elderly are more susceptible in view of impaired thirst mechanism in presence of water loss and inability to increase water intake due to compromised mobility and swallowing disability, altered mental status, and intubation. The common causes of hypernatremia are mentioned in table 1.

**Table 1: Causes of hypernatremia**

- Febrile illnesses
- Insensible losses -fever, tachypnea, increased catabolism
- Infirmary
- Surgery
- Nutritional supplementation
- Inadequate free water intake.
- IV solutes (sodabcarb, hypertonic saline etc.)
- Diabetes mellitus
- Diarrhoea
- Gastro intestinal bleeding
- Diuretic abuse
- Progressive and impaired thirst mechanism
- Diabetes insipidus

### Signs and symptoms

It is manifested as depression of sensorium, irritability, seizures, focal neurological deficits, signs of volume depletion, fever, nausea or vomiting, laboured respiration, intense thirst (if thirst mechanism is intact).

The signs and symptoms of hypernatremia are most likely related to a variety of anatomical

derangements. The loss of volume and shrinkage of brain cells associated with the hyperosmolar state cause tearing of cerebral vessels with cerebral bleed, subarachnoid hemorrhage, permanent neurological damage or death. Brain shrinkage is countered by an adaptive response that is initiated promptly and consists of solute gain by the brain that tends to restore lost water. This response leads to normalization of brain volume and accounts for the milder symptoms of hypernatremia that develops slowly.

In early phases, the entry of sodium and chloride mitigates the loss of water. After seven days, the brain cells incorporate idiogenic osmoles with increased intracellular osmolality by agents like amino acid taurine, urea, glutamine, glycerophosphoryl choline and myoinositol.

### Treatment

Management of hypernatremia requires correction of prevailing hypertonicity and management of the underlying causes like fever, hyperglycemia, restricting diuretics, laxatives etc.

In patients with hypernatremia that has developed over a period of hours (e.g. with sodium overloading) early correction improves the prognosis without increasing the risk of cerebral edema, as accumulated electrolytes are rapidly extruded from brain cells. In such patients, reducing the serum sodium concentration by 1meq/L/hour is appropriate. A slow rate of correction is required in patient's with hypernatremia of longer or unknown duration because the full dissipation of accumulated brain solutes occurs over a period of several days. In these patients, reducing the serum sodium concentration at a maximal rate of 0.5 meq/L/hour prevents cerebral edema and convulsions. It is recommended to lower 10 meq/L/day in all patients of hypernatremia (except in those with acute hypernatremia) with target level to be achieved of 145 meq/L. Management of ongoing losses of hypotonic fluids by replacement, treatment of seizures with anticonvulsant therapy and adequate ventilation is desired.

The preferred route of administration is the oral route or nasogastric tube. Intra venous supplementation is desired if this is not feasible. Hypotonic fluids like 5% dextrose in water, 1/2 normal saline are preferred. The more hypotonic the infusate, lower is the infusion rate required. Volume of the infusate should be restricted to that required to correct hypertonicity. Isotonic saline is not suitable for primary correction of

hypernatremia. The rate of infusion has to be adjusted taking care not to correct the deficit very quickly. In elderly having hypernatremia associated with hypodipsia, 1 to 2 litre of water per day can be given. Chlorpropamide and desmopressin (dDAVP) have been used in elderly patients with adipsia.

### Hyponatremia

It is defined as a decrease in the serum sodium <135 meq/L.<sup>6</sup> It may be associated with low, normal or high tonicity of plasma while hypernatremia is associated mainly with hypertonicity. The severity of symptoms and signs depend on the degree of hyponatremia and the rapidity with which serum sodium concentration decreases. Hyponatremia is a common cause of delirium in elderly patients.

#### Types of Hyponatremia

A) Dilutional hyponatremia: It is the most common type of hyponatremia encountered in clinical practice which is caused by water retention. If water intake exceeds the capacity of the kidneys to excrete water, dilution of body solutes result, leading to hypoosmolality and hypotonicity which can cause cerebral edema.

B) Hypertonic/Translocational hyponatremia: This can be illustrated by hyperglycemia which increases solute concentration leading to shift of water from intracellular to extra cellular space eventually causing dilution of sodium in the extra cellular fluid space.

C) Isotonic or euvolemic hyponatremia: It is found in various conditions associated with SIADH, hypothyroidism, adrenal insufficiency etc.

D) Pseudo hyponatremia: Increase in solid phase of plasma by hypertriglyceridemia or hyperproteinemia and serum sodium estimation by flame photometry (v/s ion specific electrode estimation of serum sodium) causes decreased estimation of serum sodium (laboratory artifact).

Elderly are predisposed to have a higher incidence of hyponatremia on account of medications or may have diseases like congestive heart failure, cirrhosis, nephrotic syndrome which present with dilutional hyponatremia.

Intake of hypotonic fluids or hypotonic fluid administration iatrogenically in the post operative period with varied conditions leads to increased ADH levels with increased retention of water causing ECF dilution and dilutional hyponatremia.

Primary polydipsia (excessive fluid intake in association with a neuropsychiatric disorder) may also be present in the elderly.

Massive absorption of irrigant solutions which do not contain sodium (e.g. during transurethral resection of prostate) can cause severe hyponatremia. If irrigant used contains glycine or sorbitol, hypotonic hyponatremia results and if irrigant contains mannitol, then isotonic hyponatremia may be caused.

The signs & symptoms of hyponatremia are mentioned in table 2.

**Table 2: Signs & Symptoms of Hyponatremia**

Symptoms	Signs
Lethargy, apathy Disorientation	Abnormal sensorium, delirium Depressed tendon reflexes Cheyne-Stokes respiration
Muscle Cramps	Hypothermia
Anorexia, nausea	Pathological reflexes
Agitation	Pseudo bulbar palsy, seizures

**Table 3: Factors predisposing to increased risk of neurological complication in elderly.**

Acute cerebral edema	Osmotic demyelination
1. Post operative state	1. Alcoholics
2. Elderly women on thiazide diuretics	2. Malnourished patients
3. Polydipsic patients	3. Hypokalemia
	4. Burns
	5. Previous Hypoxic episodes
	6. Elderly female on thiazide diuretics

Hypotonic hyponatremia causes entry of water into the brain, resulting in cerebral edema. As the surrounding cranium limits expansion of brain, intracranial hypertension develops with a risk of brain injury. Fortunately, solutes leave brain tissues within hours, thereby inducing water loss and ameliorating brain swelling.

This process accounts for relatively asymptomatic nature of even severe hyponatremia if it develops slowly. Nevertheless, brain adaptation is also the source of the risk of osmotic demyelination. Although rare, osmotic demyelination is a serious complication and can develop one to several days after aggressive treatment of hyponatremia by any method, including water restriction alone.

Shrinkage of the brain triggers demyelination of pontine and extra pontine neurons that can cause neurological dysfunction including quadriplegia, pseudobulbar palsy, seizures, coma or even death. Hepatic failure, potassium depletion and malnutrition increase the risk of this complication.

Diagnosis:

For evaluation of hyponatremia we have to look for volume status, orthostatic hypotension, mental status and plasma osmolality. Plasma osmolality (Posm) is calculated using formula

$$\text{Posm} = 2 \text{ Na}^+ + \text{glucose}/18 + \text{BUN}/2.8$$

If Posm is low, then assess volume status - if edema is present then causes like cirrhosis, nephrosis, etc should be looked into.

If orthostatic hypotension is present, then renal, adrenal, GI disorders that lead to salt and water loss in GI fluids and urine should be searched.

When neither edema nor orthostasis is present, the diagnosis of pure water retention due to either excessive oral intake or SIADH may be present.

If hyponatremia is present with normal plasma osmolality and without neurological changes, the diagnosis is possibly of pseudohyponatremia.

The various types of hyponatremia with their diagnosis and prospective treatment strategies are illustrated in figure-5.

We have to treat the underlying conditions. In CHF and cirrhosis fluid retention has to be managed while hormone replacement therapy is required in Addison's disease and hypothyroidism. Management of severe hyponatremia (<125 mmol/L) depends upon the rapidity of its occurrence and presence of symptoms. Symptomatic acute (<48 hrs) hyponatremia requires emergency correction with hypertonic saline 1-2 ml/kg/hr with co administration of furosemide. While chronic symptomatic hyponatremia (>48 hrs.) requires some immediate correction followed by water restriction upon 10 % increase of sodium or resolution of symptoms. We have to perform frequent measurements of serum and urine electrolytes. Sodium correction should not exceed 1.5 mmol/ L /hr or 20 mmol / day. For asymptomatic chronic hyponatremia no immediate correction is needed however long term management is required. Identification and treatment of reversible causes and water restriction is useful. Demeclocycline

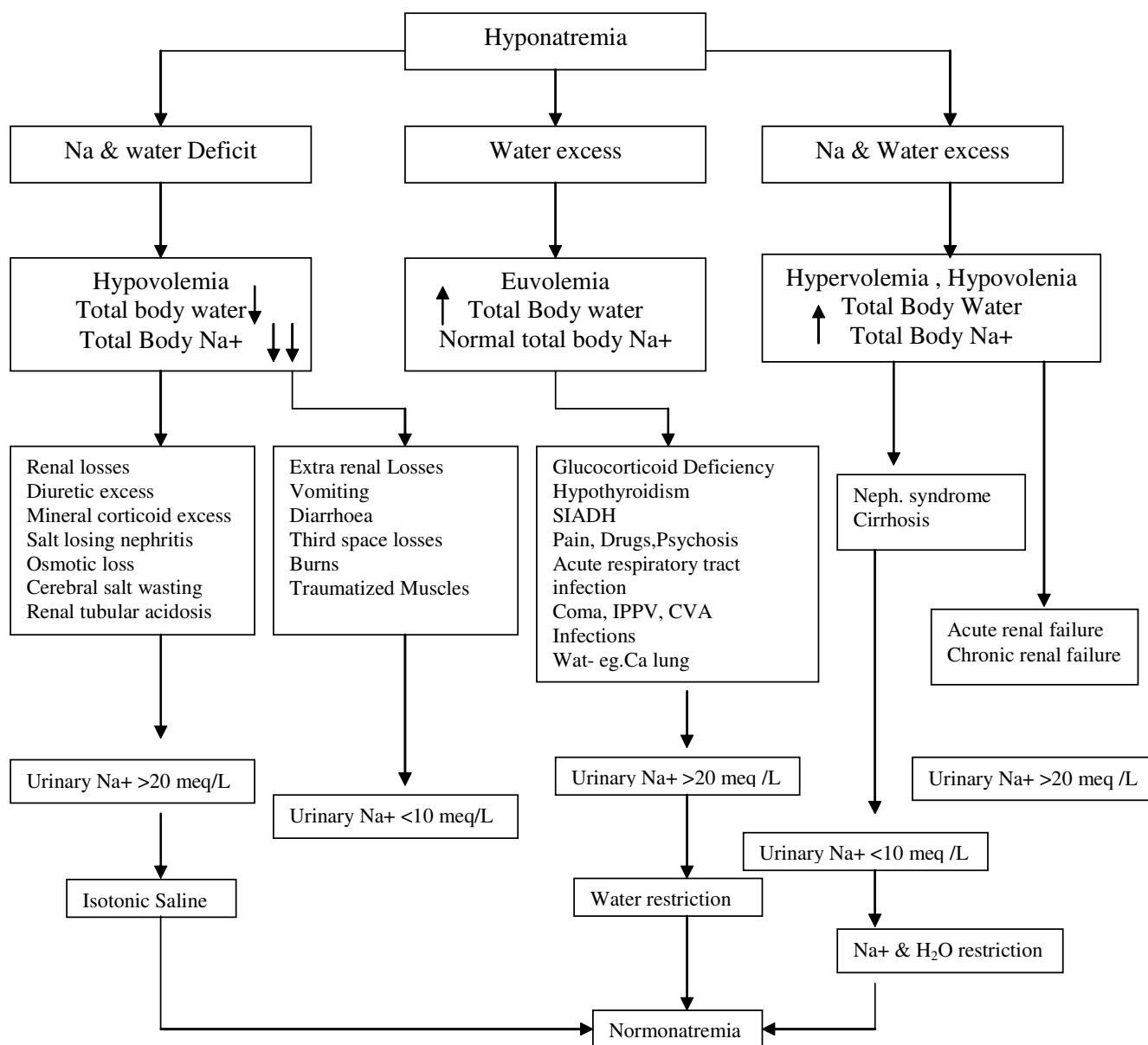


Fig. 5: Flow chart illustrating the type of hyponatremia, causes, diagnosis and treatment options.

300 – 600 mg bid and Urea 15-60 g/d have also been used.

**Potassium metabolism and its associated disorders**

Potassium is physiologically responsible for maintaining neuroexcitatory and cell membrane functions. Geriatric population has a higher incidence of hypertension, diabetes mellitus, coronary artery disease, congestive heart failure and renal insufficiency. All these conditions predispose to the use of medications that interfere with transcellular potassium homeostasis and renal handling of potassium. Ageing is usually

accompanied by a decline in glomerular filtration rate and a decrease in the kidney's ability to compensate for electrolyte abnormalities. There is also associated decreased plasma renin activity and aldosterone levels.<sup>13,14</sup> The net effect is increased susceptibility to potassium level imbalance in this age group. Intricate mechanisms are involved to maintain the narrow normal range of 3.5-5 meq / L despite widely varying dietary intake and relatively huge intra cellular potassium stores (98 % of total body potassium).

Age related changes in potassium homeostasis

**Table 4: Renal causes of hyperkalemia**

GFR >20ml/min	GFR<20ml/min	
Aldosterone deficiency	Tubular hyperkalemia	
<ul style="list-style-type: none"> <li>• Addison's disease</li> <li>• Hyporeninemic - Hypoaldosteronism</li> <li>• Drugs                             <ul style="list-style-type: none"> <li>-ACEI</li> <li>-NSAID's</li> <li>-Heparin</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• Acquired</li> <li>• Obstructive uropathy</li> <li>• Amyloidosis</li> <li>• Connective tissue diseases</li> <li>• Interstitial nephritis</li> <li>• AIDS</li> <li>• Drugs</li> <li>• K+Sparing diuretics</li> <li>• Trimethoprim etc.</li> </ul>	<ul style="list-style-type: none"> <li>•ARF</li> <li>•CRF</li> </ul>

**Table 5: Extra renal causes of hyperkalemia**

Pseudohyperkalemia	Increased intake or tissue release	Redistribution
<ul style="list-style-type: none"> <li>• Hemolysis</li> <li>• Thrombocytosis</li> <li>• Leucocytosis</li> </ul>	<ul style="list-style-type: none"> <li>• I.V./oral intake</li> <li>• Hemolysis</li> <li>• Rhabdomyolysis</li> <li>• Tumorlysis</li> <li>• Stored blood administration</li> </ul>	<ul style="list-style-type: none"> <li>• Metabolic</li> <li>• Insulin deficiency/DKA</li> <li>• B Blockade</li> <li>• Periodic paralysis</li> <li>• Digitalis over dosage</li> </ul>

includes a physiological decline in GFR and renal blood flow after the fourth decade by approximately 1 ml/ min / year or an average rate of 8 ml/ min/ 1.73m<sup>2</sup> / decade in most patients without renal disease. There is a documented decline in distal tubular function with impairment in the tubular response to acidosis. The ability to conserve sodium in response to low salt intake is affected and the potential for disposal of potassium and acid load declines with increasing age.<sup>7,15, 16</sup> Age related neurohormonal changes which affect potassium balance include a decline in plasma renin and aldosterone levels, blunted aldosterone response to potassium administration and a rise in levels of atrial natriuretic peptide (ANP) with ageing. ANP is a powerful suppressor of aldosterone secretion both in vivo and in vitro. The mechanism of rise of ANP levels is unknown. These neurohormonal changes lead to hypo reninemic hypoaldosteronism in elderly with increased susceptibility to hyperkalemia. Clinically they present with weakness, syncope and a tendency to develop cardiac arrhythmias. The laboratory features include hyperkalemia, variable degree of renal impairment,

glucose intolerance, unresponsive hyporeninemia and hypoaldosteronism.<sup>16</sup> Use of various medications with altered physiological function in relation to potassium metabolism in elderly individuals can predispose to potassium level imbalance e.g. use of ACE inhibitors, angiotensin receptor blockers, NSAID's, potassium sparing diuretics, β adrenergic antagonists, use of immunosuppressants like cyclosporine and tacrolimus, lithium intake, digitalis intoxication, trimethoprim etc.<sup>16</sup>

### **Hyperkalemia**

It is defined as plasma potassium concentration >5 meq / L occurring as a result of either increased potassium release from intracellular stores or decreased renal losses. Increased intake is rarely the sole cause of hyperkalemia. Age related decline in renal functions which are associated with alterations in neurohumoral mechanism predispose the elderly to hyperkalemia. It may present in a subtle manner but it may be a life threatening electrolyte abnormality. It often becomes clinically relevant following prescription of one or more drugs which raise serum potassium levels, during

Table 6: Treatment of hyperkalemia

Medication	Mechanism of action	Dosage	Peak action
Calcium gluconate	Antagonism of membrane action	10-30 ml 10% solution I.V. over 2 min	~ 5 Min
Insulin and glucose	Increased K <sup>+</sup> entry into the cells	Insulin 10 u in 25-30 g of glucose I.V. over 30 min	2-4 hours
Sodium bicarbonate	"	44 meq. I.V. over 5 min	30-60 min.
B <sub>2</sub> agonist	"	Nebulisation	30-60 min
Sodium polystyrene sulphonate	Removal of excess K <sup>+</sup>	15 g resin in 100 ml sorbitol repeated after 4-6 hours, orally	2-4 hours
Hemodialysis	Removal of excess K <sup>+</sup>	Dialysate K <sup>+</sup> nil, over 2-4 hours session	30-60 min.

episodes of inter current illnesses or diseases which interfere with potassium homeostasis e.g. renal disease.<sup>6</sup>

### Signs & Symptoms

The elderly individual may present with weakness, paresthesias in moderate hyperkalemia (5.5-6 meq / L) and with weakness, progressing to flaccid paralysis if severe hyperkalemia is present (>6 meq /L).

The electrocardiogram changes include tall T waves, increased PR and QRS duration, AV conduction delay and loss of P waves which finally merge to sine wave configuration, ventricular fibrillation eventually leading to cardiac asystole.

### Treatment

Various modalities of treatment are available (Table 6). According to the clinical setting, severity and rapidity of hyperkalemia the choice has to be made.

### Hypokalemia

It is defined as plasma potassium concentration <3.5 meq / L. The common causes are:

- **Gastrointestinal**
  - Dietary deficiency
  - Vomitings, diarrhea
- **Renal**
  - Metabolic alkalosis
  - Osmotic diuresis

-Mineralo corticoid / gluco corticoid excess  
-RTA

- **Translocational**

- a) Insulin, b) alkalosis, c) B<sub>2</sub> agonist,
- d) hypokalemic periodic paralysis,
- e) hypothermia, f) TPN

In elderly individuals, the pertinent causes are diuretic abuse. On account of various co-morbidities like hypertension, congestive heart failure, elderly are frequently prescribed diuretics for long duration where potassium losses may be detected on routine investigations. Acute illness with uncontrolled vomiting, acute diarrhoeal illness and frequent laxative abuse in elderly individuals may also predispose to hypokalemia.

### Signs & Symptoms

The patient may present with proximal muscle weakness like difficulty in getting up from squatting position, lifting objects from the shelves, or combing hairs etc which may gradually progress to flaccid paralysis. This may be associated with abdominal distention (due to paralytic ileus), constipation and polyuria. The presence of polyuria is often marked with increased frequency of urine. Careful history taking and detailed elucidation of the symptoms with volume of urine passed in 24 hours, previous estimation of urine volume status if available, is desired. Severe hypokalemia may lead to respiratory paralysis and rhabdomyolysis. Thus, a strong clinical suspicion with detection of early subtle symptoms and recognition of grave symptoms

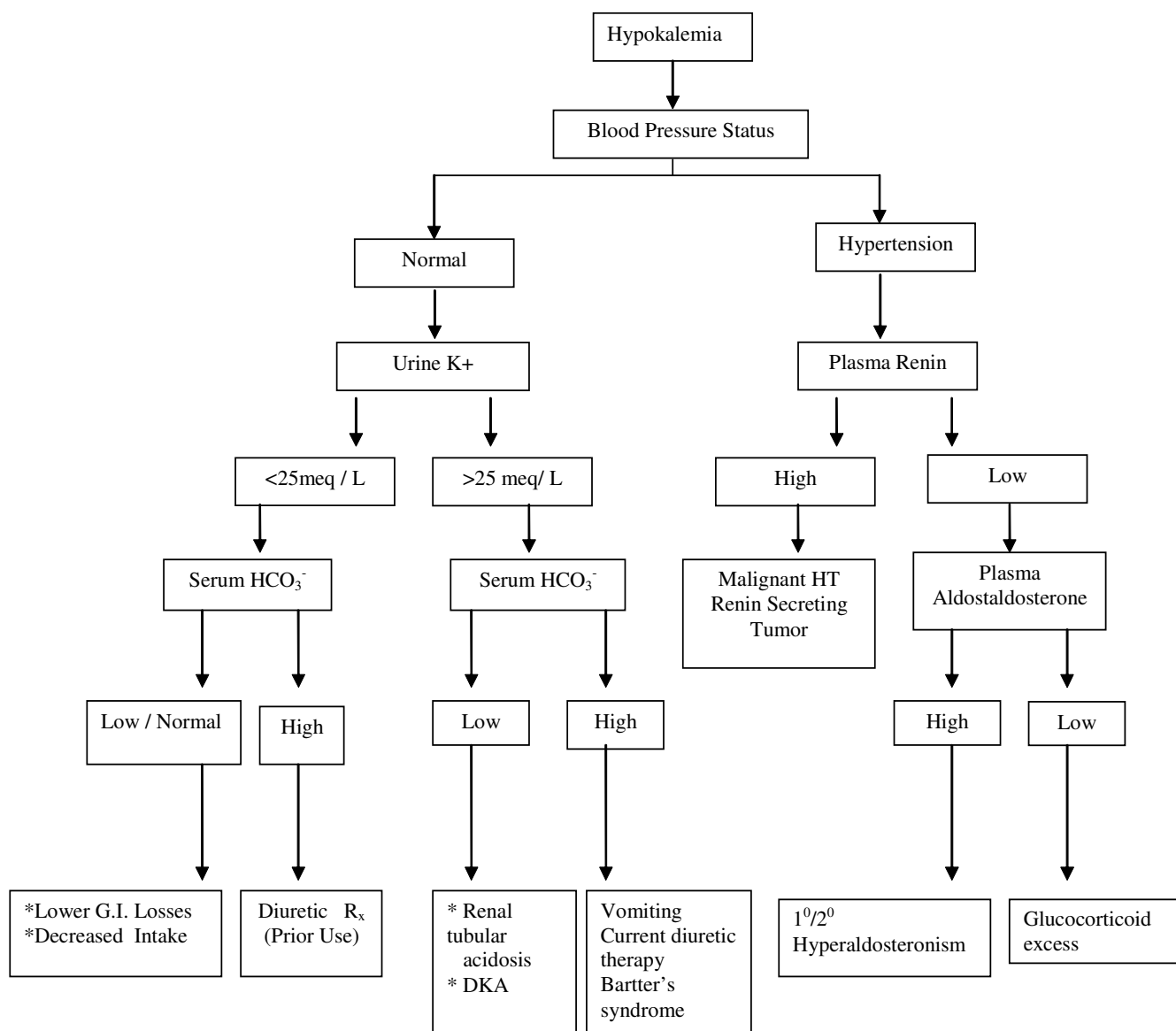


Fig. 6: Diagnostic workup of Hypokalemia.

with prompt institution of appropriate corrective measures may help in saving many lives in daily clinical settings. Electrocardiogram changes include ST segment depression (frequently mistaken as angina), T and U wave abnormalities may be detected. Arrhythmias may be present especially if patient has a prior history of coronary artery disease and digitalis therapy. Diagnostic work up of hypokalemia has been presented in figure 6.

**Treatment**

For mild to moderate hypokalemia, potassium

chloride tablets containing 10-20 meq potassium may be given orally at every 4-8 hour interval. The chloride salt may be preferred if there is concomitant metabolic alkalosis with hypokalemia. Potassium citrate or potassium bicarbonate is the preferred salt if there is coexistent metabolic acidosis. Replacement with bananas, orange juices which contain citrate and phosphate form of potassium may not be effective if metabolic alkalosis is present.

In severe hypokalemia, intravenous potassium chloride is given with following requisites:

1. Not >40 meq / L of potassium should be administered in a given solution

2. The rate of infusion should be <10-20 meq / hour with total dose not >200 meq / 24 hour is a safe rate of infusion.

3. Intravenous potassium chloride should ideally be given through a central line.

Unmasked hypocalcemia, hypomagnesemia should also be searched as neuro muscular weakness in this setting may not improve with potassium supplementation alone

### Preventive measures

The above fluid and electrolyte disorders require following strategies for their prevention

1. Maintenance of adequate fluid intake and focus on reduction of insensible losses by tepid sponging, antipyretics ( when indicated) and moving the elderly individual to cooler environment when fluid and sodium abnormalities are present e.g. hypovolemia, hypernatremia.

2. Monitoring of serum electrolytes, blood urea, serum creatinine would complement regular clinical assessment and would help to identify electrolyte abnormalities and renal failure at an early stage.

3. Maintenance of adequate hydration status by regular hydration status evaluation of patients with poor intake or on diuretic therapy can prevent its initiation or worsening of preexistent fluid and electrolyte abnormality.

4. Inter-current illnesses such as infection should be treated promptly and when deterioration is present in clinical condition, to shift the patients to intensive care can reduce likelihood of progression to established renal failure or life threatening electrolyte derangements.

5. A detailed enumeration of co existent co-morbid conditions in history and from previous treatment records, including the various medications prescribed for their management, is essential as disorders like hypokalemia, hyperkalemia can be detected at an early phase by these measures. Judicious use of various drugs like ACE inhibitors with loop diuretics can prevent the occurrence of electrolyte imbalance from the beginning.

6. Identification and early treatment of at risk patients with awareness of the physiological decline

in body's restorative function is essential. A combined effort and care from treating units can significantly decrease the related morbidity and mortality from these disorders.

### References

1. Warren JL, Bacon WB, Harris T, et al. The burden and outcomes associated with dehydration among US elderly. *Am J Public Health*. 1994;84:1265-1269.
2. Weinberg AD, Pals JK, Mc Glinchey-Bertho R, et al. Indices of dehydration among frail nursing home patients: highly variable but stable over time. *J Am Geriatr Soc* 1994; 42:1070-1073.
3. Snyder NA, Feigal DW, Arief AI, Hypernatremia in elderly patients. *Ann Intern Med* 1987;107:309-319.
4. Kleinfeld J, Casimir M, Bona S. Hyponatremia as observed in a chronic disease facility. *J Ann Geriatr Soc* 1979; 27: 156-160.
5. Mange K, Matsuura D, Cizman B, et al. Language guiding therapy: The case of dehydration versus volume depletion. *Ann Intern Med* 1997; 127: 848-853.
6. Singer GG, Brenner BM : Fluid and electrolyte disturbances in Kasper, DL, Braunwald, E, Fauci, AS et al (ed) Harrison's Principals of Internal Medicine, 16<sup>th</sup> ed Mc Graw Hill 2005 pp 252-263
7. Marcias-Nunez JF, Cameron JS; Renal functions in elderly. Oxford Textbook of Nephrology. 1st ed. Cameron JS, Davison A, et al (eds.). Oxford University Press, 1992, p 56-70.
8. Fisló LC, Murphy DJ, Elahi D, et al. Renal sodium excretion in normal ageing: Decreased excretion rates lead to delayed handling of sodium loads. *Geriatric Nephrology and Urology* 1995; 4:145-149.
9. Asplund R, Aberg H. Diurnal variation in the levels of antidiuretic hormone in the elderly. *J Intern Med* 1991; 229: 131-134.
10. Albert SG, Nakra BRS, Grossberg GT, et al. Vasopressin response to dehydration in Alzheimer's disease. *J Am Geriatr Soc* 1989;37 :843-849.
11. Gross CR, Lindquist RD, Woolley AC, et al. Clinical indicators of dehydration severity in elderly patients. *J Emerg Med* 1992;10:267-2674.
12. Lavizzo-Mourey R, Jhonson J, Stolley P. Risk factors for dehydration among elderly nursing home residents. *J Am Geriatr Soc* 1988; 36:213-218.
13. Silvd P, Brown RS, Epstein FH. Adaptation to potassium. *Kidney Int* 1977;11: 466-75
14. Weiner ID, Wingo CS. Hyperkalemia in acid base and electrolyte disorders. A companion to Brenner and rector's the kidney 1<sup>st</sup> ed. Philadelphia. Saunders.
15. Flynn A, Mc Greevy C, Mulkerrin EC. Why do older patients die in a heat wave? *Q J Med* 2005;98:227-229
16. Biswas K, Mulkerrin E C. Potassium homeostasis in the elderly *Q J Med* 1997; 90:487-492.