Some sodium, potassium and water changes in the elderly and their treatment

Vittorio E. Andreucci, Domenico Russo, Bruno Cianciaruso and Michele Andreucci
Chair of Nephrology, Faculty of Medicine, University Federico II of Naples, Naples, Italy

Abstract. Creatinine clearance decreases with age by 1 ml/min/year after 40 years of age, although serum creatinine remains constant because of reduction of muscle mass. Reduction of water intake may occur in the elderly because of a reduced sensation of thirst; this is associated with a tendency to lose water with urine. The capacity to respond to sodium load is impaired in aged kidneys, thereby leading to ECV expansion and hypertension. But there is also, in the elderly, a reduced capacity for retaining sodium (FENa is higher than in young subjects), making old subjects sensitive to salt depletion and ECV contraction. Hypernatraemia (Na > 150 mmol/l) is not infrequent in the elderly (1%) and is usually due to water deficiency (old subjects should be forced to drink), and rarely to iatrogenic excess of sodium. It is the abrupt occurrence of severe hypernatraemia that causes neurological symptoms due to dehydration and brain shrinking, which may lead to cerebral haemorrhage and death. Hyponatraemia (Na < 130 mmol/l) is frequent among the elderly (7–11%) and is mainly due to water overload, which is usually iatrogenic. Hypovolaemic hyponatraemia occurs when salt depletion causes ECV contraction >10%, and is due to water retention in an attempt to normalize ECV. Hypervolaemic hyponatraemia is due to ADH hypersecretion because of a decrease in ‘effective’ circulating blood volume. ‘Pseudohyponatraemia’ may occur because of hyperlipidaemia or hypoproteinaemia. It is the abrupt occurrence of severe hyponatraemia that causes neurological symptoms (water intoxication), secondary to the oedematous swelling of the brain within the skull. While rapidly occurring hyponatraemia may be lethal, slowly occurring hyponatraemia is usually asymptomatic. Rapid correction of hyponatraemia may cause cerebral dehydration and ‘osmotic demyelination syndrome’ (‘central pontine myelinosis’). Decrease (e.g. by diuretics) or increase (e.g. by ACE-inhibitors, non-steroidal anti-inflammatory drugs, beta-blockers) or serum potassium may occur in the elderly. Diuretics should be used with caution in elderly subjects to avoid salt depletion, hypotension and renal function impairment.

Key words: demyelination; diuretics; hypernatraemia; hyponatraemia; potassium; sodium

Behaviour of glomerular filtration rate in the elderly

Renal mass and function decrease in man from the age of ~40 years on. Glomerular filtration rate (GFR) reduction is ~1 ml/min/year, while reduction in renal blood flow (RBF) is even greater. The decrease of RBF in the elderly is attributed both to vascular atheromathosis and sclerosis and to increasing renal vascular resistance due to persistent arteriolar vasoconstriction. A significant contribution to the progressive decrease of GFR is undoubtedly the increase in the number of sclerotic glomeruli with time: 5% of

Correspondence and offprint requests to: Professor Vittorio E. Andreucci, Direttore Cattedra di Nefrologia, Nuovo Policlinico, Via S. Pansini 5, I-80131 Napoli, Italy.

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glomeruli are sclerotic at the age of 40, increasing up to 10–30% at the age of 80 [1].

In clinical practice serum creatinine is used as an index of renal function. In the elderly, however, such an index may be misleading. While creatinine clearance, in fact, decreases with age, even without renal disease, from a mean value of 125 ml/min at the age of 40 to a mean value of 78 ml/min at the age of 80, serum creatinine remains constant (between 0.74 mg/dl and 0.80 mg/dl) throughout life [8]. This phenomenon is due to the reduced production of creatinine from muscle creatine because of a reduction of muscle mass with age [1,2].

In the same fashion that GFR and RBF decrease, other renal functions—such as Tm\textsubscript{PAH}, Tm\textsubscript{Glucose}, and water and sodium excretion—are impaired with age, even without renal disease.

**Behaviour of water metabolism in the elderly**

Total body water is reduced (to ~55% of body wt) in healthy aged persons [3], presumably because of a greater proportion of fat in the body mass [4], but plasma and blood volume are usually normal [4–6].

The ability to excrete a water load and to dilute urine is impaired in the elderly [7–10] because of the reduction in the number of functioning nephrons with age. On the other hand, there is in the elderly a loss of water with urine, together with an increase in the thirst threshold, resulting in a reduction of fluid intake [11].

**Thirst**

In normal subjects water intake usually exceeds water needs so that normal subjects are not thirsty; if they are water deprived or challenged with water loss, hypertonicity of both intra- and extracellular fluids will occur. This stimulates thirst and the resulting ingestion of water will normalize fluid tonicity. Under such circumstances, the most important stimulating factor is the decrease in cell volume secondary to the hypertonicity [12]. Another stimulus for thirst is a severe extracellular volume (ECV) depletion; in such a condition stimulation of the left atrial or arterial baroreceptors will transmit the signal to the central nervous system, leading to a sensation of thirst [12].

A reduced water intake may occur in elderly subjects due to a variety of illnesses that can limit a patient's opportunities to ask for water; this may occur, for instance, following cerebrovascular accidents that impair their consciousness. Under such circumstances, medical staff should take care of to administer an adequate amount of water, to prevent water dehydration and hypernatremia. A reduction in water intake may occur even in healthy, alert, aged subjects due to a reduced sensation of thirst (hypodipsia) [12,13].

**Antidiuretic hormone**

The main stimulus for secretion of antidiuretic hormone (ADH) is an increase in plasma osmolality; an increase of 1% is sufficient to stimulate ADH secretion. Thus the main factor in controlling ADH secretion is plasma tonicity [12]. A severe salt depletion (10%), however, can override the tonicity control of ADH, by stimulating ADH secretion even in conditions of hypotonicity [12].

Circulating plasma ADH in the elderly population is normal [14–16]—and even greater than in young subjects—because of a greater sensitivity of osmoreceptors [12].

**Renal regulation of water excretion**

Despite the normal blood ADH, the renal capacity for concentrating urine is impaired in the elderly [7,8,17,18] because of several factors: (i) decrease of GFR due to a decrease in the number of functioning nephrons [7,8,17]; (ii) reduction of the medullary interstitial tonicity with age, which is secondary to the osmotic diuresis of the remaining nephrons; osmotic diuresis impairs sodium chloride reabsorption in the ascending limb of the loop of Henle (as mirrored by the decrease of free water clearance) [19] and urea recirculation; (iii) relative increase of medullary blood flow, due to the relatively restricted amount of degenerative change in the medullary vasculature [20,21]; the increased medullary blood flow will dissipate medullary tonicity, thereby contributing to the reduction of the medullary interstitial tonicity; (iv) reduced sensitivity of the distal nephron to ADH.

Osmotic diuresis and impairment of antidiuretic mechanisms will cause a loss of water with urine [4,7,12,22]. There is also a tendency to nocturia in the healthy aged population [18], which may be related to the renal function impairment.

**Behaviour of sodium metabolism in elderly**

The capacity of the aged kidney to respond to sodium load is impaired [23–27]. Thus, unnecessary salt loading will cause ECV expansion which may lead to hypertension and heart failure.

There is, in the elderly, a reduction in the renal capacity for retaining sodium. In old subjects, dietary salt restriction takes much longer than in the young to produce a reduction in urinary salt excretion [19,28–30], i.e. the external sodium balance upon any change in salt intake is reached, but the time necessary for that is much longer, and a significant sodium loss via urine will occur. This phenomenon makes old subjects more sensitive to salt depletion, which is consistent with the clinical observation of the frequent occurrence of salt depletion in geriatric patients [19,28]. Since sodium, with the anions chloride and bicarbonate, is the main extracellular solute, salt depletion will
cause ECV contraction. Hence elderly subjects are prone to ECV contraction.

Fractional urinary excretion of filtered sodium (FE\textsubscript{Na}) is greater in healthy elderly people than in young healthy people, indicating an important impairment of tubular function in normal old subjects [4]. Consistent with the concomitant nocturia, there is a frequent tendency for elderly normal subjects to have a greater urinary excretion of sodium during the night [18].

Macias-Núñez [4] has demonstrated that the site of impairment of sodium tubular reabsorption in aged individuals is not in the proximal tubule, but in the thick ascending limb of the loop of Henle. The reduction of sodium reabsorption in the loop of Henle leads to two consequences: (i) a reduction in medullary tonicity, thereby causing a loss of water with urine and a reduction in maximal urine concentration as is typically observed in elderly subjects; (ii) an increase in urinary sodium excretion. However, the reasons for such a reduction in tubular renal sodium handling by the ageing kidney has not been clarified as yet. Factors such as interstitial fibrosis, peritubular forces, aldosterone and sex hormones may contribute to the defect.

On the other hand, in the elderly, plasma renin activity (PRA) and serum and urinary aldosterone are reduced, when compared to young subjects, particularly after salt restriction [28, 31–33]. The reduction in aldosterone secretion associated with a reduced sensitivity of the distal nephron to aldosterone both contribute to the urinary loss of sodium.

Circulating atrial natriuretic factor (ANF), which is known to increase urinary sodium excretion as well as to suppress renin and aldosterone production, is increased after postural changes [34], thereby contributing to the tendency to lose salt with urine. On the other hand, a bolus of ANP will induce a greater diuresis, natriuresis, calciuresis, and urinary and plasma cGMP concentrations than in young subjects [35].

The tendency of elderly subjects to lose sodium via urine does not imply a reduction of total body sodium under normal circumstances [4, 36]. With a varied diet, their sodium intake can, in fact, balance the high urinary sodium excretion; only when the capacity of salt retention is challenged, as occurs in fasting subjects (i.e. with no salt intake), is total body sodium reduced. If we consider that any disease in old subjects causes a loss of appetite, fasting in these subjects is quite frequent [4].

Sodium content in red blood cells (RBCs) is increased in healthy elderly persons [37–39], presumably because of impaired ion transport by the RBC membrane [37, 39, 40], possibly due to alteration of the Na"K"-ATPase dependent pump [41].

In addition to the above factors, as mentioned previously osmotic diuresis in the surviving nephrons will also contribute to the urinary loss of sodium.

**Serum concentration of sodium**

Serum concentration of sodium is usually normal in the healthy elderly population, with no gender difference [42, 43]. Problems may arise only under stressed conditions, when the renal capacity to maintain normal serum sodium is challenged [4].

**Hypernatraemia**

Hypernatraemia (i.e. serum sodium concentration >150 mmol/l) is not infrequent in the elderly population [44], occurring in 1% of elderly subjects, and is usually due to absolute or relative (i.e. in excess of sodium) water deficiency. Hypernatraemic dehydration is often observed in hospitalized elderly individuals; this reflects the tendency of the elderly to suffer water loss, as they are unable to replace water [45, 46].

Clinical conditions leading to absolute or relative water deficiency may be summarized as follows:

1. **Reduced intake of water**, due to dementia, comatose state, cerebrovascular accident, dysphagia, etc. To prevent water deficiency in these conditions it is necessary to infuse glucose solutions i.v., as it is not possible for such patients to drink water.

2. **Loss of water due to fever** (e.g. in the course of infectious diseases); the reduced sensation of thirst in aged individuals tends readily to cause insufficient water replacement by drinking. This is why, during infectious diseases or in any disease causing fever, elderly subjects should be forced to drink at least 2 l of fluids, in form of tea, juice, soup or plain water (provided that renal function is not severely impaired).

3. **Loss of water relative to sodium** due to sweating (e.g. due to a hot climate or fever) or to osmotic diuresis (due to hyperglycaemia, use of radiocontrast media, etc.).

Only in rare conditions is hypernatraemia due to excess of sodium; this usually occurs because of iatrogenic causes, such as i.v. infusions of hypertonic sodium chloride or sodium bicarbonate solutions or, in uraemic patients, the use of excessive sodium concentrations in dialysis solutions [4].

**Symptoms and treatment of hypernatraemia**

Symptoms caused by hypernatraemic dehydration are neurological, and are due to the diminution of brain cell volume with progressive obtundation, leading to coma and even death [12, 47]. In fact, the brain can adapt well to osmotic changes (patient survival has occurred with serum sodium concentrations ranging from 85 to 272 mmol/l), but such adaptation requires time—the brain does not tolerate abrupt changes of serum sodium [48–51]. It is therefore the abrupt occurrence of severe hypernatraemia that causes dehydration and shrinking of the brain, sometimes resulting in cerebral haemorrhage [52].

Prevention of hypernatraemia is based on adequate daily fluid administration by drinking (at least 2 l/day) or by i.v. infusion of 5% glucose solution, when the aged subject is unable to drink. It should be stressed,
however, that the use of large volumes of 5% glucose may cause hyperglycaemia, which can lead to loss of water by osmotic diuresis [47].

Treatment of hypernatraemia is based on adequate, but cautious, administration of water by mouth, when possible. Otherwise, once ECV contraction has been corrected with rapid i.v. isotonic saline infusion, hypotonic fluid should be infused i.v., very slowly (over 2–3 days), until the serum sodium concentration is <150 mmol/l. Water deficit may be calculated as follows:

Elderly men
\[ \text{water deficit} = \text{body weight} \times 0.55 (P_{Na} - 140)/P_{Na} \]

Elderly women
\[ \text{water deficit} = \text{body weight} \times 0.50 (P_{Na} - 140)/P_{Na} \]

Hyponatraemia

Hyponatraemia (i.e. serum sodium concentration <130 mmol/l) is frequent in aged individuals [53,54], occurring in 7–11% of elderly subjects; its occurrence is particularly high (up to 50%) in hospitalized aged patients [55–57]. The incidence of severe hyponatraemia (i.e. serum sodium <120 mmol/l) is 1–4% [28,53]. It has been claimed that females are more intolerant of hyponatraemia than male subjects, and have a greater susceptibility to brain injury. However, this has not been clearly demonstrated and, even if present, would be limited to young women in their reproductive years [51].

Hyponatraemia is usually due to water overload, which is mainly a secondary effect of iatrogenic causes, i.e. excess of water drinking or excessive i.v. infusion of electrolyte-free solutions (glucose or levulose solutions), as may occur in patients who are comatose or in a post-operative state.

During transurethral prostatic resection in the elderly, the isosmolar but electrolyte-free fluids used as irrigating solution may be absorbed and cause, after surgery, a severe hyponatraemia with normal plasma osmolality; this hyponatraemia is due to absorption of the solute (glycine, mannitol or sorbitol) that remains confined to the extracellular fluid [50,58].

Another important cause of hyponatraemia is severe salt depletion (hypovolaemic hyponatraemia) with an ECV contraction >10%; under such circumstances, the need to normalize ECV leads to water retention, despite the low tonicity of extracellular fluids.

Salt depletion in the elderly, as in young subjects, may be due to non-renal or renal losses. Non-renal losses of sodium include vomiting; diarrhoea; gastric, enteric or biliary drainage; enterostomy; abuse of laxatives; skin burns; sweating; sequestration of plasma volume in the ‘third space’, because of pancreatitis, traumatized muscles, intestinal obstruction; and peritonitis. Renal losses include abuse of diuretics, mineralcorticoid deficiency and salt-losing nephritis. Hypovolaemic hyponatraemia in the elderly population is frequently due to prolonged use of diuretics [57], usually given to aged patients for treating hypertension or for control of oedema caused by congestive heart failure.

The clinical signs of salt depletion are postural hypotension and reduction in central venous pressure; skin turgor does not help because of the reduction in skin elasticity in the elderly.

Hyponatraemia may be observed also in oedematous states; these include nephrotic syndrome, congestive heart failure and hepatic cirrhosis. In these conditions total body water and sodium is in excess and hyponatraemia (hypervolaemic hyponatraemia) is due to ADH hypersecretion resulting from the decrease in ‘effective’ circulating blood volume. In other words the kidney behaves as a hypoperfused kidney, thereby increasing proximal tubular reabsorption, in an attempt to restore its ‘normal’ perfusion; by doing so, however, the distal delivery is reduced with a decrease in free water generation and excretion [12].

A hyperglycaemic syndrome due to insulin deficiency is frequently observed in the elderly. This syndrome is characterized by a slow and progressive increase of blood glucose, with ECV contraction due to loss of water and sodium by glucose-induced osmotic diuresis and inadequate salt intake; it is frequently associated with hypernatraemia. This insulin deficiency is often precipitated by stroke, myocardial infarction or sepsis [47,59].

In diabetic patients, severe hyperglycaemia may cause dilutional hyponatraemia because of osmotic redistribution of water between intra- and extracellular compartments. In old uraemic patients undergoing regular dialysis treatment, (iatrogenic) hyponatraemia may occur following the use of low sodium concentrations in dialysis solutions.

Rare conditions of hyponatraemia due to chronic water overload are: hypothyroidism, deficiency of glucocorticoids and the syndrome of inappropriate ADH secretion [28]. The latter syndrome is usually secondary to ectopic hormone production by lung (bronchogenic) carcinoma or to central release of ADH due to central nervous system diseases (meningitis, encephalitis, brain tumours, cerebral thrombosis or haemorrhage, subdural haematoma), intratracheal diseases (pneumonia, pulmonary tuberculosis), but may also be due to carcinoma of the duodenum, thymus or pancreas, Hodgkin’s disease, myeloid leukaemia, or even drugs such as cyclophosphamide, clofibrate, barbiturates or chlorpropamide.

Pseudohyponatraemia

The condition known as ‘pseudohyponatraemia’ occurs in patients with severe hyperlipidaemia or hyperproteininaemia. It is caused by the fact that electrolytes are located in the aqueous phase of plasma, and may be diagnosed by measuring sodium concentration in plasma water rather than in total plasma.

Pseudohyponatraemia in hyperlipidaemia is usually due to high values of triglycerides (>1500 mg/dl) rather
than to increased cholesterol; such high values are quite uncommon in an elderly population.

**Symptoms and treatment of hyponatraemia**

Usually there are no symptoms of hyponatraemia, unless serum sodium decreases below 130 mmol/l and the decrease is acute. The important factor, in fact, is the acuteness of the osmolarity change: the faster the change, the more severe the symptoms, which are mainly neurological—the skull restricts osmotic swelling in the brain.

The blood–brain barrier is permeable to water but not to solutes. Thus, in hyponatraemia, water, but not solutes, will cross the blood–brain barrier, along the osmotic gradient, to restore the osmotic equilibrium across it [48–50]. Because of its confinement within the rigid skull, the brain can tolerate only small oedematous increases of its volume, i.e. only changes not greater than 10% of serum concentration of sodium [48–50,60]. However, there are two mechanisms that prevent excessive cerebral enlargement: (i) the movement of interstitial fluid from the cerebral interstitium to the cerebrospinal fluid and of the latter to the systemic circulation; and (ii) the movement of solutes (potassium, free amino acids, methylamines, myoinositol) out of the brain cells, thereby decreasing the osmotic gradient across the cell membrane [48–50, 61,62]. These protective mechanisms, however, require 1–2 days to be effective [48–50]. Thus, the abrupt occurrence of severe hyponatraemia, causes brain cells to expand, resulting in severe cerebral oedema which may lead to transtentorial herniation; this can be fatal because of the inability of brain to adjust quickly to the change in its volume [12,60]. Even a severe hyponatraemia, which would be well tolerated if it occurred slowly, may be fatal if it takes place rapidly [51,60].

The syndrome of severe, rapidly occurring hyponatraemia is called ‘water intoxication’ [51,60] and includes the following symptoms: anorexia, nausea, vomiting, headache, weakness, loss of coordination, muscle cramps, agitation, tremors, disorientation, psychosis, delirium, seizures and coma [12,51]. It has been reported that, in humans, death may occur when serum sodium concentration is <120 mmol/l with a rate of decrease >0.5 mmol/l/h (12 mmol/l/day) [49,63].

Frequently, despite serum sodium levels of <130 mmol/l, the slow occurrence of hyponatraemia will make it asymptomatic, thereby preventing the therapeutic removal of its cause [55].

Symptoms of chronic, slowly occurring hyponatraemia are mild and usually appear at lower serum sodium concentration than in rapidly occurring hyponatraemia; they include anorexia, nausea, vomiting, weakness, muscle cramps, irritability; the old patient becomes confused or hostile, and sometimes stupor or seizures may occur [51].

Severe hyponatraemia (e.g. serum sodium of 108 mmol/l) requires treatment with hypertonic saline. The suggested safe infusion rate in severe cases of hyponatraemia is 1–2 ml/kg body wt/h of 3% saline solution for no more than 2–4 h; this will increase serum sodium by 1–2 mmol/l/h. In mild cases a correction rate of 0.5 mmol/l/h throughout the treatment (i.e. 12 mmol/l/day) is the ideal form of therapy [51]. These procedures will avoid neurological complications [53,64]. The rapid correction of hyponatraemia may, in fact, cause cerebral dehydration, because of the impossibility of rapidly restoring the normal cellular content of the solutes (potassium, free amino acids, methylamines, myoinositol), initially lost as a protection mechanism [48–50,61].

The syndrome resulting from rapid correction of chronic hyponatraemia has been called ‘osmotic demyelination syndrome’ [65], and occurs—for unknown reasons—several days after treatment. In some reported cases demyelination has been observed in the central pons (‘central pontine myelinolysis’) [64–81]. Severe liver disease, debilitating diseases and alcoholism predispose to the syndrome [49,50,65,76].

Symptoms of osmotic demyelination syndrome include transient behavioural disturbances, movement disorders and seizures in mild forms, a pontine disorder with quadriaparesis and unresponsiveness in more severe forms, up to a dramatic condition of a patient being awake, but unable to move or communicate, and requiring ventilator support [49–51,64–66,70–74,76–78, 80,81]. Fatal cases or cases with permanent neurological sequelae usually follow a treatment exceeding 12 mmol/l/day [51,63,65]. Sometimes improvement of a patient’s condition may occur within several weeks, but complete recovery does not take place [51].

Demyelination with spared neurons and axons in the centre of the basal pons is a common autopsy finding in fatal cases (central pontine myelinolysis); similar lesions, however, may be observed symmetrically distributed in extrapontine areas [51]. Magnetic resonance imaging (MRI) can demonstrate demyelination areas in patients with ‘osmotic demyelination syndrome’ 3–4 weeks after the clinical onset [73,75,80,81], but a normal MRI does not exclude the diagnosis [51,75,79,80].

Chronic hyponatraemia is frequently induced by diuretics (thiazide) in the elderly [49,50,64,65,81,82] and is quite often rapidly corrected by physicians [64,65]; on the other hand the recovery of diluting ability once diuretic has been stopped and the replacement of the potassium lost because of the diuretic will contribute to a fast correction of hyponatraemia [49,50]. Hence, diuretic-induced hyponatraemia may be followed, in elderly patients, by osmotic demyelination syndrome secondary to a rapid normalization of serum sodium concentration [51].

When hyponatraemia is due to salt depletion, i.v. infusion of isotonic saline solutions will readily normalize serum sodium concentration. Physicians are usually reluctant to infuse saline in depleted, aged subjects, because they are afraid of causing an increase in ECV, thereby precipitating congestive heart failure [47].
However, should be used with caution in old patients, to congestive heart failure, nephrotic syndrome. Diuretics, main indications to the use of diuretics in the elderly are similar to those in young subjects: hypertension, hypokalaemia, dislypidaemia, impotence and hypokalaemia -140/80, but standing BP was <90/50 with the patient complaining of nausea, vomiting and malaise. The patient was not thirsty. The i.v. infusion of isotonic saline solution readily normalized urine output, increased both supine and standing BP to 180/90, and decreased serum creatinine to 0.8 mg/dl and plasma urea to 40 mg/dl.

**Behaviour of potassium metabolism in the elderly**

In healthy aged subjects serum potassium is usually normal [83-85]. Because of the reduction of PRA and serum aldosterone, however, there is a tendency for the aged kidney to excrete less potassium than normal in urine, thereby accounting for the ready occurrence of hyperkalaemia [86] under particular circumstances, such as following administration of ACE inhibitors, non-steroidal anti-inflammatory drugs, potassium-sparing diuretics or beta-blockers [4].

The use of loop diuretics, however, makes hypokalaemia a frequent condition [57,87]. Red blood cell potassium is decreased in the healthy aged population [24,88,89], thereby suggesting that intracellular potassium is decreased [4]. Many studies have demonstrated that total body potassium is lower in elderly than in young subjects [2,24,40,89-94], presumably because of the reduction of muscle mass [24,40,88,90,93,94], and because of a tendency to a lower potassium intake with food (old subjects usually ingest less fish, meat and fresh fruits than young individuals) [84,95,96].

**Use of diuretics in elderly**

Main indications to the use of diuretics in the elderly are similar to those in young subjects: hypertension, congestive heart failure, nephrotic syndrome. Diuretics, however, should be used with caution in old patients, to avoid salt depletion, hypotension and renal function impairment.

Hypertension is frequent in the elderly, in the form of combined systolic and diastolic hypertension (systolic BP >160 mmHg; diastolic BP >90 mmHg), as it occurs in young subjects, or in form of systolic hypertension (systolic BP >160 mmHg; diastolic BP <90 mmHg). In the elderly standing BP should be measured as well as supine BP, since a sudden reduction in BP may easily occur, particularly under anti-hypertensive therapy.

In recent years many authors have suggested the use of diuretics for treating not only combined systolic and diastolic hypertension, but also systolic hypertension [97,98]. The low values of PRA in hypertensive aged patients suggest an increase of plasma volume and this represents a clear indication for diuretic treatment [99].

In a retrospective analysis of 8428 hypertensive patients aged >65 years treated between 1982 and 1988, diuretics were the most common drugs used as anti-hypertensive agents, with a prevalence of 51%, followed by calcium channel blockers (14%), betablockers (13%), adrenergic antagonists (11%) and ACE inhibitors (5%); the use of diuretics was even greater in very old patients [100]. Diuretic therapy has been shown to decrease cerebrovascular and cardiovascular complications and the incidence of sudden deaths in hypertensive elderly patients [97-99,101].

The dosage of diuretics in elderly hypertensive patients should be low to avoid hypovolaemia, postural hypotension, reduction in central venous pressure and prerenal azotaemia, or even shock, in addition to other side-effects, such as glucose intolerance, hyperuricaemia, dislypidaemia, impotence and hypokalaemia with extrasystolic beats. The low dosage is necessary considering that after the same dose of frusemide, the urinary excretion of sodium has been shown to be greater in aged subjects than in young individuals [102].

Thiazide is the most used diuretic drug in aged hypertensive patients, at a dosage of 12.5 mg/day. In order to avoid hypokalaemia with the use of thiazide, the combination with amiloride, a potassium-sparing diuretic [99,101,103], or with ACE inhibitor [104], has been suggested. Diuretics are contraindicated in diabetic patients [105].

Loop diuretics for long-term therapy should be avoided in hypertensive elderly, because of their brisk and long-acting effects followed by a rebound phenomenon [106]. Torasemide, a new loop diuretic with a more prolonged diuretic effect than frusemide without rebound phenomenon [107-112], at a dosage of 2.5 mg/day in a single morning dose, has been shown to have the same anti-hypertensive efficacy as 25 mg/day of hydrochlorothiazide [113].

When treating hypertension in elderly patients, a beta-blocking drug may be used if diuretic therapy alone is not enough, [99].

The use of diuretics in congestive heart failure is undoubtedly useful, not only to correct the oedematous state, but also to improve heart function (and this may improve renal perfusion and GFR). A final indication for diuretic therapy is nephrotic syndrome.
Some electrolyte changes in the elderly

The abuse of powerful loop diuretics may be particularly dangerous in the elderly. It may cause severe salt depletion and functional renal failure (Fig. 2).

References

Some electrolyte changes in the elderly


