

Electrolyte Disorders Following Oral Sodium Phosphate Administration for Bowel Cleansing in Elderly Patients

Yichayaou Beloosesky, MD; Josef Grinblat, MD; Avraham Weiss, MD; Boris Grosman, MD, PhD; Uzi Gafter, MD, PhD; Avry Chagnac, MD

Background: Oral sodium phosphate is currently used for colon preparation prior to colonoscopy or barium enema. Sodium phosphate induces hyperphosphatemia, hypocalcemia, and hypokalemia. Elderly patients are at an increased risk for phosphate intoxication due to decreased glomerular filtration rate, medication use, and systemic and gastrointestinal diseases. We investigated these electrolyte disorders and their correlation with creatinine clearance, coexistent diseases, medications, and functional status.

Methods: Thirty-six hospitalized patients were included in the study. On day 1, patients were administered 2 doses of oral sodium phosphate. Venous blood samples for electrolyte determination were obtained at 7 AM on days 1, 2 (the procedure day), and 3. Urine samples were obtained from 10 patients.

Results: An increase in serum phosphorus level was correlated with a decreased creatinine clearance ($R = -0.52$; $P = .001$). Hypocalcemia and hypokalemia were present in 21 (58%) and 20 (56%) patients, respectively. Patients with a serum potassium concentration of 3.5 mEq/L

or less on day 2 had a lower serum potassium concentration on day 1 vs those with a serum potassium concentration greater than 3.5 mEq/L on day 2 ($P = .03$). Five (dependent patients) had a serum potassium concentration of 3 mEq/L or less and 2 had severe diarrhea, necessitating treatment. There were more demented patients with hypokalemia compared with normokalemic patients ($P < .05$). Urinary fractional excretion of phosphorus tripled on day 2 ($P = .01$). Potassium and sodium fractional excretion remained unchanged.

Conclusions: Sodium phosphate induces serious electrolyte abnormalities in the elderly. The frequency and severity of hypokalemia is due to intestinal potassium loss associated with inadequate renal potassium conservation and is apparently more prevalent in frail patients. Assessment of serum electrolytes, phosphorus, and calcium prior to sodium phosphate preparation is advised, and in selected patients, postprocedural assessment and correction may be required.

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COLONIC DISORDERS are of particular importance in elderly patients because of marked age-associated increases in their prevalence and differences in their presentation and prognosis compared with the young. More than two thirds of colorectal cancers occur in people 65 years or older, with the prevalence of colonic diverticula over 50% after age 70 years.¹

Oral sodium phosphate, a small volume osmotic cathartic, is currently used as a bowel cleansing preparation. Several studies have demonstrated that sodium phosphate is effective, less costly, and easier to prepare compared with polyethylene glycol and is the preferred method of preparation for colonoscopy for certain patient subgroups.²

Adverse effects of sodium phosphate use include consistent, transient in-

creases in serum phosphorus and sodium levels³⁻⁷ and transient decreases in calcium concentration.^{5,7} Other significant changes were noted in serum potassium, chloride, bicarbonate, magnesium, albumin, and serum osmolarity.^{3,5} However, no clinical adverse effects accompanied these metabolic changes. Marked metabolic acidosis with a large increase in the anion gap was reported in exogenous phosphate intoxication following accidental ingestion of a Fleet enema (C. B. Fleet Company Inc, Lynchburg, Va).⁸

Although the plasma phosphorus concentration may transiently increase above normal ranges, the well-hydrated adult patient with normal kidney function will not develop hyperphosphatemic acidosis after receiving a standard preparation of oral sodium phosphate.⁹ Hyperphosphatemia associated with phosphate preparation might result from ex-

From the Departments of Geriatrics (Drs Beloosesky, Grinblat, Weiss, and Grosman) and Nephrology (Drs Gafter and Chagnac), Rabin Medical Center, Beilinson and Golda Campuses, Sackler School of Medicine, Tel Aviv University, Petach Tikva, Israel. The authors have no relevant financial interest in this article.

cessive and/or repeated doses, increased intestinal absorption, or impaired renal excretion. Intestinal absorption will be facilitated by impaired transit with prolonged retention.^{10,11} Indeed, severe hyperphosphatemia has previously been described in 15 patients after administration of oral or rectal phospho-soda for bowel preparation.¹¹⁻¹⁴

Elderly patients are at an increased risk for phosphate intoxication due to a more sedentary lifestyle, altered gut motility, constipation, use of medications decreasing bowel motility and diuretics, and systemic and gastrointestinal diseases. There is also an age-related decline in renal function, which is frequently overlooked because the plasma creatinine level may remain within the normal range despite a 50% or greater decrease in the glomerular filtration rate compared with young individuals.⁸

Although hundreds of patients aged 15 to 91 years were enrolled in studies comparing the safety and efficacy of sodium phosphate vs polyethylene glycol preparations given for bowel cleansing before a colonoscopy, there are no studies investigating the electrolyte and acid-base changes induced by the oral sodium phosphate load, specifically in the elderly.²⁻⁷ The aim of the present study was 2-fold: (1) to investigate the electrolyte and acid base disorders induced by standard oral sodium phosphate preparation given before a colonoscopy or barium enema in elderly patients and (2) to assess these changes in relation to creatinine clearance, coexistent diseases (ie, Parkinson disease), medications (ie, anticholinergic agents), and functional and cognitive status.

METHODS

STUDY POPULATION

The study population comprised 36 consecutive patients 65 years or older and hospitalized in a geriatric department from January to October 2001, who underwent bowel cleansing with the standard phospho-soda preparation (Dexxon Ltd, Or-Akiva, Israel) for colonoscopy or barium enema. Exclusion criteria were creatinine level greater than 2.3 mg/dL (203.3 μ mol/L), symptomatic congestive heart failure, massive ascites, myocardial infarction within 6 months, cerebrovascular accident within 3 months, active inflammatory bowel disease, active diverticulitis, and bowel obstruction. The study was approved by the Rabin Medical Center Helsinki Committee, and all patients or their proxy gave informed written consent.

CLINICAL ASSESSMENT AND BOWEL PREPARATION

Baseline demographic details, medical conditions, medications, and indications for colon investigation were recorded. We used the Katz Index of Independence in Activities of Daily Living (Index of ADL)¹⁵ to categorize the patients into 3 functional groups based on a score of up to 12 points: independent (10-12 points), partially dependent (6-9 points), and fully dependent (0-5 points). The Mini-Mental State Examination¹⁶ was used to assess cognitive level. Each patient was assessed on day 1 of hospitalization and reassessed prior to discharge, and the higher score was recorded. Patients were classified into 3 cognitive groups: severely demented (≤ 14 points), moderately demented (15-23 points), and normal (24-30 points).

Patients followed the usual standard bowel preparation: on day 1, prior to the colon procedure day, 45 mL of sodium phos-

phate (containing 48 g of monobasic sodium phosphate and 18 g of dibasic sodium phosphate per 100 mL) were taken with a glass of water (200 mL) at 8 AM. The second dose of 45 mL of sodium phosphate was given between 6 and 7 PM. The total amount of phosphorus and sodium ingested was 11.6 g and 434 mEq, respectively. Patients were encouraged to drink fluids and eat a normal breakfast and light, semi-solid lunch and supper. This provides an additional 0.6 g of phosphorous. On day 2, the procedure day, a water rectal enema was done at 7 AM, and patients had a light breakfast. After the procedure, the patients were allowed to eat and drink ad libitum.

LABORATORY ASSESSMENTS

Venous blood samples were obtained in all patients at 7 AM on days 1, 2, and 3. Urine samples were obtained simultaneously in the last 10 patients. Blood samples were assessed for serum sodium, potassium, chloride, calcium, phosphorus, magnesium, creatinine, urea, albumin, and globulin, and acid base status was determined. Urine samples were assessed for osmolality, sodium, potassium, chloride, calcium, phosphorus, and creatinine. To estimate the glomerular filtration rate (GFR), the Cockcroft and Gault formula was used to calculate the creatinine clearance.¹⁷

Serum unbound calcium (Ca), corresponding to the ionized calcium, was calculated using the following formula:

$$\text{Serum Unbound Ca} = \text{Serum Ca} \times (100\% - \text{\%Serum Protein-bound Ca}),$$

whereas

$$\text{\%Serum Protein-bound Ca} = 0.8 \times \text{Albumin (g/L)} + 0.2 \times \text{Globulin (g/L)} + 3^{18}$$

Δ Phosphorus and Δ unbound Ca were calculated as the difference between their respective serum values on days 2 and 1. Urinary excretion of solutes was expressed as milliequivalent per milligram creatinine for sodium and potassium and as milligrams per milligram creatinine for phosphorus. Fractional excretion of solutes (potassium, sodium, and phosphorus) was calculated as

$$\frac{\text{Urine Solute (mg/dL or mEq/L)} \times \text{Serum Creatinine (mg/dL)}}{\text{Serum Solute (mg/dL or mEq/L)} \times \text{Urine Creatinine (mg/dL)}}$$

STATISTICAL ANALYSIS

Statistical analysis was performed by 1-way analysis of variance or *t* test to compare continuous variables, and the Pearson correlation test was used to assess the relationship between them. Association between 2 dichotomous variables was analyzed by the Fisher exact test. Results are expressed as mean \pm SD.

RESULTS

The patients' mean age was 80.5 \pm 6.17 years (range, 65-90 years). Twenty (56%) were women and 30 (83%) lived at home, while the other 6 lived in nursing homes. The functional and cognitive status, indications for colon investigation, major comorbidities, diuretics, and medications affecting intestinal motility are summarized in **Table 1**. No patient was treated with narcotics.

Serum and urinary parameters on days 1 and 2 are presented in **Table 2** and **Table 3**. Serum phosphorus levels increased by almost 100% between days 1 and 2, ranging from 2.6 to 4.7 mg/dL (0.84-1.52 mmol/L) to 4.5 to 10.4 mg/dL (1.45-3.36 mmol/L), respectively. Dur-

Table 1. Functional and Cognitive Groups, Indication for Colon Investigation, Coexistent Diseases, and Medications

Parameter	No. (%) of Patients
Functional group	
Dependent	9 (25)
Partially dependent	16 (44)
Independent	11 (31)
Cognitive group	
Severely demented	7 (19)
Moderately demented	9 (25)
Normal	20 (56)
Indications for colon investigation	
Anemia	19 (53)
Bleeding	8 (22)
Altered bowel habits	5 (14)
Weight loss	4 (11)
Major coexistent diseases	
Hypertension	15 (42)
Degenerative joint disease	15 (42)
Diabetes	14 (39)
Gastrointestinal	13 (36)
Cerebrovascular disease	12 (33)
Ischemic heart disease	11 (31)
Urologic	10 (28)
Congestive heart disease	8 (22)
Arrhythmias	7 (19)
Chronic obstructive lung disease	6 (17)
Parkinson disease	4 (11)
Oncology and hematology disease	4 (11)
Medication classes	
Diuretics	13 (36)
Calcium channel blockers	10 (28)
Benzodiazepines	10 (28)
Neuroleptics	6 (17)
Anticholinergics	3 (8)
Antiparkinsonian drugs	2 (6)

ing the same period, serum sodium and chloride levels increased, while serum calcium and potassium concentrations decreased. Serum calcium ranges were 8.3 to 10.2 mg/dL (2.08-2.55 mmol/L) on day 1 and 7.0 to 9.3 mg/dL (1.75-2.32 mmol/L) on day 2. Overall, 21 patients (58%) had a calcium concentration of 8.4 mg/dL (2.1 mmol/L) on day 2. Serum potassium concentration ranged from 3.61 to 5.21 mEq/L and from 2.45 to 4.95 mEq/L on days 1 and day 2, respectively. Twenty patients (56%) had a potassium concentration of 3.5 mEq/L or less on day 2, 5 (14%) of whom had a potassium concentration of 3 mEq/L or less (2 had severe diarrhea) necessitating appropriate treatment. Patients with a serum potassium concentration greater than 3.5 mEq/L on day 2 had a higher serum potassium concentration on day 1 compared with those with a serum potassium concentration of 3.5 mEq/L or less on day 2 (4.58 ± 0.32 mEq/L vs 4.34 ± 0.31 mEq/L, respectively; $P = .03$). There were no significant differences in serum potassium concentrations between patients treated with or without diuretics, neither on day 1 nor on day 2. Serum magnesium concentrations decreased on day 2, without statistical significance ($P = .08$). Serum urea, creatinine, and bicarbonate levels remained unchanged.

Urinary phosphorus excretion increased significantly on day 2. The urinary fractional excretion of phos-

Table 2. Serum Electrolyte Levels on Days 1 and 2

Parameter	Mean \pm SD		P Value*
	Day 1	Day 2	
Urea, mg/dL	43.4 \pm 22.7	42.5 \pm 20.0	NS
Creatinine, mg/dL	1.03 \pm 0.3	1.02 \pm 0.25	NS
Calcium, mg/dL	9.14 \pm 0.49	8.24 \pm 0.59	<.001
Unbound calcium, mg/dL	5.75 \pm 0.29	5.29 \pm 0.64	<.001
Phosphorus, mg/dL	3.55 \pm 0.50	6.97 \pm 1.48	<.001
Sodium, mEq/L	137.8 \pm 4.5	142.2 \pm 4.17	<.001
Potassium, mEq/L	4.45 \pm 0.34	3.51 \pm 0.54	<.001
Magnesium, mEq/L	2.13 \pm 0.33	2.03 \pm 0.33	.08
Chloride, mEq/L	102.5 \pm 4.7	104.2 \pm 4.0	.01
Albumin, g/dL	3.47 \pm 0.39	3.43 \pm 0.42	NS
Globulin, g/dL	3.09 \pm 0.52	3.09 \pm 0.55	NS
Bicarbonate, mEq/L	24.9 \pm 2.7	24.2 \pm 3.2	NS

Abbreviation: NS, not significant.

SI conversion factors: To convert calcium to millimoles per liter, multiply by 0.25; creatinine to micromoles per liter, multiply by 88.4; magnesium to millimoles per liter, multiply by 0.50; phosphorus to millimoles per liter, multiply by 0.3229.

*Between day 1 and day 2.

Table 3. Urinary Parameters on Days 1 and 2

Parameter	Mean \pm SD		P Value*
	Day 1	Day 2	
Urine osmolality, mOsm/kg	411 \pm 123	497 \pm 105	.02
Urine phosphorus, mg/mg creatinine	0.68 \pm 0.32	4.96 \pm 1.5	<.001
Urine potassium, mEq/mg creatinine	0.089 \pm 0.08	0.056 \pm 0.02	NS
Urine sodium, mEq/mg creatinine	0.21 \pm 0.11	0.23 \pm 0.07	NS
Fractional excretion phosphorus, %	24.7 \pm 20.3	61.2 \pm 15.3	.01
Fractional excretion potassium, %	13.9 \pm 6.3	16.8 \pm 7.4	NS
Fractional excretion sodium, %	1.37 \pm 0.77	1.39 \pm 0.51	NS

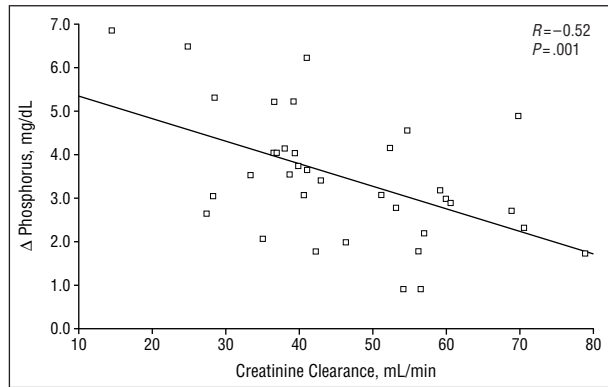
Abbreviation: NS, not significant.

*Between day 1 and day 2.

phorus increased significantly and ranged from 10.1% to 66.2% on day 1 to 39.5% to 87% on day 2 ($P = .01$). Urinary excretion and fractional excretion of sodium and potassium remained unchanged (**Table 3**).

The mean creatinine clearance on day 1 was 46.1 ± 14.2 mL/min (0.77 ± 0.24 mL/s) with a wide range of 15.2 to 78.6 mL/min (0.25-1.31 mL/s). Creatinine clearance on day 1 was correlated with Δ phosphorus ($R = -0.52$; $P = .001$) (**Figure**), and with Δ unbound calcium ($R = -0.32$; $P = .06$). No correlation was found between creatinine clearance on day 1 and the fractional excretion of phosphorus on days 2 or 3 and between Δ phosphorus and Δ unbound calcium. All parameters studied reverted to near normal values on day 3, although the fractional excretion of phosphorus remained relatively high but statistically insignificant.

There were more demented patients in the group with a serum potassium concentration of 3.5 mEq/L or less on day 2 (60%) than in the group with serum potassium concentrations greater than 3.5 mEq/L (25%)



Correlation between creatinine clearance on day 1 and Δ phosphorus level.

($P < .05$). Only 2 of those with a serum potassium concentration of 3 mEq/L or less on day 2 were prescribed loop diuretic treatment. All were partially or fully dependent, and only 1 of them was cognitively normal. Analysis of other serum and urinary electrolyte parameters, Δ phosphorus, or Δ calcium did not demonstrate any significant differences between cognitive or functional groups nor an association with the coexistent diseases or with medications.

COMMENT

The cathartic action of sodium phosphate, a small volume saline laxative, results largely from its osmotic properties, drawing plasma water into the gastrointestinal tract. This study demonstrates that sodium phosphate induced mainly a marked and transient increase in serum phosphorus, sodium, and chloride levels with a concomitant decrease in serum calcium and potassium concentrations. These statistically significant changes may be even an underestimation, since the second blood sample was drawn only 12 hours after the second dose of sodium phosphate. This probably explains why we could not demonstrate any change in bicarbonate level, as did Cohen et al⁵ who found that serum bicarbonate level decreased 6 hours after administration of the second dose of sodium phosphate.

The phosphorus concentration of the extracellular fluid is regulated mainly by 2 factors: the phosphorus load and its renal excretion. The phosphorus load depends on the ingested amount and on the fractional intestinal absorption. Renal phosphorus excretion depends on the filtered load ($\text{GFR} \times \text{plasma phosphorus}$) and on the renal threshold (TmP/GFR), where TmP is the limiting rate of tubular phosphorus reabsorption.^{19,20}

Phosphorus is absorbed principally in the jejunum and duodenum by both passive and active transport under the influence of vitamin D.²¹ The net phosphorus absorption increases linearly with its intake in normal subjects.²² Serum phosphorus is freely filtered in the kidney; 80% to 90% is reabsorbed by the renal tubules, of which 70% occurs in the proximal convoluted tubule and about 10% in the distal convoluted tubule. Proximal tubular reabsorption occurs via sodium-phosphate cotransporters, mainly type 2. This cotransporter is regulated by phos-

phorus delivery and parathyroid hormone (PTH).²³ In patients with normal GFR, an increase in serum phosphorus level is associated with an increase in urinary phosphorus excretion due to an increase in its filtered load and to PTH-mediated inhibition of the proximal tubule sodium-phosphate cotransporter. When the GFR is decreased, the phosphaturic effect is blunted and acute hyperphosphatemia may develop. In effect, we have found a fair negative correlation between the baseline creatinine clearance and Δ phosphorus ($R = -0.52$) (Figure 1), in parallel with a 3-fold increase in the fractional excretion of phosphorus. This increase in phosphorus renal excretion reflects the effect of PTH secretion increase, as has been shown previously.²⁴

Debate exists as to the accuracy of GFR measurement using the Cockcroft and Gault formula in the elderly.²⁵ A recent study of healthy elderly subjects found it to be too inaccurate for clinical use.²⁶ This could explain in part why variations in GFR account for only about 27% of the phosphorus change ($R^2 = 0.27$). However, other important factors should be taken into consideration such as variability in intestinal phosphorus absorption (which is itself affected by different factors, such as intestinal transit time and serum vitamin D), volume of distribution, and serum PTH, which inversely increases when GFR decreases.

The patients' renal function in the present study varied markedly from normal to severe renal insufficiency. A decrease in GFR is associated with low serum level of the active vitamin D metabolite, 1,25-dihydroxyvitamin D₃, resulting in a decreased phosphorus absorption. Thus, despite a similar phosphorus load, the net absorption is expected to vary markedly in this population.

Concomitant significant hypocalcemia (total and unbound) accompanied the hyperphosphatemia on day 2, mainly due to the deposition of unbound calcium in the vascular bed and soft tissues. However, since the hyperphosphatemia was moderate and transient, this process had a negligible, if any, effect. DiPalma et al⁹ confirmed the physiologic consequences of these electrolyte variations by measuring rises in PTH and urinary cyclic adenosine monophosphate.

While calcium-phosphorus variations were predictable, the magnitude of the hypokalemia and the number of patients affected were not. Vanner et al³ reported that 28% of their patients had a serum potassium concentration below 3.5 mEq/L after administration of sodium phosphate, with a lowest value of 2.9 mEq/L (56% and 2.45 mEq/L in our study, respectively). Clarkston et al⁷ reported a serum potassium concentration below 3.5 mEq/L in 20% of their patients. Lieberman et al²⁷ reported that the lowest serum potassium value secondary to sodium phosphate administration was 3.2 mEq/L.

We can hypothesize that since blood samples were drawn up to 6 hours after the second dose of sodium phosphate in these 3 previous studies, the maximal cathartic effect was not attained. Therefore, the patients in these studies^{3,7,27} did not reach the potassium nadir, as did ours. Moreover, the patients' mean age was 57 to 62.3 years in these previous studies,^{3,7,27} and therefore the cathartic effect might be less aggressive than in the older popu-

lation. Indeed, our patients were significantly older and frailer, and hypokalemia was more prevalent in the demented and dependent patients.

One can argue that patients treated with diuretics, mainly furosemide and thiazides, are more prone to develop hypokalemia. However, in our study, patients treated with or without diuretics had the same serum potassium concentration on days 1 and 2. Nevertheless, patients whose serum potassium concentration was 3.5 mEq/L or less on day 2 had a lower baseline serum potassium concentration on day 1 compared with patients with a serum potassium concentration above 3.5 mEq/L on day 2. This finding may support a relative potassium depletion state in some patients, since the serum level poorly reflects the total body potassium concentration, unmasked by the osmotic effect of sodium phosphate. Hill et al²⁸ agree with this assumption, reporting a fall in serum potassium concentration during a period of oral sodium phosphate administration, negatively correlating with intracellular potassium concentration measured prior to administration.

The hypokalemia observed on day 2 should have induced a decrease in fractional excretion of urine potassium. However, this did not occur. Different factors may have caused impaired renal potassium conservation. The intravascular space was probably moderately contracted, as previously described,^{3,4,29} which is suggested by the presence of hypernatremia. This dehydrated state should have stimulated aldosterone release, raising potassium secretion by the principal cells in the distal nephron. In addition to this mechanism, the osmotic diuresis and enhanced luminal electronegativity created by distal sodium reabsorption in the presence of a highly impermeable anion (phosphate) enhanced potassium secretion.³⁰ The distal delivery of large quantities of various nonreabsorbable anions has been shown to cause hypokalemia in humans: bicarbonate in metabolic alkalosis³¹ and in type II renal tubular acidosis,³² β -hydroxybutyrate in diabetic ketoacidosis,³³ hippurate with toluene use,³⁴ and a penicillin derivative during high-dose penicillin therapy.^{35,36} In our study, the markedly increased urinary phosphate excretion in the presence of an unchanged urinary sodium excretion suggests that this mechanism may have played a role in impairing renal potassium sparing. The severity of the hypokalemia could thus be explained by the combined effect of intestinal loss and an impaired ability of the kidney to conserve potassium. To our knowledge, this decreased ability of the kidney to retain potassium following an acute phosphate load has not been previously demonstrated.

Hypokalemia is disturbing, especially when combined with hypocalcemia,³⁷ although no patient had severe complications. Clarkston et al⁷ monitored cardiac arrhythmia before and during colon preparation, and during colonoscopy with sodium phosphate vs sulfate-free polyethylene glycol. They did not observe an increase in arrhythmias before or during colon preparation or during colonoscopy in either patient group. Although not statistically different from arrhythmia before preparation, there were 4 patients in the sodium phosphate group who had ventricular tachycardia during preparation. All 4 had a history of heart disease and 3 of them had hy-

pokalemia (potassium range, 3.1-3.3 mEq/L) after preparation. Marsh et al³⁸ concluded in a small study that colonic lavage with polyethylene glycol was associated with increased ventricular ectopy. Gupta et al³⁹ reported cardiac arrhythmia and ST-T changes in 20.6% of patients undergoing upper and lower gastrointestinal endoscopy. The incidence of these changes was higher in patients with cardiac or pulmonary disease. A more recent and larger study demonstrated a relatively low intraprocedural colonoscopic complication rate of arrhythmia (0.1%), bradycardia (0.8%), and hypotension (1.2%).⁴⁰

CONCLUSIONS

This study demonstrates important and serious electrolyte abnormalities in elderly patients undergoing colon preparation with sodium phosphate. The role of the kidney in the pathogenesis of hypokalemia following the use of sodium phosphate has been previously underestimated. Creatinine clearance should be calculated to estimate the magnitude of calcium-phosphorus variations, albeit other intervening factors exist. The frequency and severity of the hypokalemia is of serious concern, especially in the presence of hypocalcemia and preexistent cardiovascular diseases. It is apparently more prevalent in dependent and cognitively impaired patients. Assessment of serum creatinine, electrolytes, calcium, and phosphorus prior to sodium phosphate preparation in elderly patients is advised, and in selected cases post-procedural electrolyte assessment and correction may be required.

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Corresponding author: Yichayaou Beloosesky, MD, Department of Geriatrics, Rabin Medical Center, Beilinson Campus, Petach Tikva 49100, Israel (e-mail: belo-7@zahav.net.il).

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