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A New Graduated Dosing Regimen for Phosphorus Replacement in Patients Receiving Nutrition Support

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ABSTRACT. *Background:* Hypophosphatemia is a common metabolic complication in patients receiving specialized nutrition support. We changed our previously reported dosing algorithm because the low dose no longer appeared to be effective at increasing serum phosphorus concentrations. The purpose of this study was to evaluate the safety and efficacy of a revised weight-based phosphorus-dosing algorithm in critically ill trauma patients receiving specialized nutrition support. *Methods:* Seventy-nine adult trauma patients with hypophosphatemia (serum phosphorus concentration ≤0.96 mmol/L) receiving nutrition support received an IV dose of phosphorus on day 1 according to the serum concentration of phosphorus: 0.73–0.96 mmol/L (0.32 mmol/kg, low dose), 0.51–0.72 mmol/L (0.64 mmol/kg, moderate dose), and ≤0.5 mmol/L (1 mmol/kg, high dose). The IV phosphorus bolus dose was administered at 7.5 mmol/hour. Generally, patients with a serum potassium concentration <4 mmol/L received potassium phosphate and patients with a serum potassium concentration ≥4 mmol/L received sodium phosphate. Patients who still had hypophosphatemia on day 2 were dosed using the new dosing algorithm by the nutrition support service according to that day's serum concentration of phosphorus, or empirically by the trauma service. *Results:* Of the 79 patients studied, 57 were male and 22 were female with a mean age of 44.8 ± 20.6 years. Mean Injury Severity Scores and APACHE-II scores were 27.1 ± 11.6 and 15.2 ± 6.8, respectively. There was no difference in baseline characteristics among the 3 dosing groups. Of the 79 patients, 34 received the low dose, 30 received the moderate dose, and 15 received the high dose of phosphorous. Mean serum phosphorous concentrations on day 2 were significantly increased in the moderate-dosed group (0.64 ± 0.06 to 0.77 ± 0.22 mmol/L, $p < .05$) and high-dosed group (0.38 ± 0.06 to 0.93 ± 0.32 mmol/L, $p < .01$), respectively, when compared with day 1. Mean serum phosphorus concentrations were normal in all 3 groups on day 3. Serum concentrations of magnesium, sodium, and potassium, as well as arterial pH, were stable across the study. Mean concentrations of ionized calcium were not significantly different in any of the 3 dosing groups across the study period. *Conclusions:* This weight-based phosphorus-dosing algorithm is safe for use in critically ill patients receiving nutrition support. The moderate- and severe-dose regimens effectively increase serum phosphorus concentrations. (*Journal of Parenteral and Enteral Nutrition* **30:**209–214, 2006)

Hypophosphatemia is a common occurrence in critically ill patients that can result in serious complications, given the important role phosphorus plays in normal physiology. Hypophosphatemia is encountered in hospitalized medical and surgical patients but may be even more prevalent in patients receiving nutrition support.[1–4] This metabolic complication can occur as the result of phosphorus depletion or due to a shifting of phosphorus to the intracellular compartment. Depletion of total body phosphorus can be encountered in nutritionally wasted patients, such as those with cancer cachexia, HIV infection, and patients with chronic alcohol consumption.[5] In nondepleted patients, risk factors for the development of hypophosphatemia can include glucose infusion with or without aggressive refeeding,[3,6–8] medications,[9,10] trauma,[11] severe head injury,[12] thermal injury,[13,14] and sepsis.[3] In recent years, several studies have been published that detail the negative effects of hypophosphatemia and emphasize the importance of maintaining normal serum phosphorus concentrations. Depending on the degree of hypophosphatemia, manifestations can include leukocyte dysfunction,[6] rhabdomyolysis,[15] glucose intolerance,[16] decreased diaphragmatic contractility,[17] arrythmias,[18] reduced cardiac output,[4,19,20] and even death.[21]

We previously reported a weight-based phosphorus-repletion regimen for hypophosphatemic patients receiving nutrition support.[22] Recently, we reevaluated this algorithm internally as we had noticed an ineffectiveness of the low dose (0.16 mmol/kg) used in our previous study. This necessitated the development

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of a more aggressive dosing regimen. The purpose of this study was to assess the safety and efficacy of this revised algorithm in critically ill trauma patients receiving nutrition support.

MATERIALS AND METHODS

This retrospective, observational study was approved by the University of Tennessee Health Science Center institutional review board, and the need for informed consent was waived. Adult trauma patients >18 years old who were hospitalized at the Regional Medical Center at Memphis were included in the study. Study patients resided in the Trauma Intensive Care Unit (Trauma ICU), General ICU, Neurotrauma ICU, or the Trauma Stepdown Unit and were followed by the Nutrition Support Service (NSS) during data collection. Any subject receiving nutrition support *via* enteral or parenteral routes who also had a serum phosphorus concentration ≤0.96 mmol/L was entered in the study.

Patients with acute renal failure, chronic kidney disease (calculated creatinine clearance <30 mL/min[23], hypercalcemia [serum ionized calcium >1.32 mmol/L], or hypocalcemia [serum ionized calcium <1.12 mmol/L]) were excluded. Other patient exclusions included a history of parathyroid hormone disease, metabolic bone disease, or class III obesity (body mass index >40 kg/m^2).

Patients admitted to the above units had daily blood drawn at 3 AM, which was sent for a basic metabolic panel with magnesium, ionized calcium, and phosphorus, and a complete blood count with differential. According to initial serum phosphorus concentrations, patients were placed into one of 3 categories. The normal range for serum phosphorus concentrations at our institution is 0.8–1.44 mmol/L (2.5–4.5 mg/dL). Those who had a serum phosphorus concentration of 0.73–0.96 mmol/L (2.3–3 mg/dL) were empirically designated as having mild hypophosphatemia; those with a serum concentration of 0.51–0.72 mmol/L (1.6–2.2 mg/dL) were assigned to the moderate hypophosphatemic group; and those with a value of <0.5 mmol/L (≤1.5 mg/dL) were placed in the severe hypophosphatemic group, as previously described.[22] Phosphorus laboratory tests were determined by a colorimetric phosphorus molybdate reaction in the presence of sulfuric acid. The patients were then assigned to an IV phosphorus bolus according to their assigned group as follows: mild hypophosphatemia (0.32 mmol/kg, low dose), moderate hypophosphatemia (0.64 mmol/kg, moderate dose), and severe hypophosphatemia (1 mmol/kg, high dose). For ease of preparation, phosphorus doses were rounded to the nearest 7.5 mmol. Doses were calculated according to actual body weight for subjects weighing <130% of their ideal body weight (IBW). In subjects who exceeded 130% of IBW and had a body mass index <40 kg/m^2, an adjusted body weight was used with the following equation [IBW + 0.25(actual body weight − IBW)]. Patients with a serum potassium concentration <4 mmol/L on study day 1 received potassium phosphate, whereas subjects with a serum potassium concentration ≥4 mmol/L received sodium phosphorus. Due to the amount of phosphorus required in select subjects, especially in the severe group, a combination of the 2 salt forms of phosphorus was occasionally used (eg, mild hypokalemia with severe hypophosphatemia). Doses of phosphorus were diluted in 100 mL (mild and moderate groups) or 250 mL (severe group) of normal saline (NS) or 5% dextrose in water, and given intravenously at a rate not to exceed 7.5 mmol phosphorus/hour. Phosphorus infusions were given in the morning after calculation and preparation of the respective phosphorus dose.

The phosphorus infusions were given in addition to the phosphorus provided in patients' nutrition support formulation. The only enteral formulas used in the study were Isosource VHN (26 mmol phosphorus/L), Impact Glutamine (39 mmol phosphorous/L), and Resource Diabetic (34 mmol phosphorous/L; all formulas by Novartis Medical Nutrition, Minneapolis, MN). Most patients enrolled were receiving either enteral formulas without additional phosphorus or parenteral nutrition (PN) containing only standard amounts of phosphorus (15 mmol/L).

Data for the 2 days after the phosphorus infusion were collected. Patients who required phosphorus replacement on the subsequent day were dosed by the NSS according to the algorithm or by the primary trauma team. On day 2, patients generally had the phosphorus content of their nutrition regimen increased if they still had hypophosphatemia. This could be accomplished by increasing the phosphorus in the parenteral formulation or by adding injectable potassium phosphate or sodium phosphate (Fleet Phosphasoda, C.B. Fleet Company, Lynchburg, VA) to enteral formulations.

All interval data are reported as means ± SD. Data analysis was conducted using SPSS for Windows, version 12 (SPSS, Inc, Chicago, IL) or SigmaStat for Windows, version 3.1 (Systat Software, Inc, Point Richmond, CA). Continuous or interval data were analyzed by 1-way analysis of variance with post hoc pairwise comparisons between groups determined using Tukey's honestly significant difference test. For data expressing the same variable measured on multiple occasions over time, repeated-measures analysis of variance (RMANOVA) was performed to detect differences in these measurements between the 2 populations. The populations were tested for sphericity, and then the univariate RMANOVA was conducted if the assumption was correct. If the sphericity assumption was rejected, then the multivariate RMANOVA was performed. The significance testing and reported p values were 2 sided for all variables. A p value <0.05 was considered statistically significant.

RESULTS

We identified 79 patients during a 5-month period who met entrance criteria. Of these 79 patients, there were 34 in the mild group, 30 in the moderate group, and 15 in the severe group. Demographic data including age, gender, height, weight, admission diagnosis, Injury Severity Score (ISS), Modified Trauma Score, and APACHE II score are presented in Table I. There

TABLE I
*Demographics of the 79 patients included in the study**

Gender (M/F)	57/22
Age (y)	44.8 ± 20.6
Height (cm)	172 ± 11
Weight (kg)	76.3 ± 14.9
Injury Severity Score (ISS)	27.1 ± 11.6
Modified Trauma Score	9.6 ± 3.4
APACHE II score	15.2 ± 6.8
Closed head injury (No./%)	36/46
Mechanical ventilation (No./%)	63/80

*The data are presented as mean ± SD.

was no statistical difference in any baseline characteristic between the 3 hypophosphatemic groups. Admitting diagnoses included motor vehicle crash (61%), gunshot wound (11.4%), falls (10.1%), miscellaneous trauma (6.3%), pedestrian struck (6%), knife stab wound (2.5%), and assault (2.5%). Seventy-four patients received only enteral nutrition, 2 received PN only, and 3 patients received both parenteral and enteral nutrition during the study. The majority of patients were studied early in their hospital stay, most within the first week after admission.

The mean serum phosphorus concentration for all patients increased significantly at study days 2 and 3. Mean phosphorus values for all patients were 0.67 ± 0.19 mmol/L, 0.83 ± 0.26 mmol/L, and 0.93 ± 0.32 mmol/L for days 1, 2, and 3, respectively. When separated by dosing groups, the mild group experienced a slight increase in serum phosphorus concentrations from day 1 to 2, but the increase was not statistically significant (Table II). In contrast, the graduated dosing algorithm produced a statistically significant increase in serum phosphorus concentration from day 1 to 2 in both the moderate and severe hypophosphatemic groups (Table II). Serum phosphorus concentrations on day 3 were available in 77/79 patients. Both the mild and moderate groups continued to increase from day 2 to 3, whereas the severe group decreased slightly. On day 3, serum phosphorus concentrations in the moderate and severe groups remained statistically increased compared with day 1. All 3 groups had mean serum phosphorus concentrations within the normal range by day 3. The change in serum phosphorus concentration from day 1 to day 2 in the respective dosing groups is depicted in Figure 1. The change in the 1 mmol/kg dosing group was significantly greater than either of the other 2 groups (Figure 1).

The number of patients who required an additional bolus on day 2 was 24 (71%), 24 (80%), and 11 (73%) for the mild, moderate, and severe groups, respectively. Seventeen patients in the mild group, 12 patients in the moderate group, and 11 patients in the severe group were also treated by increasing the phosphorus content of their nutrition support formulation on day 2. In the mild group, 59% of patients had serum phosphorus concentrations within the normal range on day 2, and 59% were normal on day 3. Fifty percent of the moderate group had serum phosphorus concentrations within the normal range on day 2, which increased to 70% by day 3. Fifty-three percent of patients in the severe group were within the normal range on day 2, and it increased to 60% on day 3. Phosphorus doses were given as potassium phosphate in 42/79 (53%) patients, as sodium phosphate in 18/79 (23%) patients, and as a combination of the 2 salts in 19/79 (24%) patients. Mean doses of phosphorus on day 1 for the mild, moderate, and severe groups were 27.4 ± 5.2 mmol, 49.1 ± 9.7 mmol, and 75 ± 12 mmol, respectively.

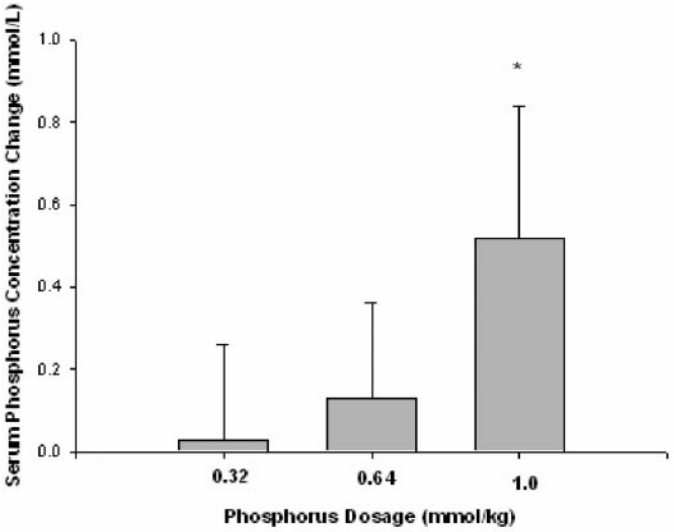

FIGURE 1. Mean change in serum phosphorus concentration (mmol/L) from day 1 to day 2 by dosing group. *The change in serum phosphorus concentration in the group receiving 1 mmol/kg was statistically greater than the changes with either of the other 2 doses.

Changes in serum concentrations of other electrolytes between days 1 and day 2 are depicted in Table III. Serum ionized calcium concentrations were available in 75 patients on day 1 and 76 patients on day 2.

TABLE II
Mean serum phosphorus concentrations (mmol/L) by dosing group

	Day 1	Day 2	Day 3
Mild	0.83 ± 0.06	0.86 ± 0.22	0.96 ± 0.35
Moderate	0.64 ± 0.06	0.77 ± 0.22*	0.93 ± 0.29†
Severe	0.38 ± 0.06	0.93 ± 0.32†	0.86 ± 0.26†

*$p < .05$ compared to day 1; †$p < .001$ compared to day 1.

TABLE III
*Mean (± SD) serum electrolyte concentrations for the mild, moderate, and severe hypophosphatemic groups for days 1 and 2**

	Mild	Moderate	Severe
Potassium 1 (mmol/L)	3.7 ± 0.3	3.8 ± 0.3	3.5 ± 0.5
Potassium 2	3.7 ± 0.3	3.6 ± 0.3	3.6 ± 0.4
Sodium 1 (mmol/L)	141 ± 6	143 ± 6	141 ± 7
Sodium 2	141 ± 6	145 ± 8	144 ± 7
Ionized Ca 1 (mmol/L)	1.21 ± 0.05	1.21 ± 0.05	1.21 ± 0.06
Ionized Ca 2	1.21 ± 0.05	1.21 ± 0.07	1.20 ± 0.06
Magnesium 1 (mmol/L)	0.82 ± 0.12	0.9 ± 0.12	0.86 ± 0.12
Magnesium 2	0.86 ± 0.08	0.86 ± 0.12	0.98 ± 0.57
Creatinine 1 (micrommol/L)	70.7 ± 17.7	70.7 ± 17.7	79.6 ± 17.7
Creatinine 2	70.7 ± 17.7	70.7 ± 17.7	70.7 ± 26.5
Urea Nitrogen 1 (mmol/L)	3.3 ± 1.9	4.1 ± 2.8	3.1 ± 1.4
Urea Nitrogen 2	3.5 ± 2.1	4.5 ± 2.6	3.4 ± 1.4

*Divide serum magnesium, creatinine, and urea nitrogen concentrations by 0.41, 88.4, and 0.36, respectively, to convert to mg/dL.

There was no statistically significant change in ionized calcium between days 1 and 2 in the study population (p = NS). Four of the 79 study patients (5%) became hypocalcemic (ionized calcium <1.12 mmol/L), though none were symptomatic. These 4 patients all received the moderate dose of phosphorous for hypophosphatemia. Serum potassium concentrations were clinically unchanged in all 3 groups from day 1 to 2, as was sodium, creatinine, and serum urea nitrogen. The serum magnesium concentrations were clinically unchanged in the mild- and moderate-dosed group. In the severe-hypophosphatemic group, the mean serum magnesium concentration increased from day 1 to 2, though the change was not statistically significant. Patients with hypomagnesemia were treated with IV magnesium sulfate.

Over the 3 study days and 235 phosphorus values collected, only 8 serum phosphorus concentrations were above our normal range of 0.8–1.44 mmol/L. Two patients in the severe group had serum phosphorus concentrations on day 2 of 1.57 and 1.63 mmol/L. The day 2 serum phosphorus concentrations had decreased by day 3 to 0.74 and 0.96 mmol/L, respectively. These data suggest that in patients requiring aggressive treatment, mild hyperphosphatemia after this infusion is not sustained. On day 3, 6 patients were above our normal range. One patient had received only 1 dose (mild) on day 1 and continued to increase throughout the study. Two of the other 5 patients had received aggressive doses on day 2 that were greater than the algorithm would have provided. The other 3 patients had received appropriate boluses of phosphorus on day 2. None of these patients were symptomatic or had any complications as a result of their transient hyperphosphatemia.

DISCUSSION

This is a follow-up to our previous study that demonstrated that phosphorus doses of 0.16, 0.32, and 0.64 mmol/kg resulted in increases in the serum phosphorus concentrations of 0.22, 0.26, and 0.32 mmol/L, respectively.[22] After we noticed a lack of effect with the low dose (0.16 mmol/kg) from our original algorithm,[22] this dose was abandoned. We then increased the doses for phosphorus replacement given to each hypophosphatemic group. The maximum dose of phosphorus of 1 mmol/kg was decided upon according to earlier work we published in thermally injured patients who had hypophosphatemia despite receiving approximately 1 mmol/kg over a 24-hour period.[14] Because our original dosing algorithm appeared in some book chapters and clinical guidelines, we felt obligated to study and report the safety and efficacy of the new dosing regimen.

The use of larger doses of phosphorus in critically ill patients has steadily evolved. Vannatta et al[24] were one of the first groups of investigators to document aggressive phosphorus repletion. They administered 9 mmol of potassium phosphate over 12 hours to 10 patients with severe hypophosphatemia (<0.32 mmol/L). In a follow-up study by this same group in 10 patients with serum phosphorus concentrations <0.32 mmol/L, patients were administered 0.32 mmol/kg of phosphorus (as potassium phosphate) over 12 hours.[25] All patients were >0.64 mmol/L by 48 hours; however, serum phosphorus concentrations were below the normal range in 40% of patients. This study was the first to demonstrate the efficacy of a weight-based dosing regimen with a rapid rate of repletion.

Two studies were later conducted to evaluate the effects of phosphorus doses as rapid infusions in critically ill patients.[4,19] Both groups used doses of glucose-1-phosphate over 30 or 60 minutes.[4,19] These investigators documented a significant increase in myocardial function and observed no adverse effects. It is important to note that the above 2 studies were not designed to show efficacy of replacement or safety, but rather the effects of phosphate repletion on myocardial function. None of these patients were receiving nutrition support during phosphorus therapy.

Our original study demonstrated safety and efficacy using a graduated dosing algorithm of phosphorus according to serum phosphorus concentrations and body weight in patients receiving specialized nutrition support.[22] Patients were enrolled into one of 3 groups: mild hypophosphatemia (0.73–0.96 mmol/L), moderate hypophosphatemia (0.51–0.72 mmol/L), or severe hypophosphatemia (≤0.5 mmol/L). Subjects were dosed intravenously as follows: mild (0.16 mmol/kg), moderate (0.32 mmol/kg), severe (0.64 mmol/kg). Patients were followed for 2 days after the infusion and rebolused as needed on day 2. Similar to the current study, phosphorus was infused at a rate of 7.5 mmol/hour. After 24 hours, 81% of patients in the mild group, 68% of the moderate group, and 21% of the severe group had serum phosphorus concentrations within the normal range. There were no significant changes in serum concentrations of total calcium, urea nitrogen, or creatinine. This study validated the use of a graduated weight-based approach to phosphorus supplementation and documented the safety of using doses that were higher than those used in previous studies.

Recently, a group of investigators studied the effects of a more rapid repletion of phosphorus in 47 medical/surgical ICU patients with moderate (<0.64 mmol/L) to severe (<0.4 mmol/L) hypophosphatemia.[26] Patients in the moderate group were randomized to 30 mmol of IV phosphate over 2 or 4 hours, whereas patients in the severe group were randomized to 45 mmol of phosphate over 3 or 6 hours (15 mmol/hour *vs* 7.5 mmol/hour). All patients received potassium phosphate. At the end of the infusion, 98% of the patients had a phosphorus concentration >0.64 mmol/L. There was, however, no statistical difference in end-of-infusion serum phosphorus concentrations between those who received slow infusions *vs* faster infusions. All groups had serum phosphorus concentrations above their baseline at 24 hours, though no statistical significance was reported. When compared with the slower infusion groups, more patients in the rapid infusion groups experienced hyperkalemia. In addition, urinary fractional excretion of phosphorus was increased in those patients who received phosphorus more rapidly. These data suggest that giving doses of phosphorus rapidly (15 mmol/hour) may exceed the renal threshold

for this mineral, resulting in a higher percentage of the dose being lost in the urine. These data also suggest that the use of potassium phosphate should be restricted for patients with hypokalemia or a low-normal serum concentration of potassium.

Taylor and colleagues[27] published the results of a weight-based phosphorus-dosing protocol in surgical intensive care patients. This protocol used a single dose of phosphorus in an attempt to alleviate the need for repeated dosing as had been documented in most previously conducted studies. Patients were divided into 3 dosing categories according to serum phosphorus concentrations (<0.32 mmol/L, 0.32–0.55 mmol/L, and 0.56–0.7 mmol/L) and prescribed doses based on these concentrations and body weight (40–60 kg, 61–80 kg, and 81–120kg). All doses, ranging from 10 to 50 mmol, were given over 6 hours. Any patient who did not have a 18- to 24-hour postrepletion phosphorus concentration >0.74 mmol/L or who required additional phosphorus supplementation at any point during their ICU stay, regardless of postrepletion concentration, was deemed a treatment failure. In the 111 patients studied in the prospective arm, the success rates of this protocol were 78% in moderate hypophosphatemia (defined as 0.51–0.7 mmol/L) and 63% in severe hypophosphatemia (defined as ≤0.5 mmol/L). These success rates are higher than those of previously reported studies. It is not clear why these results were obtained in this surgical intensive care population. The number of patients in this study who received nutrition support was not reported. The dosing scheme used in this protocol contained 9 different dosing categories, making it more cumbersome than other previously reported protocols. It is also unclear why the investigators used 3 categories of serum concentrations for dosing and then reported results in just 2 categories that were different from the dosing groups. In addition, Taylor and colleagues[27] did not address patients with serum phosphorus concentrations >0.7 mmol/L, citing these patients as being within their institution's normal range.

With respect to electrolytes other than phosphorus, our dosing algorithm had minimal adverse effects. Large phosphorus doses have been documented to decrease calcium concentrations[28]; however, in our study, mean ionized calcium concentrations were unchanged between days 1 to 2. Most previous studies have used total calcium concentrations to assess safety. It is well known that total calcium concentrations are not accurate measures of calcium balance in critically ill patients, even when adjusted for a depressed serum albumin concentration.[29] Despite 77% of subjects receiving some or all potassium phosphate, mean serum potassium concentrations were unchanged in the mild group, slightly decreased in the moderate group, and slightly increased in the severe group. None of these changes were statistically significant and point to the difficulty in regulating potassium balance in this type of patient. Serum magnesium concentrations were statistically unaffected; however, the increase in serum magnesium concentrations was greatest in the severe hypophosphatemia group. Those patients who had hypomagnesemia did receive IV magnesium sulfate replacement.

For reasons that are not entirely clear to us, the results of the current study differ from those of our previous study. The changes in serum phosphorus concentration produced from the common doses (0.32 and 0.64 mmol/kg) were greater in the original study than we observed in the current protocol. This occurred despite our elimination of the low dose and escalation of all doses for the respective serum phosphorus concentrations. We anticipated success of the high dose (1 mmol/L), but the ineffectiveness of the low dose (0.32 mmol/kg) was unexpected. It is likely that the patient acuity level of our ICU population was higher than the original population studied in 1995. All patients in the current study were trauma patients hospitalized in the ICU, whereas the patient population in the original study included some patients in step-down beds. We also report a large number of patients with closed head injuries in this current study. This may have affected the overall success of the dosing algorithm as patients with traumatic brain injury are at increased risk of developing hypophosphatemia.[12]

The only patients we used in this study were those in whom clinicians used the current dosing algorithm. We feel that these patients more closely represent the types of patients encountered by most NSS and help to validate the use of these doses in patients who are receiving extra phosphorus as part of their nutrition formulation. It is important to note that the majority of these patients were studied early in their nutrition course (many on the day of NSS consult), and the amount of phosphorus received from the nutrition formulation was likely quite low. Because the amount of nutrition received was generally very low, we did not record caloric intake or percent of goal received at the time of phosphorus dosing. Most of the patients in this study were receiving exclusively enteral nutrition. We believe this to be an accurate reflection of our practice, given our aggressive use of early enteral nutrition, especially in trauma patients.[30]

CONCLUSIONS

The results of this study verify the safety and efficacy of a weight-based phosphorus dosing algorithm in patients receiving specialized nutrition support. This algorithm is more aggressive than other published dosing regimens and uses a higher dose of phosphorus than previously documented, without compromising safety. Serum phosphorus concentrations increased significantly in the moderate and severe groups after 1 dose. All groups had a mean serum phosphorus concentration within our institution's normal range by day 3. No adverse events were encountered, and other serum electrolytes were not negatively affected. Although this study included only trauma patients, this nomogram is currently used by our NSS for the treatment of hypophosphatemia in a variety of critically ill patients.

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