Phosphate Deposition Capacity of Athletes During Hypokinesia, Phosphate Loading, and Ambulation

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ABSTRACT

Hypokinesia (diminished movement) induces significant phosphate (P) excretion; however, little is known about the P deposition ability of the body during hypokinesia (HK). Using P loads, the aim of this study was to establish the deposition ability of the body to retain P during prolonged HK. Studies were done during a 30-d period of pre-HK and a 364-d period of HK. Forty male trained athletes aged 24.7 ± 8.0 yr were chosen as subjects. They were equally divided into four groups: unloaded ambulatory control subjects (UACS), unloaded hypokinetic subjects (UHKS), loaded ambulatory control subjects (LACS), and loaded hypokinetic subjects (LHKS). All hypokinetic subjects were limited to an average walking distance of 0.7 km/d. Loading tests with 85.0 mg of calcium phosphate/kg body weight were performed on the LACS and LHKS.

Fecal P loss, urinary calcium (Ca) and P loss, serum P, Ca, and the ionized calcium (Ca) levels increased significantly (p ≤ 0.05) in the LHKS and UHKS groups when compared with the LACS and UACS groups, respectively. Serum intact parathyroid hormone (iPTH) and the 1,25-dihydroxyvitamin D3 [1,25-(OH)2 D3] levels decreased significantly (p ≤ 0.05) in the LHKS and UHKS groups when compared with the LACS and UACS groups, respectively. After the P load, significant (p ≤ 0.05) differences were observed between LHKS and UHKS groups regarding serum, urinary, and fecal P changes. Thus, the deposition capacity of P decreased significantly (p ≤ 0.05) more in the LHKS group than in the UHKS group. The deposition of P, fecal P, urinary P and Ca, serum Ca, P, Ca, 1,25-(OH)2 D3, and

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iPTH changed insignificantly ($p > 0.05$) in control groups when compared with their baseline values.

It was shown that after the P load, significant differences were observed between the loaded and unloaded hypokinetic subjects regarding serum, urinary, and fecal P values and P retention. The oral P load intensified P loss from the body. It was concluded that the higher the P intake increased the greater P loss and the lower P deposition and thus the less likely it is for the P load to benefit hypokinetic subjects.

**Index Entries:** Phosphate deposition; phosphate metabolism; calcium; parathyroid hormone; 1,25-dihydroxyvitamin D.

**INTRODUCTION**

Hypokinesia (diminished movement) may be present as a result of occupation, lifestyle, disability, illness, or several other conditions. The term “hypokinesia” in contrast to immersion, weightlessness, or bed rest neglects hydrostatic pressure and compensatory force on long-bones factors and implies that the adaptive and deconditioning response is caused solely by hypokinesia (HK). Thus, using the term “hypokinesia” as a synonym to bed rest, weightlessness, and immersion is inappropriate. In this study, trained subjects were selected because increased muscular activity is associated with the need for a significant activation of anabolic processes, mobilization of electrolytes, and increased functional activity of organs and systems of the body. In contrast, decreased muscular activity is the exact opposite state in terms of the need of activation of anabolic processes. Hypokinesia is accompanied by a significant inhibition of the anabolic process and the prevalence of a catabolic process characterized by a significant increase of excretion of end products, negative nitrogen balance, and decreased tissue protein concentration in animals and humans (1–4). Moreover, it was found that prolonged HK is a factor for catabolism induction.

It has been shown that during prolonged HK, physiological systems that control and regulate the concentration of each electrolyte in blood and other endogenous fluids and the balance between consumption and elimination and, thus, electrolyte deposition and total electrolyte content in the body, are markedly affected (5,6). At the same time, sufficient information has been accumulated regarding the effect of long-term HK on electrolyte metabolism (7–9), which assumes that prolonged HK induces significant electrolyte changes in whole blood, urine, and feces and thus possibly contributes to the decreased electrolyte deposition and total electrolyte content in the body (5,6).

During prolonged HK, decreased electrolyte deposition capacity and electrolyte content in the body is characterized by increased plasma electrolyte concentration (5,6) and not by decreased plasma electrolyte levels as it happens in various clinical conditions. This reaction may suggest the presence of some other mechanisms that might affect the