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The Effect of Intravenous Magnesium Sulphate on Parathyroid Function in Primary Hyperparathyroidism

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Ten patients with primary hyperparathyroidism caused by enlargement of a single parathyroid gland were studied preoperatively. An intravenous bolus of 4 g magnesium sulphate followed by a continuous infusion of 2 g per hour for 3 hours increased serum magnesium from 0.76 to 2.12 mmol/l (median values, $p < 0.005$). Serum C-terminal parathyroid hormone (PTH) decreased from 94 to 78 pmol/l ($p < 0.005$), and serum intact PTH from 7.0 to 4.8 pmol/l ($p < 0.008$). The PTH changes preceded decreases in serum total calcium from 2.79 to 2.55 mmol/l ($p < 0.01$), and serum ionized calcium from 1.52 to 1.45 mol/l ($p < 0.007$). Urinary calcium excretion increased and urinary phosphate excretion decreased. Serum phosphorus, pH, albumin, creatinine, alkaline phosphatase, and urinary cyclic AMP showed no significant changes. The study showed that an intravenous magnesium sulphate infusion which at least doubled the normal serum magnesium concentration significantly suppressed PTH secretion in patients with primary hyperparathyroidism and subsequently reduced the serum calcium concentration.

Magnesium is one of the major factors controlling levels of serum calcium. It acts mainly by regulation of parathyroid hormone (PTH) secretion, but it also has effects on bone and renal tubules.

There is extensive experimental evidence that magnesium has effects on PTH secretion similar to those of calcium. Studies on animal parathyroid glands in vitro have shown that the effect of magnesium as compared to calcium on PTH secretion is between 1:3 and 1:1 [1–4]. A similar potency for the effect of magnesium has been shown in intact animals [5, 6]. A role on the target-organ response to PTH has also been demonstrated for magnesium but seems less important than the effect on PTH secretion [7–11].

The most extensive use of therapeutic magnesium has been in obstetric patients with premature labor or preeclamptic toxemia [12–14] and the dosages and expected side effects have been well documented. Only a limited number of studies of the effect of hypermagnesemia on calcium and parathyroid hormone have been performed in humans, but the evidence available suggests that both are suppressed [15–20]. The effect of hypermagnesemia has not been adequately studied previously in patients with primary hyperparathyroidism in whom it may have therapeutic potential in the management of severe hypercalcemia. We designed a study to test the hypothesis that an increase in serum magnesium concentration to at least twice normal levels would decrease parathyroid hormone secretion and serum calcium concentration.

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Methods

We performed preoperative studies on 10 patients with primary hyperparathyroidism. All of them were subsequently proven to have enlargement of a singly parathyroid gland at operation and were cured by removal of the "adenoma." There were 5 males and 5 females ranging in age from 30 to 68 years with a median age of 55 yr. Seven of the patients had normal serum creatinine levels and 3 had slightly elevated levels of 0.12, 0.13, and 0.17 mmol/l, respectively, the overall median serum creatinine level being 0.08. Three of the patients had mild hypertension but were not on medication during the study. None had significant cerebral or cardiovascular disease. The study protocol was approved by the Royal Brisbane Hospital Research Ethics Committee and all patients gave written informed consent.

During the study, a doctor was in attendance, there was continuous electrocardiogram monitoring, and hourly measurement of blood pressure and assessment of tendon reflexes. The patients were allowed a small amount of oral food and fluid intake. An intravenous line was set up in one arm and an intravenous cannula placed in an antecubital vein in the opposite arm for collection of blood samples without venous stasis. During the preliminary hour, 500 ml of 5% dextrose were infused. At time zero, an intravenous bolus of 4 g magnesium sulphate was given and, over the next 3 hours, 6 g magnesium sulphate in 0.9% saline was given at rate of 2 g per hour. The magnesium sulphate infusion was then ceased and 5% dextrose continued intravenously at a rate of 125 ml/hr. Blood samples were taken at −1, 0, +0.5 hours and then hourly for up to 7 hours. The patients voided and the urine was discarded at −1

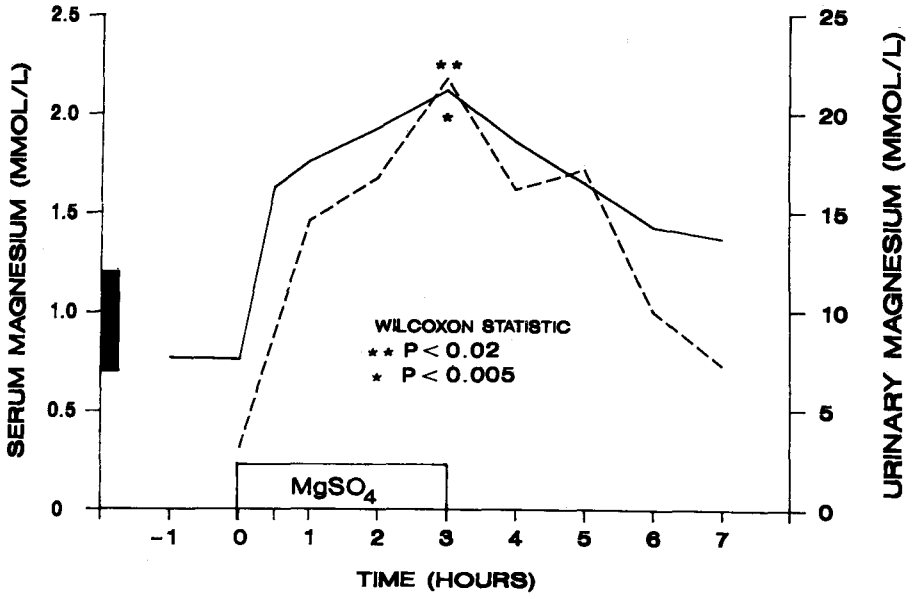

Fig. 1. Serum magnesium (solid line) and urinary magnesium (dotted line) concentrations. The reference range for serum magnesium is shown as a solid bar on the vertical axis.

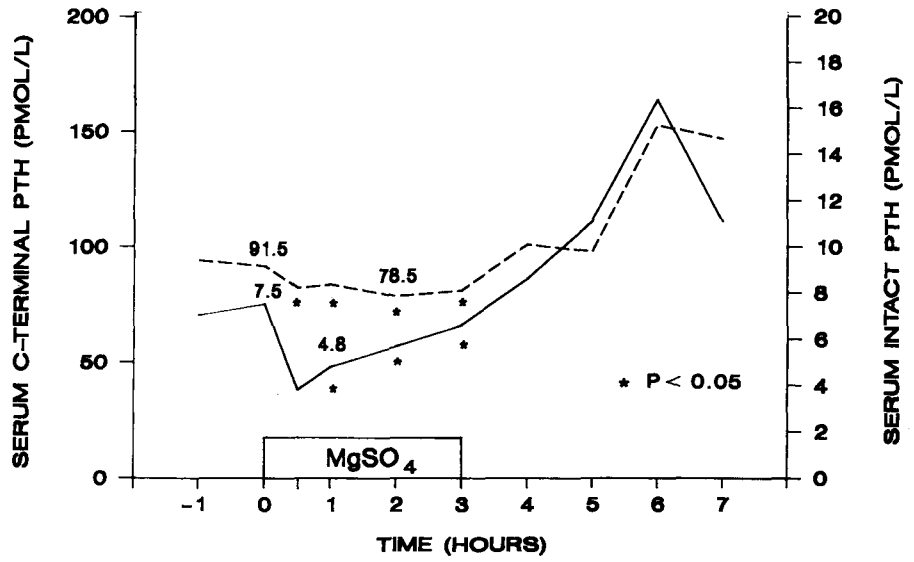

Fig. 2. Serum C-terminal PTH (dotted line) and serum intact PTH (solid line) concentrations.

hours and then urine samples were collected at time zero and hourly subsequently.

All of the data in the Results section below are expressed as median values and the lines on the graphs join median values. The statistical analyses compared the results at time zero with the results at each other time period by the nonparametric Wilcoxon matched-pairs signed-ranks test and any assumption of normal distribution of the data was thereby avoided. Serum and urinary magnesium were measured by a flame atomic absorption method with the serum results available within 30 minutes to ensure that levels of magnesium did not reach dangerous levels. Ionized calcium and pH were measured on anaerobically collected serum on a Radiometer Copenhagen ICAI analyzer. Serum C-terminal PTH was measured by the Incstar® assay (Incstar Corporation, Stillwater, Minnesota, U.S.A.) and serum intact PTH by the Nichols Institute Allegro® assay. Serum total calcium, phosphorus, albumin, creatinine and alkaline phosphatase, and urinary calcium and phosphorus were measured on a Technicon Sequential Multichannel Analyzer with computer. Urinary cyclic AMP was measured by a competitive protein binding assay [21].

Results

The magnesium sulphate infusion was well tolerated with no significant hypotension, respiratory suppression, changes in consciousness, or suppression of deep tendon reflexes. Five of the patients commented on a sensation of warm cutaneous flushing early in the period of continuous magnesium sulphate infusion, but this responded rapidly to temporary reduction in the rate of infusion in all cases. However, this mild side effect indicated that the doses of magnesium sulphate used in this study were appropriate and could probably not have been substantially increased. Serum and urinary magnesium levels

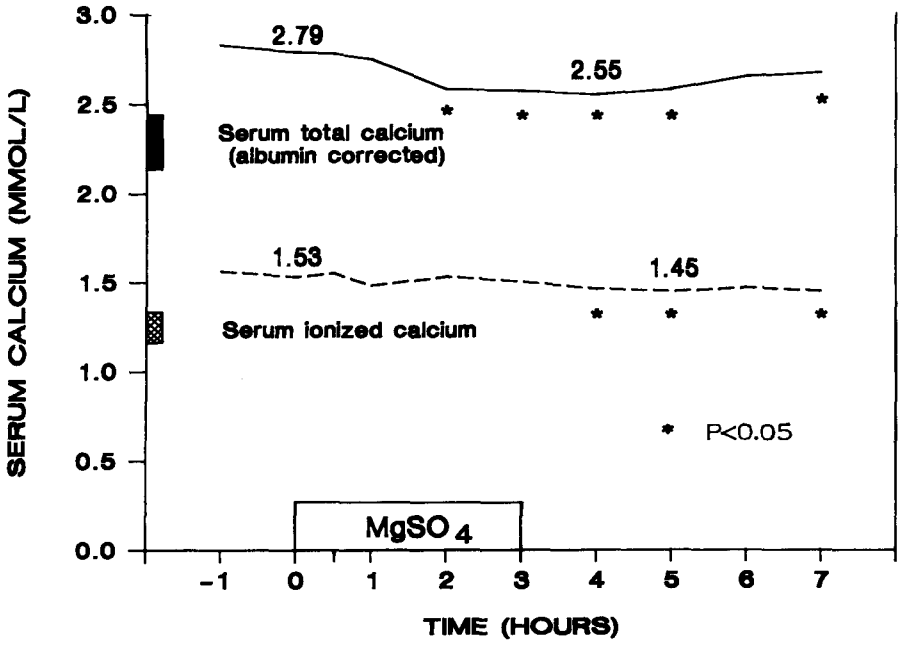

Fig. 3. Serum total calcium (solid line) and serum ionized calcium (dotted line) concentrations. The reference range for total calcium is shown as a solid bar and the reference range for ionized calcium is shown as a cross-hatched bar on the vertical axis.

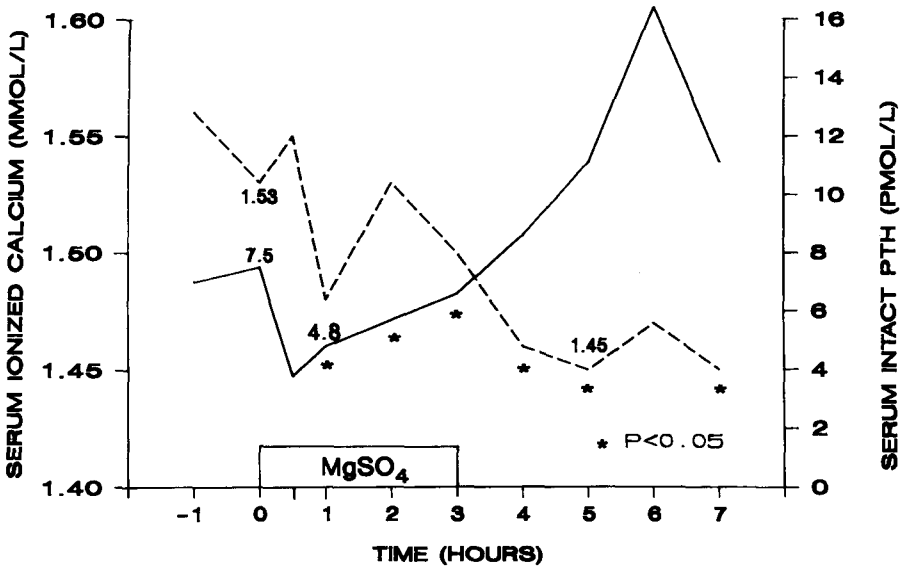

Fig. 4. Time relationship between changes in serum intact PTH (solid line) and serum ionized calcium (dotted line).

are shown in Figure 1. The serum magnesium concentrations (normal reference range, 0.7–1.2 mmol/l) rose from 0.76 at time zero to a maximum of 2.12 after 3 hours ($p < 0.005$) and declined to 1.37 after 7 hours. The first 5 patients in this study were observed for 2 hours after cessation of the magnesium sulphate infusion and the final 5 patients observed for 4 hours. The concentration of urinary magnesium increased significantly in parallel with the serum concentrations and these changes were also significant ($p < 0.02$).

Both C-terminal PTH and intact PTH showed significant decreases during the period of hypermagnesemia and then rebounded following cessation of the magnesium sulphate infusion (Fig. 2). The parathyroid hormone levels after 6 and 7 hours appear higher than control values but the data at these times are from only 5 patients and the difference is not statistically significant.

Serum total calcium (corrected to an albumin of 40 g/l) decreased from 2.79 mmol/l before magnesium sulphate to 2.55 mmol/l after magnesium sulphate ($p < 0.01$). Similarly, serum ionized calcium decreased from 1.53 to 1.45 mmol/l ($p < 0.007$) (Fig. 3). The decrease in serum parathyroid hormone preceded the decrease in serum calcium by some hours, the maximum decrease in parathyroid hormone concentration occurring during the magnesium sulphate infusion and the maximum decrease in serum calcium concentration occurring subsequently (Fig. 4).

Figure 5 shows the changes in urinary calcium and phosphorus. Urinary calcium excretion increased significantly during

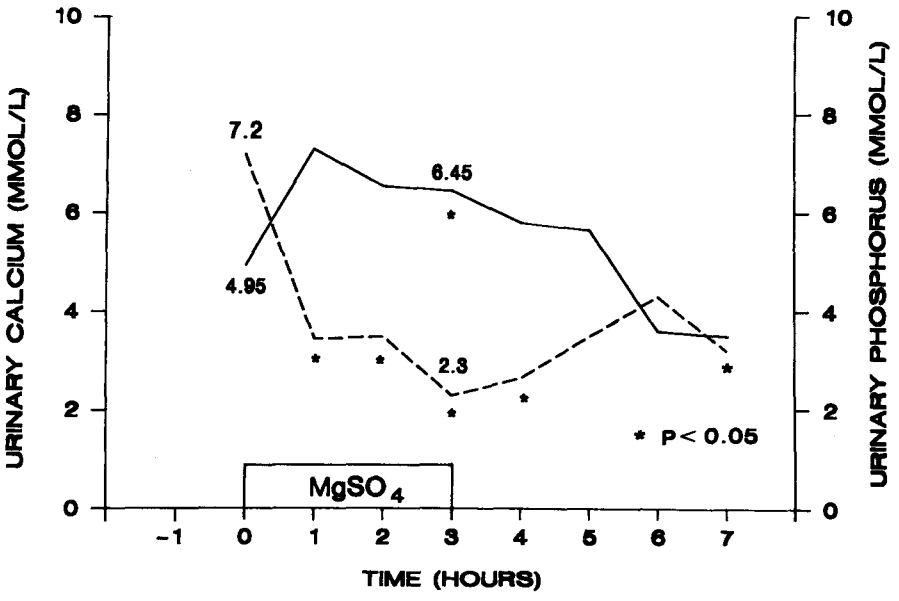

Fig. 5. Urinary calcium (solid line) and urinary phosphorus (dotted line) concentrations.

the magnesium sulphate infusion and returned to base line levels subsequently. The changes in urinary phosphorus excretion were more marked, with a significant decrease in excretion which persisted throughout the study period.

There were no significant differences between levels at time −1 and time zero for any of the measured parameters. There were no significant changes observed in serum phosphorus, pH, albumin, creatinine, or alkaline phosphatase, or in urinary cyclic AMP concentrations throughout the study period.

Discussion

This study has demonstrated that parathyroid hormone secretion in patients with primary hyperparathyroidism caused by enlargement of a single gland is not "autonomous," but is susceptible to manipulation. Specifically, induced hypermagnesemia resulted in suppression of serum parathyroid hormone levels measured by both a C-terminal assay (which measures a relatively long-lived but biologically inactive fragment) and also by an "intact" assay (which measures the biologically active molecule). Both assay methods revealed a reduction in PTH levels during the magnesium sulphate infusion period and a return to pretreatment levels subsequently. The PTH suppression preceded by some hours the maximum reduction in serum calcium levels and it may, therefore, be inferred that the lowered parathyroid hormone levels resulted in the reduction in serum calcium levels.

Parathyroid hormone normally acts on the renal tubules to reduce phosphorus resorption and increase calcium resorption. Therefore, the observed reduction in parathyroid hormone levels during magnesium sulphate infusion may explain the observed reduction in urinary phosphorus excretion and the concomitant increase in urinary calcium excretion. Although there was no change observed in urinary cyclic AMP excretion, serum cyclic AMP concentrations were not measured and we were, therefore, unable to calculate nephrogenous cyclic AMP excretion, which is a more sensitive index of the action of PTH on the renal tubule [22].

Hydration of the patients was maintained constantly from time zero and hemodilution would not account for the observed changes. This contention is supported by the lack of change in serum albumin and serum creatinine. The lowered serum PTH levels might be accounted for by increased peripheral receptor binding, but this seems unlikely in view of the rapid reversion from suppressed levels on cessation of the magnesium infusion. It is possible that magnesium has direct effects on the renal tubule or bone, but these also seem less important than the probable effect on PTH secretion.

The findings of the present study are generally consistent with and extend the results of previous work. In a study of 7 patients with premature labor treated with an intravenous loading dose of magnesium sulphate of 6 g over 30 minutes followed by a maintenance infusion of 2 g/hr, serum total and ionized calcium levels fell below the normal range at 2 and 3 hours and C-terminal PTH levels fell about 50% within 30 minutes and were undetectable after 60 minutes in 4 of the patients. PTH remained suppressed at 120 minutes but rose toward normal at 180 minutes. The changes in this study were not attributable to hemodilution or vasodilatation [15]. In another clinical study, 20 patients with preeclampsia were studied during magnesium sulphate infusion for up to 36 hours [16]. A loading dose of 4 g was given over 15–30 minutes followed by a continuous infusion of 1–2 g/hr. The mean serum PTH levels (assay not specified) increased significantly by the end of the infusion and serum ionized and total calcium declined. In this and another study, the authors concluded that the principal maternal response to magnesium-induced hypocalcemia involved increased PTH secretion, which tended to preserve calcium homeostasis [17].

An experimental study of hypermagnesemia in 22 normal subjects was conducted with 5 g magnesium sulphate given as an intravenous infusion over 2 hours. Serum total calcium levels decreased, but PTH was not measured [18]. A study of 7 normal males who were given an intravenous injection of 170 mg magnesium sulphate showed a decrease in C-terminal PTH

at 45 minutes, but no change in serum calcium concentrations over 210 minutes [19].

Only one previous study [20] has attempted to assess the effects of magnesium on serum calcium and PTH in patients with hyperparathyroidism. There were 3 patients with adenomas, 1 with carcinoma, and 2 with secondary hyperparathyroidism. Magnesium sulphate was given as an intravenous bolus of 1 g and levels rose from 1 to 2 mg/100 ml after 15 minutes with a higher peak (3.5) after 1 minute. Little change was detected in serum total calcium or N-terminal PTH over 15 minutes. This study did not achieve adequate levels of hypermagnesemia and its results are, therefore, difficult to interpret.

There is increasing evidence that secretion from hyperplastic and adenomatous parathyroid glands is not "autonomous." In vitro studies [23] of human adenomatous parathyroid tissue showed that it responded similarly to normal parathyroid tissue but with different sensitivity. Although PTH synthesis did not change greatly during short-term incubation, changes in secretory rates resulted in rapid changes in intracellular PTH stores within 2 hours [23]. This time frame is consistent with that observed in the present in vivo human study and with the effects of intravenous calcium injection [22]. Other in vitro studies [24] have demonstrated that changes in the responsiveness of pathological parathyroid cells contribute to hormonal hypersecretion, the most important parameter being an elevated set-point ("set-point" being the extracellular calcium concentration causing half-maximal inhibition of PTH secretion). The decreased sensitivity to the inhibitory effects of extracellular calcium on PTH release may be mediated by a relatively low cytosolic calcium at a given extracellular calcium concentration [25]. It has also been suggested [26] that cytosolic calcium may act as a second messenger mediating the effects of divalent cations (calcium and magnesium) on PTH release. Studies of dispersed bovine parathyroid cells showed that raising extracellular magnesium concentrations caused a dose-related increase in cytosolic calcium which, in turn, correlated strongly with inhibition of PTH release [26].

It should be noted that the present study, and those referred to previously, concern the control of PTH release. PTH biosynthesis is probably independent, recent observations suggesting that it is controlled by vitamin D [27, 28].

We have not used magnesium sulphate therapeutically to lower serum calcium and have only used it in cases of suspected or known hypomagnesemia. We do not recommend its use in the routine management of hypercalcemia, but it may be considered in patients with life-threatening complications of severe hypercalcemia. Reduction of very high levels of serum calcium may take up to several days using the traditional methods of intravenous saline and frusemide. In patients with severe hypercalcemia who are slow to respond, particularly those with cardiac arrhythmias, therapeutic magnesium sulphate may be worth consideration. Careful monitoring of levels of serum magnesium is mandatory, and, particularly in elderly patients, serum magnesium levels should not be allowed to exceed 3 mmol/l. Increasing levels of magnesium can cause suppression of nerve, and smooth, skeletal, and cardiac muscle activity with death occurring at serum levels of approximately 7 mmol/l [29].

In summary, this study has documented the effects of induced hypermagnesemia in patients with primary hyperparathyroidism. An increase in serum magnesium concentration to at least twice normal resulted in suppression of parathyroid hormone secretion and a subsequent decrease in serum calcium concentration.

Résumé

Dix patients présentant un hyperparathyroïdisme dû à l'hypertrophie d'une seule glande parathyroïdienne ont été étudiés avant d'être opérés. Une injection intraveineuse de sulfate de magnésie, 4 g, en bolus, suivie d'une infusion continue de 2 g/h pendant 3 heures a augmenté la concentration sérique en magnésie de 0.76 à 2.12 mmol/l (valeurs médianes, $p < 0.005$). La concentration en parathormone sérique (PTH), dosée par la méthode immunologique C-terminale a baissé de 94 à 78 pmol/l ($p < 0.005$) alors que celle de la PTH intacte a baissé de 7.0 à 4.8 pmol/l ($p < 0.008$). Ces changements en concentration de PTH ont précédé des diminutions en calcium total de 2.79 à 2.55 mmol/l ($p < 0.01$), et de calcium ionisé de 1.52 à 1.45 mmol/l ($p < 0.007$). Le taux d'excrétion urinaire de calcium a augmenté alors que l'excrétion en phosphates a diminué. Il n'y avait pas de modifications significatives dans les valeurs de la phosphorémie, le pH, l'albuminémie, la créatinine ou les phosphatases alcalines dans le sérum; de même, le taux d'AMP cyclique urinaire n'a pas changé. Cette étude montre qu'une perfusion intraveineuse de sulfate de magnésie, augmentant la concentration sérique en magnésie d'au moins le double, déprime de façon significative la sécrétion en PTH chez les patients présentant un hyperparathyroïdisme primitif et par conséquent a réduit l'hypercalcémie.

Resumen

Diez pacientes con hiperparatiroidismo primario causado por una glándula única aumentada de tamaño fueron estudiados preoperatoriamente. Un bolo intravenoso de 4 g de sulfato de magnesio seguido de una infusión continua a razón de 2 g por hora por 3 horas, produjo un aumento en el magnesio sérico de 0.76 a 2.12 mmol/l (valores promedio, $p < 0.005$). El nivel sérico de la fracción C-terminal de la hormona paratiroidea (PTH) descendió de 94 a 78 pmol/l ($p < 0.005$), y el nivel de la PTH intacta de 7.0 a 4.8 pmol/l ($p < 0.008$). Los cambios en la PTH precedieron a la disminución en la concentración del calcio sérico de 2.79 a 2.55 mmol/l ($p < 0.01$), y del calcio ionizado sérico de 1.52 a 1.45 mmol/l ($p < 0.007$). La excreción de calcio urinario aumentó y la excreción de fosfato urinario disminuyó. El fósforo sérico, pH, albúmina, creatinina, fosfatasa alcalina, y AMP cíclica urinaria no mostraron cambios significativos. El estudio demostró que una infusión intravenosa de sulfato de magnesio que logre por lo menos doblar la concentración normal de magnesio sérico suprime en forma significativa la secreción de PTH en pacientes con hiperparatiroidismo primario y consecuentemente reduce la concentración sérica de calcio.

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Invited Commentary

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The authors report a decrease in serum parathyroid hormone (PTH) and serum calcium values during an intravenous infusion of magnesium sulphate (Mg^{2+}) in patients with primary hyperparathyroidism (HPT). The patients apparently had only moderately elevated total and ionized serum calcium values prior to the infusion. The effect of Mg^{2+} with respect to total serum calcium values appeared earlier and was somewhat more pronounced as compared to the effects on ionized calcium values. As may perhaps be expected, intact PTH values fell more rapidly than C-terminal PTH after initiation of the Mg^{2+} infusion. Values of intact PTH, for some reason, started to rise again during the infusion and both PTH methods showed a rebound rise a few hours after its termination. This rebound rise in PTH was not significant since values were registered in only a few patients, but the rise seemed to reach values exceeding those measured prior to the infusion. During and after the infusion there was a significant rise in urinary magnesium and calcium.

It has previously been shown both in vivo [1–3] and in vitro [2, 4, 5] that the PTH secretion in HPT may generally be