THE EFFECTS OF REGULAR LONG TERM HEMODIALYSIS ON SERUM ELECTROLYTES AND PARATHYROID HORMONE LEVELS

by

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ABSTRACT

Regular long term hemodialysis is the accepted method of treatment for patients with advanced renal failure. While it is effective in maintaining a patient in reasonably good health for long periods of time many problems remain, not the least of which is metabolic bone disease. This work was undertaken to determine the effects of hemodialysis on serum electrolyte and parathyroid hormone levels. It was found that the dialysis regimen was effective in maintaining electrolyte and acid base balance. There was no definite evidence to suggest that hemodialysis contributed to the osteodystrophy which was evident in some of the patients.

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INTRODUCTION

The first practical form of hemodialysis was introduced by Scribner et al in 1960. Since then, due to rapid development of selectively permiable materials and many improvements in hemodialysis equipment and techniques, long term regular hemodialysis has become an accepted method of treatment for patients with end stage renal failure (1). Thus, many patients who, without hemodialysis would not have survived, are able to lead relatively normal and useful lives.

Although hemodialysis is effective in removing waste metabolites, such as urea, uric acid and creatinine, and in maintaining electrolyte balance many problems remain. Of these probably the most distressing for the patient are metabolic bone disease and metastatic calcification of parenchymous tissue.

The pathogenesis of the osteodystrophy of renal disease is complex and many factors have been implicated. Secondary hyperparathyroidism occurs in the early stages of renal failure as evidenced by increased serum parathyroid hormone levels and parathyroid hyperplasia. This may result from a chronic serum ionic calcium deficit (2). Skeletal resistance to circulating parathyroid hormone has also been cited as a possible cause of renal osteodystrophy (3, 4) and there is much evidence to support each

theory. The increased serum phosphate levels which occur in renal failure and are difficult to control by hemodialysis may also play a part, either directly or indirectly, in the progress of the disease, especially in the metastatic calcification of parenchymous tissue which often occurs (5, 6).

Probably the greatest advance in recent years in our understanding of mineral homeostasis was the discovery by De Luca and his co-workers (7, 8) and others (9, 10) of the role of the kidney in vitamin D metabolism.

The object of this study was to determine the effects of hemodialysis on plasma electrolytes and parathyroid hormone levels. Special attention was given to the levels of ultrafilterable calcium and magnesium, phosphate and parathyroid hormone and their possible relationship to renal osteodystrophy.

A group of twelve patients on long term hemodialysis was studied over a period of five months. In this type of study it is difficult to obtain a large series because the number of patients on hemodialysis at any given time varies between ten and fifteen. The members of the group are also constantly changing because many of them accept kidney transplant and therefore no longer need dialysis.

The range of ages, length of time on hemodialysis and duration of the chronic renal failure prior to commencement of hemodialysis were variables which may have influenced the results of the study but were difficult to assess.

Sample volume and frequency of sampling were also limiting factors. As all the patients were anemic it was decided that the sample size would be limited to 4 ml of blood, except for two occasions when extra samples were taken for parathyroid hormone assay. The frequency of sampling was limited to once a month.

LITERATURE REVIEW

It is generally accepted that long term regular hemodialysis is effective in removing waste products of metabolism from the body and in maintaining the electrolyte balance of patients with terminal renal failure (1, 13) but it has little or no effect on the osteodystrophy which causes so much discomfort and distress and which is often progressive in patients with renal failure (1, 2).

The biochemical and clinical abnormalities related to the osteodystrophy of chronic renal failure are similar to those found in pseudohypoparathyroidism (14). They are, increased circulating parathyroid hormone, mild hypocalcemia and impaired bone mineralization (15). Thus, much of the research carried out on pseudohypoparathyroidism is helpful in elucidating the underlying causes of renal osteodystrophy.

Metabolic acidosis and parathyroid secretion

The metabolic acidosis which occurs early in chronic renal failure has been implicated as a possible cause of secondary hyperparathyroidism, but this was not borne out by the work of Weber et al (16). They induced prolonged metabolic acidosis in normal subjects with repeated doses of ammonium chloride and found that although calciuria

occurred there was no change in intestinal absorption of calcium, phosphate or magnesium, and that plasma levels of 25-hydroxycholecalciferol and parathyroid hormone did not increase. They concluded that neither parathyroid hormone nor vitamin D and its metabolites mediate the net bone reabsorption which occurs during acidosis.

Altered albumin binding of calcium ions

Alteration of the affinity of albumins for calcium ions has been also suggested as a possible cause of secondary hyperparathyroidism in renal failure. Eastman et al (17) have demonstrated that in normal subjects the binding of calcium by albumins increases with increase in pH. Further experiments by Leme et al (18) have shown that although uremic subjects present a mild ionic hypocalcemia at any level of pH, their serum albumin-calcium binding is normal. They concluded that alterations in the binding affinity of albumins for calcium can be excluded as a factor in the hyperparathyroidism of chronic renal failure.

Magnesium and metabolic bone disease

Many workers have shown that hypomagnesemia will inhibit parathyroid hormone secretion (19, 20, 21) and that magnesium repletion alone will produce a rise in serum calcium in cases of hypocalcemia associated with hypomagnesemia. Targovnich et al (22) demonstrated that ionic magnesium is equally as effective as ionic calcium in suppressing parathyroid hormone secretion by isolated parathyroid glands

in vitro but there is little evidence to suggest that ionic magnesium plays any part in the regulation of the parathyroids in vivo. The evidence does suggest, however, that ionic magnesium is an activator for the enzyme systems responsible for secretion of parathyroid hormone. Contiguglia et al (23) found that the total body magnesium was increased in patients with chronic renal disease and that bone was the major reservoir. Their study showed that magnesium replaces calcium to a certain degree in the bone matrix of patients with chronic renal failure.

Prostaglandins and bone resorption

Klein et al (24) have demonstrated that prostaglandins stimulate bone resorption in tissue culture and that their effects are similar to those of parathyroid hormone. Experiments with parathyroidectomized rats showed, however, that prostaglandin E_1 did not induce an increase in serum calcium while parathyroid hormone did. It is interesting to note that Robertson et al (25) found very high levels of prostaglandins in the serum of a patient with renal cell carcinoma who exhibited hypercalcemia and a low level of serum parathyroid hormone. They hypothesized that the prostaglandins may have either induced the hypercalcemia or were part of a counterregulatory event. While prostaglandins may play some part in bone metabolism it is probably minor.

Skeletal resistance to parathyroid hormone

Llach et al (2) and Massry et al (2, 9) have shown that there is an apparent skeletal resistance to parathyroid hormone in patients with chronic renal failure and propose this as a possible cause for the secondary hyperparathyroidism which accompanies this condition. By inducing hypocalcemia in normal volunteers and in patients with chronic renal failure, with ethylene diamine tetra-acetate (E.D.T.A.) Llach et al (2) were also able to show that patients with renal failure displayed a delayed recovery from acute hypocalcemia despite higher than normal parathyroid hormone levels.

To demonstrate that the apparent skeletal resistance was not due to defective parathyroid hormone, Neer et al (12) gave patients with pseudohypoparathyroidism, biologically active parathyroid hormone from both bovine and human sources. They were unable to detect any changes in urinary phosphate or cyclic adenosine monophosphate (cyclic AMP) excretion which indicated that there was a lack of end organ response in these subjects. More recently Werder et al (24) have demonstrated a similar lack of response in another group of patients with pseudohypoparathyroidism.

Impaired parathyroid metabolism

Martin et al (26, 27) and Hruska et al (28) have demonstrated that the kidney is the main site for parathyroid hormone metabolism. About 60% of the circulating parathyroid hormone, both intact hormone and carboxyl terminal fragments, are removed by the kidney, glomerular filtration being the only mechanism for the removal of carboxyl terminal fragments. Hepatic uptake on the other hand is selective for the intact hormone. Reduced glomerular filtration may therefore account for at least some of the increase in plasma parathyroid hormone in chronic renal failure.

The role of calcitonin in renal osteodystrophy

Many workers have demonstrated that calcitonin plays an important role in the prevention of hypercalcemia in animals (29,30,31,32) but attempts to demonstrate a similar role in man have been inconclusive. A major problem is that several forms of immunoreactive calcitonin occur which differ in size and degree of immunoreactivity. Thus circulating levels of immunoreactive calcitonin may not reflect biological activity (33). Kanis et al (34) found that patients maintained on hemodialysis had low levels of circulating immunoreactive calcitonin, and that after bilateral nephrectomy their plasma levels of inorganic phosphate decreased while their plasma calcitonin levels increased.

There was also a decrease in osteoblast counts although this was transitory. They conclude that low levels of calcitonin may allow an increase in bone turnover in chronic renal failure. While this is an interesting possibility it is open to question because other workers (35) have found higher than normal levels of immunoreactive calcitonin in the plasma of patients with chronic renal failure. Much of the calcitonin was larger than normal, however, and was possibly biologically inactive.

Because of the uncertainty regarding the biological activity of immunoreactive calcitonin it is difficult to correlate plasma levels of this hormone with pathological states.

The role of phosphate in renal osteomalacia

Recent observations tend to confirm that plasma inorganic phosphate levels play an important part in the pathogenesis of renal disease. Ibels et al (36), using a remnant kidney model in rats, demonstrated that dietary restriction of phosphate prevented renal calcification and retarded degeneration of the remnant kidney. They concluded that renal calcification was produced by altered phosphate metabolism which occurred in the uremic state and that this caused an inflammatory and fibrotic reaction which resulted in progressive destruction of the kidney. Slatopolsky et al (37) using a remnant

kidney experiment in dogs, demonstrated that with a low phosphate diet (less than 100 mg/day) no increase in plasma parathyroid hormone occurred, even with a falling glomerular filtration rate.

Bellavia et al (38) also demonstrated that in rats; phosphate, magnesium, or magnesium and phosphate infusion increased renal calcium by 50 - 300% and decreased serum phosphate by 20 - 30%. They concluded that phosphate infusion may adversely effect parenchymal organs, especially the kidneys, when used to treat hypercalcemia, and that magnesium may enhance this effect. Thus, it is important that the magnesium and phosphate levels be maintained at near normal levels in order to reduce the risk of metastatic calcification of parenchymal tissues.

The role of calcium in osteodystrophy

While most patients with chronic renal failure have lower than normal serum ionic calcium levels, patients on regular hemodialysis tend to have ionic calcium levels within normal limits (13). Despite this many workers have found that dialysed patients have higher than normal serum parathyroid hormone levels (2). This suggests that a factor or factors other than circulating ionic calcium levels play a part in the regulation of parathyroid hormone secretion and that this factor is missing in patients with renal disease and pseudohypoparathyroidism.

Vitamin D and renal osteodystrophy

The renal rickets, or osteodystrophy which develops in patients with advanced renal failure is histologically similar to the lesions produced by chronic vitamin D deficiency. This suggests that defective metabolism of vitamin D occurs in renal failure (39, 40). Tanaka et al (41) and De Luca (7) have shown that cholecalciferol (vitamin D_3) is converted to 25-hydroxycholecalciferol by the liver and then further hydroxylated to 1,25-dihydroxycholecalciferol by the kidney. They, and other workers (42,43) have also demonstrated that 1,25-dihydroxycholecalciferol is the active form of vitamin D which promotes absorption of calcium and phosphorus from bone. Drezner et al (44) concluded that the hypocalcemia and bone disease in pseudohypoparathyroidism was probably due to a deficiency of 1,25-dihydroxycholecalciferol. Other workers (45,46) have noted that plasma 1,25-dihydroxycholecalciferol levels were low or undetectable in patients with renal disease even though their plasma 25-hydroxycholecalciferol levels were normal. Chesney et al (11) demonstrated that orally administered 1,25-dihydroxycholecalciferol could reverse the bone disease and increase the growth rate of children with renal disease.

Hausser and McCain (47, 48), in their review of vitamin D metabolism, suggest a model for the regulation of renal 1,25-dihydroxy-

cholecalciferol production which implies a negative feedback system involving the parathyroid glands. It may be summarized as follows.

A fall in circulating ionic calcium stimulates the parathyroid glands to secrete parathyroid hormone. The parathyroid hormone then stimulates the hydroxylation of 25-dihydroxycholecalciferol to 1,25-dihydroxycholecalciferol by the 1-hydroxylase system of the kidney. The 1,25-dihydroxycholecalciferol promotes absorption of calcium and phosphorus from the intestines and mobilization of calcium and phosphorus from bone, thereby increasing the circulating ionic calcium which feeds back to the parathyroid glands, shutting off the secretion of parathyroid hormone. There is evidence that the parathyroid hormone also promotes urinary excretion of phosphate thereby preventing an increase in circulating phosphate, which would otherwise result from the action of 1,25-dihydroxycholecalciferol (49, 50). There is also evidence to suggest that 1,25-dihydroxycholecalciferol has an inhibiting effect on parathyroid secretion (47, 48).

It is probable that failure of the renal 1-hydroxylase system of the kidney in the early stages of chronic disease, is responsible for the hyperparathyroidism and osteodystrophy of chronic renal failure (11).

The role of other vitamin D metabolites such as 24,25-dihydroxy-cholecalciferol is yet to be determined but it is possible that they may also play a part in mineral homeostasis.

MATERIALS AND METHODS

Patients A group of twelve patients on regular hemodialysis were studied at monthly intervals over a period of five months.

The hemodialysers used were Travenol RSP (recirculating single pass) instruments (Travenol Ltd., 6400 Northam Drive, Malton, Ontario) equipped with Gambro Lundia parallel flow dialysers (Gambro (Canada) Ltd., 2000 Argentia Rd., Mississauga, Ontario). The dialyser bath solution was obtained as a liquid concentrate from Travenol and was diluted with deionized water to a final volume of 12 liters just before use. The final composition of the bath fluid in m. mol. l^{-1} was sodium 134, potassium 1.0, calcium 1.5, magnesium 0.75 and acetate 37.

The patients are listed by number in table 1. Three of the group were originally diagnosed as chronic pyelonephritis and the remainder as glomerulonephritis. Patients 1, 4, 7 and 11 had radiological and clinical evidence of osteomalacia. A partial parathyroidectomy was performed on patient number 4 three months prior to the commencement of this study.

Blood Samples Blood samples from patients on hemodialysis were obtained from their arterio-venous shunts just prior to and at

the completion of dialysis. The samples were placed in plain (red stoppered) Vacutainer tubes and allowed to clot at room temperature. After centrifugation at 2 500 rpm for approximately 15 minutes, the sera were removed and placed in capped plastic vials No. 566353 Beckman Instruments, Fullerton, California, U.S.A. Samples for parathyroid hormone analysis were stored at -20°C. Because most of the patients were anemic the sample size was limited to 4 ml of whole blood or less.

Serum sodium and potassium Serum sodium and potassium levels were determined by flame photometry using the I.L. 343 flame photometer equipped with an in-line dilutor (Instrumentation Laboratory, Lexington, Mas. U.S.A.)

Serum chloride and total carbon dioxide Serum chloride and total carbon dioxide were measured with a semiautomated analyser I.L. 446 which employs a mercuric thiocyanate method for chloride and a pCO_2 electrode system for total carbon dioxide measurements. (Instrumentation Laboratory, Lexington, Mas., U.S.A.)

Serum inorganic phosphate Serum inorganic phosphate was measured by the method of Goldberg et al (51) based on the classic phosphotungstate method with ferrous ions as the reducing agent.

Blood pH Blood pH levels were determined at 37°C with a Radiometer ABL-1 blood gas analyser (Bach-Simpson Ltd., London, Ontario)

Serum pH Serum pH was measured with a Beckman model 3500 digital pH meter. (Beckman Instruments, Fullerton, California, U.S.A.)

Serum ultrafiltrates Serum ultrafiltrates were prepared with Centriflo membrane cones No. CF50A with a retention cut off at 50 000 MW, obtained from Amicon Corporation, Lexington, Mass., U.S.A. The membranes were soaked in distilled water for a minimum of two hours prior to use. They were then centrifuged for 15 minutes at 2 000 rpm to remove excess water in order to eliminate dilution of the serum ultrafiltrates. Results of preliminary experiments made with the membranes are given on page 22.

TABLE 1

PATIENTS STUDIED

Patient Number	Age (years)	No. of years on dialysis	Original Clinical Diagnosis
1	44	6	Chronic pyelonephritis
4	36	6	Glomerulonephritis
7	30	4	Glomerulonephritis
2	37	2	Glomerulonephritis
5	47	2	Glomerulonephritis
6	43	2	Glomerulonephritis
8	51	1	Chronic pyelonephritis
9	49	1	Glomerulonephritis
10	48	1	Glomerulonephritis
11	29	1	Glomerulonephritis
12	42	'	Glomerulonephritis

Total and ultrafilterable serum calcium and magnesium

Total and ultrafilterable serum calcium and magnesium measurements were made with a Jarrell Ash/Fisher model 850 computor controlled atomic absorption spectrophotometer (Fisher Scientific Company, Toronto, Ont.) with lanthanum chloride as dilutent. Dilutions of sera were made with a Cordis automatic dilutor (Cordis Corporation, Miami, Fla., U.S.A.).

Serum parathyroid hormone Serum parathyroid levels were measured by a double antibody radioimmunoassay method using reagents supplied by the Cambridge Nuclear Corporation, Billerica, Mass., U.S.A.

Statistical methods Both linear regression analysis and the Student's t test were used to analyse the experimental data.

The "Student's" t test values were calculated from the mean and standard deviation values for the various measures by the following method.

$$\text{when} \quad = \frac{/N_1 S_1 + N_2 S_2}{N_1 + N_2 - 2}$$

$$t = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{1/N_1 + 1/N_2}}$$

where $\bar{X}$ is the mean, S is the standard deviation and N is the population size.

Probability values were calculated from the Student's t values as two tailed probabilities. A probability (p) of less than 0.01 was taken to indicate that there was a significant difference between the two parameters.

PRELIMINARY STUDIES

The effects of centrifugation and temperature change on ultrafilterable calcium.

It has been reported by other workers (52) that protein binding of calcium increases with increase in pH. The following experiments were therefore used to determine the extent of pH change occurring during the preparation of samples for ultrafilterable calcium measurements.

Twenty 10 ml blood samples were obtained from healthy adult donors ranging in age from 20 to 45 years. Each sample was drawn into a plain (red stoppered) Vacutainer tube and allowed to clot at room temperature for approximately 45 minutes. The pH of the supernatant serum was measured at 37^{o}C using the Radiometer ABL-1 blood gas analyser. After centrifugation of the stoppered tubes at 2 500 rpm for 15 minutes the pH of the serum was again measured at 37^{o}C and also at room temperature (23^{o}C). The results are recorded in table 2.

There was no significant difference between the two measurements at 37^{o}C ($p = 1.0$) which indicated that there was no loss of carbon dioxide during centrifugation. The range of differences between the 37^{o}C and 23^{o}C pH measurements was 0.2 + 0.02 or 0.014 + 0.002 pH units per C^{o} decrease in temperature. This value corresponded to the values obtained by other workers (53).

TABLE 2

EFFECTS OF CENTRIFUGATION AND TEMPERATURE CHANGE ON BLOOD SAMPLES FROM NORMAL ADULTS

Sample No.	Initial pH at 37°	pH after centrifugation 37°	pH after centrifugation 23°	Change in pH
1	7.39	7.38	7.56	+0.17
2	7.34	7.38	7.52	+0.18
3	7.31	7.34	7.49	+0.22
4	7.37	7.37	7.59	+0.22
5	7.32	7.34	7.51	+0.19
6	7.35	7.35	7.58	+0.23
7	7.43	7.48	7.59	+0.16
8	7.36	7.36	7.56	+0.20
9	7.33	7.33	7.54	+0.21
10	7.41	7.45	7.58	+0.17
11	7.39	7.39	7.59	+0.20
12	7.42	7.41	7.61	+0.19
13	7.31	7.32	7.52	+0.21
14	7.43	7.45	7.65	+0.22
15	7.41	7.41	7.59	+0.18
16	7.41	7.43	7.58	+0.17
17	7.38	7.39	7.59	+0.20
18	7.33	7.33	7.52	+0.19
19	7.42	7.44	7.60	+0.18
20	7.39	7.40	7.58	+0.20
Mean	7.38	7.38	7.57	+0.20
S.D.	0.04	0.04	0.04	0.02

TABLE 3

EFFECTS OF CENTRIFUGATION AND TEMPERATURE CHANGE ON BLOOD SAMPLES FROM PATIENTS ON HEMODIALYSIS

Sample No.	Initial pH at 37°	pH after centrifugation 37°	pH after centrifugation 23°	Change in pH
1	7.46	7.46	7.63	+0.17
2	7.40	7.45	7.59	+0.19
3	7.43	7.42	7.64	+0.21
4	7.38	7.39	7.56	+0.18
5	7.44	7.44	7.67	+0.23
6	7.42	7.47	7.62	+0.20
7	7.37	7.36	7.55	+0.18
8	7.35	7.37	7.52	+0.17
9	7.47	7.45	7.68	+0.21
10	7.43	7.44	7.62	+0.19
11	7.36	7.36	7.54	+0.18
12	7.31	7.32	7.52	+0.21
Mean	7.39	7.41	7.59	+0.19
S.D.	0.05	0.05	0.05	+0.02

A study was also carried out on blood samples from the patient group (see table 3). The results were similar to those of the normal group ($p = 0.20$). The range of pH change between $37^{o}C$ and $23^{o}C$ was 0.19 ± 0.02.

Based on these findings and the work of Halver et al (54) a correction factor for ultrafilterable calcium measured at room temperature was devised.

Correction factor

$$\text{Measured UF Ca (m mol } l^{-1}) + 0.4(0.014)(37.0 - t)$$

$$\text{Measured UF Ca (m mol } l^{-1}) + 0.0056(37.0 - t)$$

where 0.4 = UF Ca (m mol l^{-1})/pH unit increase (72)

$0.014 = \Delta pH/C^{o}$ (72)

t = working temperature in ^{o}C

Because of limited sample size it was impractical to measure the pH of each patient sample so the above factor was used to correct serum ultrafilterable calcium levels to the value at $37^{o}C$.

Assessment of Centriflo membrane cones

The Centriflo membrane cones, used to obtain ultrafiltrates by centrifugation, required preliminary soaking in distilled water and in order to avoid dilution of the ultrafiltrate it was necessary to make

sure that residual water was removed before introduction of the serum. To do this the membranes were removed from the water and placed in conical supports, they were then centrifuged at 2 000 rpm for varying lengths of time. A pooled serum sample was then used to measure the degree of dilution produced by the residual water. 1 ml aliquots of the serum pool were added to the membranes and they were placed in dry centrifuge tubes and centrifuged for 20 minutes at 2 000 rpm. Each ultrafiltrate was then analysed for calcium by atomic absorption. It was found that preliminary centrifugation for 15 minutes at 2 000 rpm was adequate for removal of residual water.

R E S U L T S

Effects of hemodialysis on serum electrolytes

The effects of hemodialysis on serum electrolytes were investigated by measuring their concentrations just prior to and immediately after hemodialysis.

Sodium The pre and post dialysis serum sodium levels were 138 $\pm$ 2.4 m mol l^{-1} and 137 $\pm$ 2.2 m mol l^{-1} respectively. Except for three of the pre and four of the post dialysis levels the results were within the generally accepted range of 134 - 145 m mol l^{-1} (55) for normal adults. Comparison of the pre with the post dialysis ranges did not reveal any significant difference ($p = 0.03$). The data for serum sodium is given in table 4.

Potassium The average decrease in serum potassium during dialysis was 1.4 $\pm$ 0.5 m mol l^{-1}. The pre dialysis serum potassium levels were 4.8 $\pm$ 0.7 m mol l^{-1} and the post dialysis 3.3 $\pm$ 0.5 m mol l^{-1}. Before dialysis the range of serum potassium levels was significantly high ($p = \leq 0.001$) and after dialysis significantly lower ($p = < 0.001$) than the range for normal adults of 3.5 - 5.0 m mol l^{-1} (55). The data for serum potassium is given in table 5.

TABLE 4

COMPARISON OF PRE AND POST DIALYSIS SERUM SODIUM

MONTH	1		2		3		4		5	
Pat.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	140	138	143	141	137	136	135	132	137	138
2	139	139	138	138	136	135	139	139	136	138
3.	139	138	136	138	142	143	140	140	138	137
4	135	139	138	135	140	136	139	139	137	135
5	139	136	132	133	137	137	138	134	138	136
6	139	136	142	138	139	137	139	137	142	136
7	138	137	136	137	139	136	132	133	137	137
8	138	134	138	136	140	137	139	136	138	135
9	137	136	142	136	138	137	136	137	139	136
10	132	133	139	138	137	136	136	135	135	135
11	139	137	139	137	141	139	139	139	142	140
12	140	138	139	138	136	135	130	140	141	140

All values are in m mol l^{-1}

Pre dialysis $\bar{X}$ = 138, S = 2.4 m mol l^{-1}

Post dialysis $\bar{X}$ = 137, S = 2.2

Pre vs Post dialysis values

t = 2.36

p = 0.025

TABLE 5

COMPARISON OF PRE AND POST DIALYSIS SERUM POTASSIUM

MONTH	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	5.4	3.6	5.3	3.4	5.3	3.5	5.0	3.5	3.6	3.2
2	4.1	3.0	3.7	2.7	3.5	2.6	4.4	2.9	4.5	3.3
3	4.1	3.3	5.0	3.5	4.8	3.1	4.9	3.3	4.6	3.4
4	4.5	3.5	5.6	3.2	4.7	3.6	4.8	3.4	5.0	3.5
5	4.9	3.1	4.0	3.0	4.4	3.6	3.9	3.5	5.0	3.4
6	4.5	3.2	5.3	3.3	4.7	3.7	5.3	3.3	4.7	3.7
7	4.4	3.1	4.1	3.8	4.3	3.6	4.4	3.6	5.5	3.4
8	4.6	2.8	4.7	2.8	5.0	3.3	5.5	3.2	5.1	3.2
9	4.1	2.9	4.1	3.0	3.5	2.8	5.0	2.8	5.7	3.0
10	4.8	3.3	5.2	3.6	4.7	3.7	4.6	3.4	4.1	3.2
11	4.8	3.0	5.0	3.1	4.4	2.9	4.5	3.3	4.1	2.9
12	5.1	3.2	5.5	3.8	5.4	3.5	4.7	3.2	5.2	3.9

All above values are in m mol l^{-1}

Pre vs Post dialysis - $t = 15$, $p = 0.001$

Pre dialysis potassium $\bar{X} = 4.8$, $S = 0.7$ m mol l^{-1}

Post dialysis potassium $\bar{X} = 3.3$, $S = 0.7$ m mol l^{-1}

TABLE 6

COMPARISON OF PRE AND POST DIALYSIS SERUM TCO_2, Cl AND PO_4

Mo.	Pt#	T CO_2		Cl		PO_4		Pt#	T CO_2		Cl		PO_4	
		Pre	Post	Pre	Post	Pre	Post		Pre	Post	Pre	Post	Pre	Post
1	1	24.4	28.4	100	98	2.58	1.06	7	20.7	27.2	103	102	2.58	1.49
4		23.5	27.0	97	97	2.75	1.60		20.8	24.8	94	94	1.11	1.05
5		24.4	27.2	102	96	2.75	1.60		18.0	25.1	101	97	-	
1	2	23.9	27.3	98	99	2.18	0.86	8	20.0	25.7	96	98	2.41	1.38
4		20.8	26.8	100	100	2.41	1.78		18.5	21.8	98	93	2.12	1.38
5		-	-	101	99	-	-		-	-	-	-	2.50	1.28
1	3	15.1	22.0	94	100	2.39	1.61	9	20.4	23.4	96	96	2.06	1.55
4		19.2	25.4	101	94	3.67	1.89		20.4	25.3	96	90	2.11	1.22
5		14.7	22.8			-	-		-	-	-	-	2.11	1.02
1	4	24.6	25.6	98	94	1.28	1.17	10	21.9	24.4	96	102	2.56	1.28
4		23.6	27.1	99	99	1.00	0.78		19.0	20.3	101	95	2.27	1.86
5		22.2	25.3	-			-		17.1	18.2	87	94	-	
1	5	24.1	27.6	100	97	1.94	1.06	11	20.8	25.6	102	95	1.43	0.92
4		18.6	23.8	93	93	2.11	1.78		20.5	26.8	98	96	1.94	1.15
5		-		99	94	2.78	1.78					-	-	-
1	6	26.1	27.5	100	98	2.69	1.49	12	19.0	24.8	98	94	2.58	1.32
4		22.4	27.3	95	93	2.52	1.89		18.0	22.3	102	100	2.12	1.20
5		-		-	-	2.01	1.20		17.9	22.9	-	-	-	-

	T CO_2		Cl		PO_4	
	$\bar{X}$	S	$\bar{X}$		$\bar{X}$	
Pre	20.7	2.8	98.2	3.3	2.21	0.54
Post	25.0	2.4	96.6	3.1	1.30	0.31
Pre vs Post	$t = 6.25$		$t = 1.9$		$t = 7.87$	
	$p = <0.001$		$p = 0.067$		$p = <0.001$	

(All the above values are in m mol l^{-1})

Chloride A comparison of pre and post dialysis serum chloride levels is given in table 6. There was no significant change in serum chloride levels during dialysis ($p = 0.067$). The pre and post levels were 98.2 ± 3.3 m mol l^{-1} and 96.1 ± 3.1 m mol l^{-1} respectively.

Total serum carbon dioxide Total serum carbon dioxide ($T\ CO_2$) levels increased significantly during dialysis ($p = < 0.001$). $T\ CO_2$ levels were 20.7 ± 28 m mol l^{-1} before, and 25 ± 2.5 m mol l^{-1} after dialysis. The average increase during dialysis was 4.3 ± 0.9 m mol l^{-1}. Data for serum $T\ CO_2$ is given in table 6.

Inorganic phosphate A decrease of 0.89 ± 0.38 m mol l^{-1} in inorganic phosphate occurred during dialysis. The pre and post dialysis levels were 2.21 ± 0.54 m mol l^{-1} and 1.30 ± 0.31 m mol l^{-1} respectively. Data for serum inorganic phosphate is given in table 6.

Total serum calcium Because variations in serum calcium occur with change in position (56) the patient results were compared with a normal ambulatory control group (Table 7) and with a group of supine hospital patients (Table 8) none of whom had evidence of renal, parathyroid or bone disease. The pre dialysis total serum calcium range of 2.27 ± 0.22 m mol l^{-1} did not differ significantly from the range of the supine control group ($p = 0.55$) but was significantly below the

TABLE 7

SERUM CALCIUM AND MAGNESIUM LEVELS IN 20 NORMAL SUBJECTS

	T Ca	UF Ca	UF/T Ca	T Mg	UF Mg	UF/T Mg
	2.48	1.38	0.566	0.79	0.57	0.722
	2.40	1.37	0.571	0.80	0.54	0.675
	2.34	1.21	0.517	0.78	0.57	0.731
	2.27	1.10	0.485	0.79	0.54	0.684
	2.41	1.31	0.544	0.80	0.59	0.738
	2.36	1.28	0.542	0.80	0.58	0.725
	2.37	1.22	0.515	0.81	0.56	0.691
	2.30	1.21	0.562	0.78	0.56	0.712
	2.51	1.30	0.518	0.79	0.56	0.709
	2.48	1.37	0.552	0.78	0.57	0.731
	2.38	1.23	0.517	0.76	0.56	0.737
	2.47	1.36	0.551	0.80	0.58	0.725
	2.49	1.36	0.546	0.80	0.59	0.738
	2.52	1.40	0.556	0.76	0.57	0.750
	2.34	1.23	0.526	0.78	0.58	0.744
	2.37	1.26	0.532	0.79	0.57	0.722
	2.42	1.32	0.545	0.80	0.57	0.713
	2.49	1.33	0.534	0.74	0.56	0.757
	2.36	1.23	0.521	0.76	0.57	0.750
	2.30	1.20	0.522	0.75	0.56	0.747
$\bar{X}$	2.40	1.28	0.530	0.78	0.57	0.680
S	0.08	0.08	0.020	0.02	0.02	0.020

T Ca = total calcium, UF Ca = ultrafilterable calcium

Values are in m mol l^{-1}

TABLE 8

SERUM CALCIUM AND MAGNESIUM LEVELS IN 25 HOSPITAL PATIENTS

T Ca	UF Ca	UF/T Ca	T Mg	UF Mg	UF/T Mg
2.20	0.88	0.40	0.75	0.42	0.56
2.15	0.98	0.46	1.00	0.60	0.60
2.22	0.83	0.37	0.85	0.48	0.57
2.20	0.93	0.42	0.97	0.52	0.54
2.40	1.13	0.47	1.12	0.65	0.58
2.15	1.08	0.50	0.86	0.55	0.64
2.05	1.08	0.53 ,	0.84	0.55	0.66
2.03	0.93	0.46	0.97	0.46	0.47
2.12	1.08	0.51	1.00	0.61	0.61
2.36	1.12	0.48	0.78	0.60	0.77
2.36	1.12	0.48	0.75	0.44	0.59
2.45	1.23	0.50	0.70	0.45	0.64
2.23	1.33	0.60	0.75	0.50	0.67
2.21	1.32	0.60	0.80	0.53	0.66
2.17	1.33	0.61	0.78	0.48	0.62
2.34	1.18	0.50	0.80	0.53	0.66
2.20	1.28	0.58	0.90	0.63	0.70
2.35	1.03	0.44	0.85	0.58	0.68
2.45	1.28	0.52	0.81	0.46	0.57
2.10	0.93	0.44	0.92	0.61	0.66
2.32	1.10	0.47	0.90	0.55	0.61
2.33	1.18	0.51	0.80	0.45	0.56
2.35	1.13	0.48	0.90	0.55	0.61
2.50	1.15	0.46	0.83	0.46	0.55
2.13	1.03	0.48	0.76	0.41	0.54
$\bar{X}$ 2.40	1.28	0.53	0.78	0.57	0.61
S 0.08	0.43	0.02	0.02	0.14	0.07

levels of the normal ambulatory group ($p = < 0.001$). Post dialysis values of 2.42 m mol l^{-1} on the other hand were significantly higher than those of the supine control group ($p = < 0.001$) but within the range of the normal ambulatory group ($p = 0.52$). Data for serum total calcium is given in tables 9 and 15.

Ultrafilterable serum calcium The pre dialysis ultrafilterable serum calcium (UF Ca) levels of 1.19 $\pm$ 0.15 m mol l^{-1} were significantly higher than those of the supine controls ($p = < 0.001$). but were lower than the normal ambulatory controls ($p = < 0.001$). The UF Ca levels increased during dialysis so that the post dialysis levels of 1.28 $\pm$ 0.17 m mol l^{-1} were higher than those of the supine control group ($p = < 0.001$) but within the range of the normal ambulatory group ($p = 1.00$). Data for UF Ca is given in tables 10 and 15.

Ultrafilterable/total serum calcium ratios The ultrafilterable/total serum calcium ratios (UF/T Ca) for the pre dialysis, post dialysis and normal ambulatory group were similar ($p = 0.50$, 0.55, and 0.88 respectively), while the ratios for the supine group were significantly lower ($p = < 0.001$). UF/T Ca ratios for the pre and post dialysis samples were 0.52 $\pm$ 0.07 and 0.53 $\pm$ 0.08 respectively. Data for UF/T Ca ratios is given in tables 11 and 15.

TABLE 9

PRE AND POST DIALYSIS TOTAL SERUM CALCIUM

Month	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
	2.50	2.20	2.50	2.45	2.50	2.50	2.52	2.32	2.52	2.50
	2.35	2.30	2.35	2.30	2.25	2.50	2.38	2.28	2.27	2.51
-	2.10	2.60	2.10	2.25	2.10	2.60	2.10	2.60	2.13	2.27
4	2.20	2.55	2.20	2.55	1.75	2.30	2.22	2.55	1.78	2.30
5	2.45	2.70	2.40	2.70	2.40	2.55	2.48	2.70	2.42	2.57
6	2.50	1.75	2.50	1.95	2.20	2.15	2.50	2.00	2.20	2.16
7	2.45	2.60	2.25	2.45	2.30	2.50	2.45	2.60	2.31	2.49
8	2.15	2.70	2.55	2.05	1.95	2.40	2.18	2.68	1.98	2.43
9	1.70	1.95	2.05	2.15	2.10	2.45	1.78	1.97	2.10	2.48
10	2.45	2.40	2.45	2.40	2.15	2.50	2.45	2.40	2.18	2.50
11	2.20	2.60	2.50	2.25	2.60	2.55	2.23	2.60	2.61	2.55
12	2.40	2.55	2.20	2.55	2.05	2.55	2.42	2.54	2.08	2.57

All above values are in m mol l^{-1}

Pre dialysis calcium $\bar{X}$ =2.27 S= 0.22

Post dialysis calcium $\bar{X}$ = 2.42 S= 0.21

Pre vs Post t = -3.96 p = $<$0.001

TABLE 10

PRE AND POST DIALYSIS ULTRAFILTERABLE CALCIUM

Month	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	1.28	1.03	1.33	1.48	0.98	1.23	1.29	1.05	0.98	1.23
2	1.23	1.08	1.23	1.08	1.23	1.18	1.23	1.08	1.23	1.13
3	1.13	1.28	1.13	1.28	1.13	1.13	1.13	1.28	1.13	1.15
4	1.13	1.48	1.03	1.18	1.13	1.48	1.15	1.48	1.03	1.18
5	1.53	1.58	1.13	1.43	1.53	1.58	1.53	1.48	1.15	1.43
6	1.43	1.23	0.98	1.03	1.28	1.13	1.23	1.28	1.28	1.13
7	1.28	1.28	1.03	1.23	1.13	1.18	1.28	1.28	1.15	1.18
8	1.18	1.38	1.03	1.13	1.03	1.23	1.19	1.39	1.03	1.23
9	1.08	1.43	1.03	1.13	1.03	1.18	1.08	1.43	1.00	1.18
10	1.18	1.18	1.18	1.18	1.43	1.78	1.18	1.16	1.53	1.73
11	0.93	1.23	1.28	1.18	1.28	1.33	0.87	1.23	1.28	1.33
12	1.33	1.28	1.32	1.26	1.18	1.48	1.34	1.30	1.14	1.43

All above values are in m mol l^{-1}

Pre dialysis UF Ca $\bar{X} = 1.19$ $S = 0.15$

Post dialysis UF Ca $\bar{X} = 1.28$ $S = 0.17$

Pre vs Post $t = 3.04$ $p = < 0.001$

TABLE 11

PRE AND POST DIALYSIS ULTRAFILTERABLE/TOTAL CALCIUM

Month	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	0.510	0.468	0.523	0.604	0.392	0.492	0.512	0.453	0.389	0.492
2	0.523	0.470	0.532	0.670	0.547	0.472	0.516	0.474	0.542	0.450
3	0.538	0.492	0.586	0.569	0.538	0.435	0.538	0.492	0.531	0.507
4	0.514	0.580	0.468	0.463	0.646	0.643	0.518	0.580	0.579	0.513
5	0.624	0.585	0.471	0.530	0.638	0.620	0.617	0.548	0.475	0.556
6	0.572	0.703	0.392	0.528	0.582	0.523	0.492	0.640	0.581	0.523
7	0.522	0.494	0.458	0.502	0.491	0.462	0.522	0.492	0.498	0.474
8	0.549	0.511	0.404	0.361	0.528	0.513	0.546	0.519	0.520	0.506
9	0.635	0.733	0.502	0.523	0.490	0.482	0.607	0.726	0.476	0.476
10	0.482	0.491	0.482	0.492	0.681	0.712	0.482	0.483	0.702	0.692
11	0.422	0.473	0.482	0.492	0.492	0.522	0.390	0.473	0.490	0.523
12	0.554	0.502	0.512	0.524	0.576	0.580	0.554	0.512	0.548	0.556

Pre dialysis UF/T Ca $\bar{X}$ = 0.52 S = 0.07

Post dialysis UF/T Ca $\bar{X}$ = 0.53 S = 0.08

Pre vs Post t = 0.81 p = 0.500

Total serum magnesium The data for serum magnesium is given in table 13. The pre and post dialysis levels of 1.27 ± 0.18 m mol l^{-1} and 1.19 m mol l^{-1} were significantly higher than either of the control groups ($p = < 0.001$). Comparison of the pre and post dialysis ranges revealed that there was a significant decrease in total serum magnesium during dialysis ($p = 0.009$).

The ambulatory controls had significantly lower total serum magnesium levels than the supine controls ($p = < 0.001$).

Ultrafilterable serum magnesium The pre and post ultrafilterable serum magnesium (UF Mg) levels of 1.00 ± 0.02 m mol l^{-1} and 0.91 ± 0.02 m mol l^{-1} were significantly higher than either the ambulatory ($p = < 0.001$) or supine ($p = < 0.001$) groups. The pre dialysis levels were also significantly higher than the post dialysis levels ($p = 0.001$) indicating that there was a significant decrease in UF Mg during dialysis. Data for UF Mg is given in table 13 and 15.

Ultrafilterable/total serum magnesium ratio The pre dialysis ultrafilterable/total serum magnesium ratio (UF/T Mg) range of 0.77 ± 0.08 was not significantly different from the post dialysis range of 0.77 ± 0.09 ($p = 0.20$) and both ranges were significantly higher than either the ambulatory control or supine control groups. The supine UF/T Mg ratios of 0.61 ± 0.07 were significantly lower than those of the ambulatory control groups (0.68 ± 0.02) ($p = < 0.001$). Results are given in tables 14 and 15.

TABLE 12

PRE AND POST DIALYSIS TOTAL SERUM MAGNESIUM

Month	2		3		4		5		6	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	1.30	1.30	1.30	1.20	1.30	1.25	1.30	1.30	1.30	1.20
2	1.20	1.10	1.10	1.05	1.25	1.15	1.20	1.10	1.15	1.05
3	1.30	1.25	1.35	1.15	1.35	1.20	1.30	1.25	1.35	1.15
4	1.45	1.20	1.45	1.20	1.40	1.20	1.45	1.20	1.45	1.25
5	1.20	1.15	1.20	1.15	1.25	1.10	1.20	1.15	1.20	1.15
6	1.65	1.25	1.55	1.40	1.65	1.25	1.65	1.30	1.60	1.25
7	1.30	1.30	1.25	0.85	1.30	1.25	1.30	1.30	0.95	0.85
8	1.25	1.25	0.80	0.80	1.35	1.25	1.25	1.25	0.80	0.80
9	1.30	1.30	0.85	1.05	1.35	1.15	1.30	1.30	0.85	1.05
10	1.25	1.30	1.05	1.20	1.40	1.25	1.25	1.35	1.05	1.20
11	1.30	1.30	1.35	1.20	1.35	1.25	1.30	1.30	1.35	1.20
12	1.25	1.25	1.25	1.20	1.20	1.20	1.25	1.25	1.25	1.25

All above values are in m mol l^{-1}

Pre dialysis total magnesium - $\bar{X}$ = 1.27 S = 0.18

Post dialysis total magnesium - $\bar{X}$ = 1.19 S = 0.12

Pre vs Post t = 2.84 p = 0.009

TABLE 13

PRE AND POST DIALYSIS ULTRAFILTERABLE MAGNESIUM

Month	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
	1.05	1.00	1.15	0.95	1.05	0.90	1.05	1.00	1.15	0.95
2	0.95	0.75	0.95	0.75	0.90	0.80	0.95	0.75	1.00	0.75
3	0.95	0.80	1.00	0.95	1.00	1.85	0.95	0.85	1.00	0.95
4	1.25	1.05	1.25	1.05	1.05	0.85	1.25	1.05	1.25	1.05
5	1.15	1.00	1.20	1.15	0.95	0.80	1.15	1.00	1.15	1.00
6	1.25	1.15	1.25	1.15	1.15	1.00	1.25	1.15	1.15	1.05
7	1.10	0.95	0.80	0.70	1.00	0.85	1.10	0.95	0.80	0.70
8	0.95	0.90	0.65	0.65	1.00	0.95	0.95	0.90	0.65	0.65
9	1.00	0.95	0.75	0.75	0.95	0.95	1.10	0.95	0.75	0.75
10	0.85	0.95	0.80	0.70	1.05	0.95	0.85	0.95	1.05	1.00
11	0.95	1.00	0.80	0.60	1.05	1.00	0.95	1.00	0.85	0.65
12	1.00	0.90	1.00	1.00	0.90	0.90	1.00	0.95	1.00	1.00

All above values are in m mol l^{-1}

Pre dialysis UF Mg $\bar{X} = 1.00$ $S = 0.02$

Post dialysis UF Mg $\bar{X} = 0.91$ $S = 0.02$

Pre vs Post $t = 24.4$ $p = <0.001$

TABLE 14

PRE AND POST DIALYSIS ULTRAFILTERABLE/TOTAL MAGNESIUM

Month	1		2		3		4		5	
Pt.#	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	0.808	0.769	0.885	0.792	0.840	0.720	0.808	0.769	0.885	0.769
2	0.729	0.682	0.864	0.714	0.720	0.696	0.792	0.682	0.870	0.714
3	0.731	0.640	0.741	0.826	0.741	0.708	0.731	0.708	0.741	0.826
4	0.862	0.875	0.862	0.875	0.750	0.708	0.862	0.875	0.862	0.840
5	0.985	0.870	1.000	1.000	0.760	0.727	0.958	0.870	0.958	0.870
6	0.756	0.920	0.806	0.821	0.697	0.800	0.758	0.884	0.719	0.840
7	0.846	0.731	0.640	0.824	0.769	0.680	0.846	0.731	0.842	0.824
8	0.760	0.720	0.813	0.813	0.741	0.760	0.760	0.720	0.741	0.760
9	0.769	0.731	0.882	0.714	0.704	0.826	0.846	0.731	0.714	0.625
10	0.680	0.931	0.762	0.583	0.750	0.760	0.680	0.703	0.714	0.667
11	0.731	0.769	0.593	0.500	0.778	0.800	0.731	0.780	0.731	0.780
12	0.800	0.720	0.800	0.730	0.750	0.750	0.800	0.792	0.800	0.800

All above values are in m mol l^{-1}

Pre dialysis UF/T Mg $\bar{X} = 0.97$ $S = 0.08$

Post dialysis UF/T Mg $\bar{X} = 0.77$ $S = 0.09$

Pre vs Post $t = 1.28$ $p = 0.200$

TABLE 15

COMPARISON OF SERUM CALCIUM RESULTS

	T Ca		UF Ca		UF/T Ca	
	$\bar{X}$	S	$\bar{X}$	S	$\bar{X}$	S
Pre dialysis	2.27	0.22	1.19	0.15	0.52	0.07
Post dialysis	2.42	0.21	1.28	0.17	0.53	0.08
Normal	2.40	0.08	1.28	0.08	0.53	0.02
In Patients	2.25	0.13	0.93	0.11	0.49	0.06
	t	p	t	p	t	p
Pre vs Post	-3.96	0.001	-3.04	0.006	0.81	0.500
Pre vs Normal	-4.95	0.001	-4.07	< 0.001	0.60	0.550
Post vs Normal	0.67	0.520	0.00	1.000	0.16	0.880
Pre vs In Pat.	0.60	0.549	10.70	< 0.001	2.70	0.014
Post vs In Pat.	5.29	0.001	13.30	< 0.001	3.10	< 0.001
Normal vs In.Pt.	8.47	0.001	19.80	< 0.001	5.20	< 0.001

COMPARISON OF SERUM MAGNESIUM RESULTS

	T Mg		UF Mg		UF/T Mg	
	$\bar{X}$	S	$\bar{X}$	S	$\bar{X}$	S
Pre dialysis	1.27	0.18	1.00	0.02	0.97	0.08
Post dialysis	1.19	0.12	0.91	0.02	0.77	0.09
Normal	0.78	0.02	0.57	0.14	0.61	0.07
	t	p	t	p	t	p
Pre vs Post	2.84	0.009	24.40	< 0.001	1.28	0.200
Pre vs Normal	17.30	0.001	116.70	< 0.001	10.20	< 0.001
Post vs Normal	25.80	0.001	92.30	< 0.001	7.50	< 0.001
Pre vs In Pat.	15.60	0.001	26.00	< 0.001	13.00	< 0.001
Post vs In Pat.	16.20	0.001	21.20	< 0.001	10.80	< 0.001
Normal vs In Pat.	3.15	0.005	5.72	< 0.001	7.40	< 0.001

$\bar{X}$ = mean, S - standard deviation

All values are in m mol l^{-1}

Blood pH Blood pH measurements, taken just prior to and immediately after hemodialysis indicated that there was a small but significant increase in blood pH during dialysis ($p = 0.006$). The pre dialysis range was 7.382 ± 0.056 and the post dialysis 7.436 ± 0.037. The data for blood pH is given in table 16.

Effects of hemodialysis on parathyroid hormone

Serum immunoreactive parathyroid hormone (iPTH) levels were measured on samples obtained from the patients at the beginning of the study and again after a three month interval. The results are given in table 17.

Although patient number 7 showed a decrease, and patient number 10 an increase in iPTH during dialysis, there was no significant difference between the pre and post dialysis iPTH levels ($p = 0.61$).

The three patients, numbers 1, 7 and 11, with clinical and radiological evidence of osteodystrophy, had significantly higher serum iPTH levels than the other members of the group. The pre dialysis range for patients with osteodystrophy was $16\ 219 \pm 5\ 544$ pg ml^{-1} while the range for the other members of the group was $3\ 871 \pm 2\ 882$ pg ml^{-1}.

TABLE 16

EFFECTS OF DIALYSIS ON BLOOD pH

	Pre dialysis pH	Post dialysis pH
1.	7.339	7.396
2.	7.375	7.482
3.	7.389	7.496
4.	7.481	7.475
5.	7.432	7.467
6.	7.370	7.420
7.	7.445	7.458
8.	7.359	7.402
9.	7.409	7.432
10.	7.310	7.384
11.	7.434	7.440
12.	7.274	7.370
13.	7.363	7.470
14.	7.315	7.431
15.	7.374	7.424
16.	7.440	7.472
$\bar{X}$	7.382	$\bar{X}$ 7.436
s	0.056	s 0.037

Pre vs Post dialysis pH $t = -3.11$

$p = 0.006$ (15 degrees of freedom)

TABLE 17

COMPARISON OF PRE AND POST DIALYSIS

SERUM PARATHYROID HORMONE LEVELS

Patient number	Time on dialysis	Initial iPTH**	iPTH after 3 months**	Evidence of bone disease
1 Pre	6 y	21 979	23 087	Osteodystrophy with metastatic calcification
Post		12 026	22 566	
4 Pre	6 y	3 033*	3 513*	Osteodystrophy with metastatic calcification
Post		4 168*	8 453*	
7 Pre	4 y	11 910	14 466	Osteodystrophy
Post		4 202	3 515	
3 Pre	3 y	2 176	6 227	None
Post		1 110	7 289	
2 Pre	2 y	7 866	2 846	None
Post		532	2 317	
5 Pre	2 y	777	7 379	None
Post		764	8 357	
6 Pre	2 y	1 256	5 054	None
Post		2 363	2 183	
8 Pre	1 y	7 741	7 783	None
Post		5 213	8 696	
9 Pre	1 y	205	5 649	None
Post		1 875	4 395	
10 Pre	1 y	742	751	None
Post		3 197	5 100	
11 Pre	8 m	7 419	18 451	Osteodystrophy
Post		8 343	16 917	
12 Pre	7 m	4 801	685	None
Post		4 197	5 685	

* After removal of parathyroid glands

** Values are in pg ml^{-1}

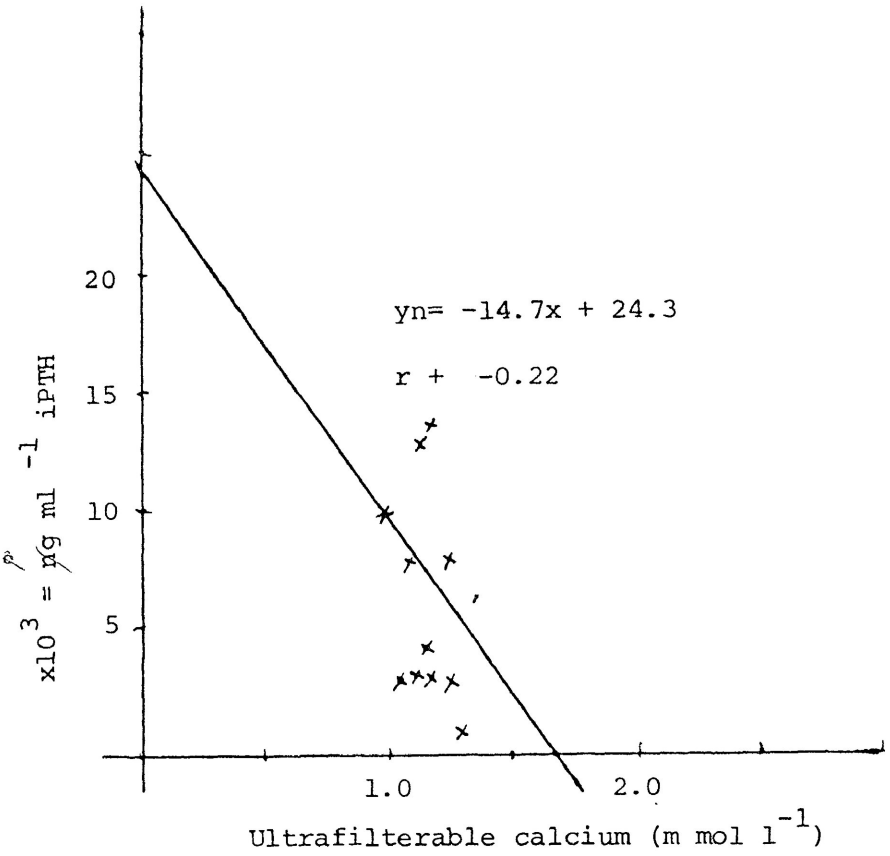

Figure 1. Comparison of ultrafilterable calcium with parathyroid hormone

Although patient number 4 had osteomalacia she was not included in this group because her parathyroid glands were removed three months prior to the commencement of this study.

Comparison of pre dialysis electrolytes with parathyroid hormone levels

In order to determine whether there was any correlation between the pre dialysis electrolyte and serum parathyroid hormone levels, the results were compared by linear regression analysis.

The pre dialysis ultrafilterable calcium levels did not correlate with the serum parathyroid hormone levels as shown in Figure 1. Ultra-

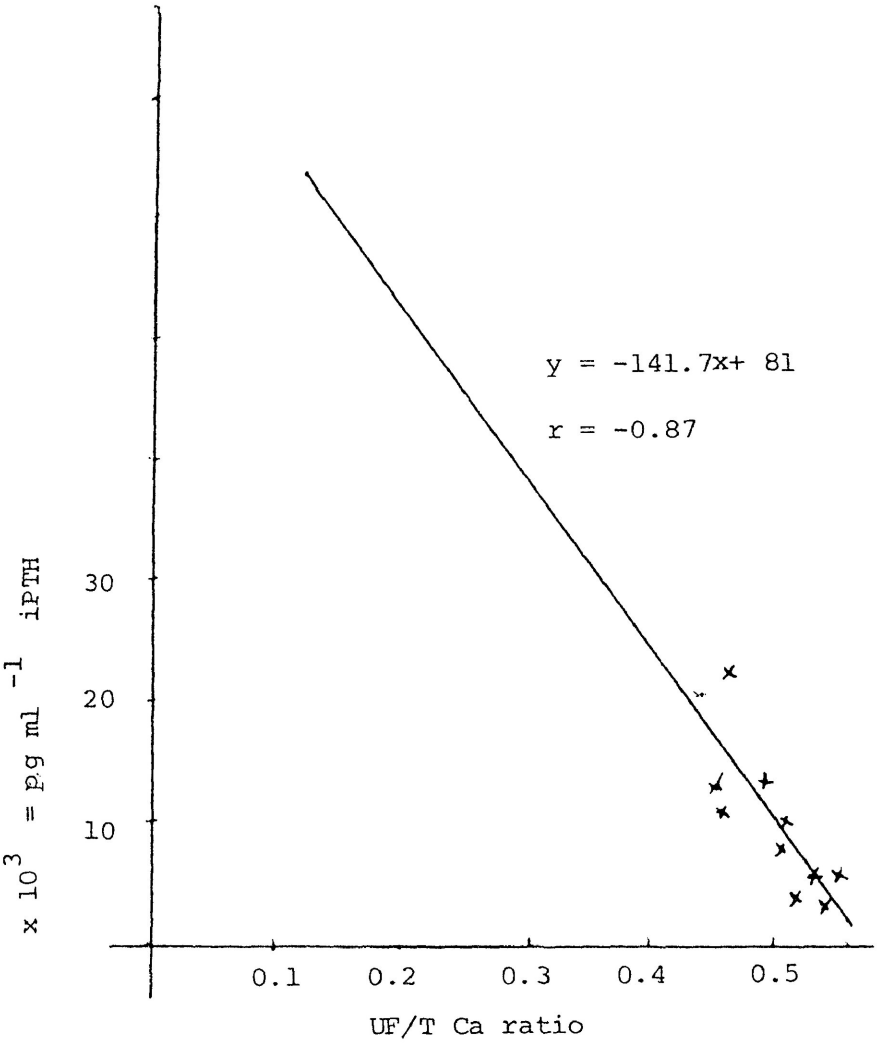

Figure 2. Comparison of ultrafilterable/total calcium ratios with parathyroid hormone levels.

filterable/total calcium ratios on the other hand were found to be related by the equation y = 141.7x+ 81 with a correlation coefficient r = -0.87 (see Figure 2).

Inorganic phosphate (Figure 3), ultrafilterable magnesium (figure 4) and ultrafilterable/total magnesium ratios (Figure 5) did not correlate with the serum parathyroid hormone levels as indicated by their correlation coefficients r = 0.03, r = -0.23 and r = 0.146 respectively.

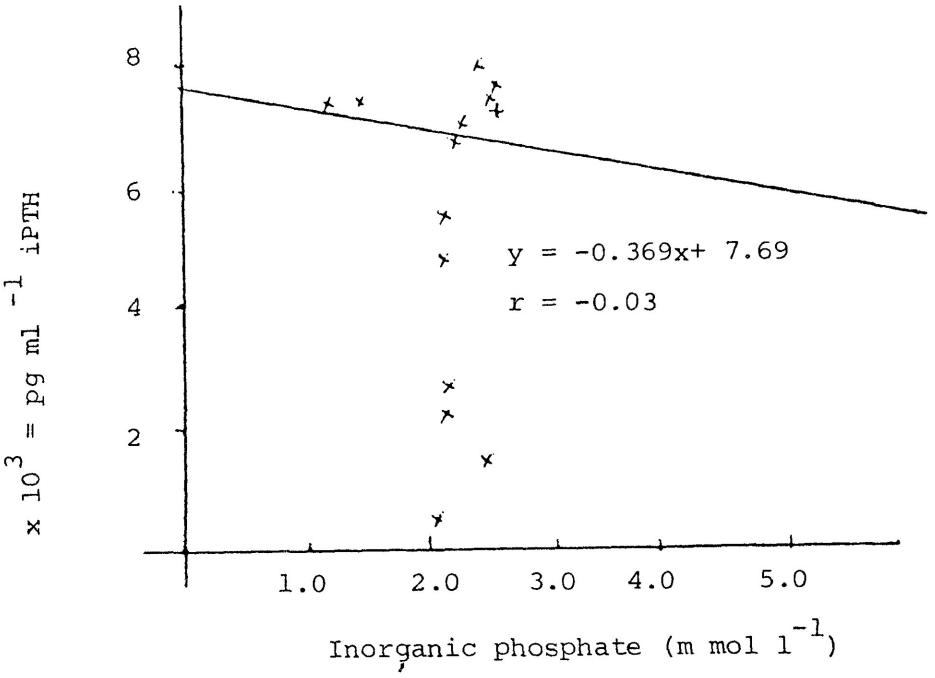

Figure 3 Comparison of serum inorganic phosphate with parathyroid hormone levels

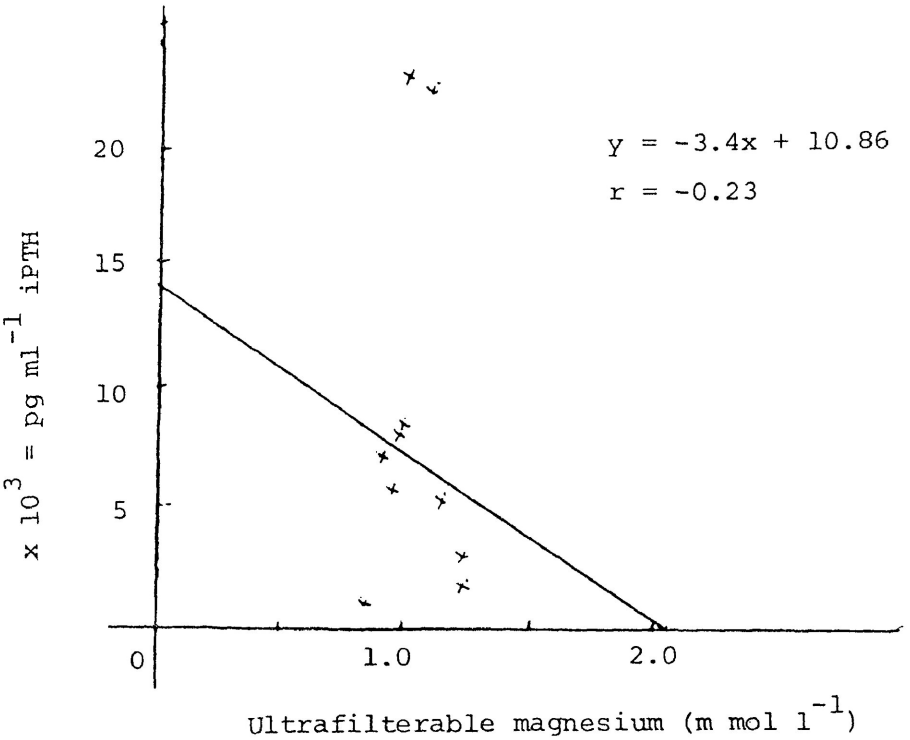

Figure 4 Comparison of serum ultrafilterable magnesium with parathyroid hormone levels.

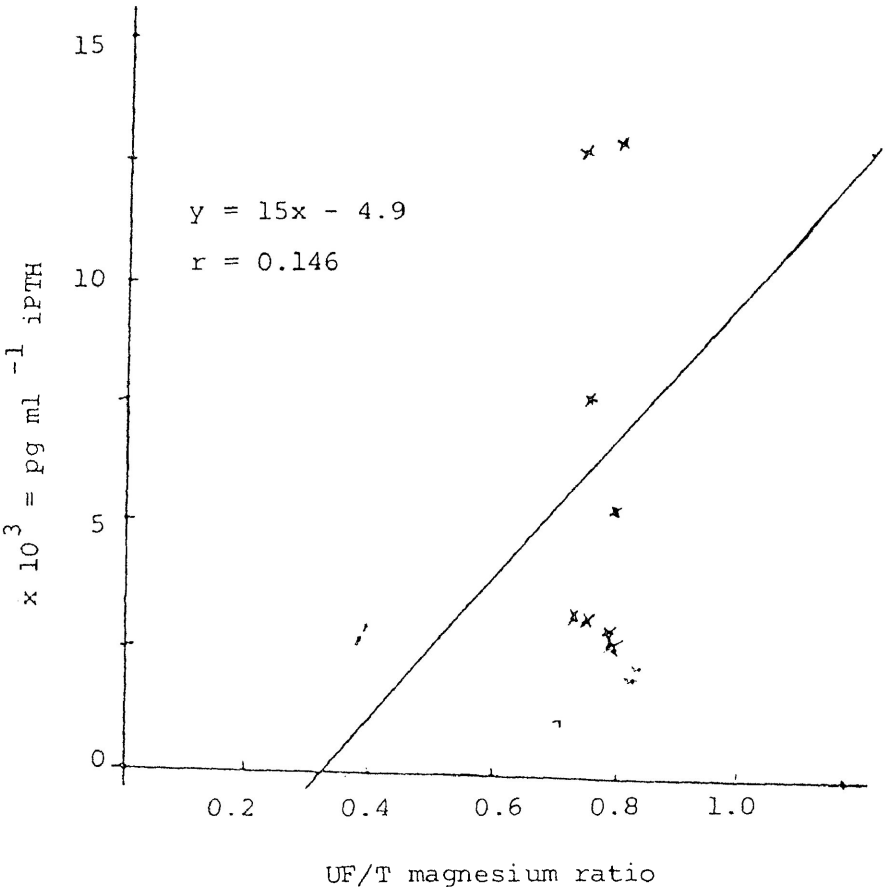

Figure 5 Comparison of ultrafilterable/total magnesium ratios with parathyroid hormone levels.

CONCLUSIONS

The results of this study indicate that the serum sodium and chloride levels of the patients were well controlled by the hemodialysis regimen, in that there was no significant difference between the pre and post dialysis values for these ions. Other inorganic ions showed varying degrees of change between dialyses but were adequately controlled.

There was an increase in serum inorganic phosphate between dialyses and the pre dialysis levels were generally above the normal adult range of 0.8 - 1.53 m mol l^{-1} (55) even though the patients were given oral aluminum hydroxide in order to limit their intestinal uptake of phosphate. During dialysis the serum inorganic phosphate decreased by 0.89 $\pm$ 0.38 m mol l^{-1} and post dialysis levels of serum inorganic phosphate were generally within the normal range. During this study none of the patients had post dialysis levels below the normal range, thus hypophosphatemia was not a cause of bone demineralization in these patients. Pre dialysis hyperphosphatemia on the other hand may increase the risk of metastatic calcification of parenchymous tissue.

Although serum potassium levels increased between dialyses, they remained within reasonable limits with only 5% of the values

above the adult normal range of 3.6 - 5.5 m mol l^{-1} (55). The mean decrease in potassium during dialysis was 1.43 $\pm$ 0.49 m mol l^{-1}. 67% of the post dialysis potassium values were below the lower limit of the normal range but were above the level which could cause cardiac arhythmia.

The pre dialysis total serum carbon dioxide levels were close to the lower limits of the normal adult range of 22 - 29 m mol l^{-1} (55) but rose to within the normal range during dialysis. The mean increase during dialysis was 4.3 $\pm$ 0.9 m mol l^{-1}. Both the pre and post dialysis pH values were generally within the normal range for adults, 7.35 - 7.42 (55), therefore systemic acidosis was not a cause of bone demineralization in these patients.

Serum calcium results were more difficult to assess in as much as the supine position (which these patients maintained during the five hours of dialysis) tends to lower serum calcium levels. The pre dialysis total serum calcium and ultrafilterable calcium levels were lower than the post dialysis levels, and those of the ambulatory control group. The pre dialysis ultrafilterable/total calcium ratios were similar to the post dialysis UF/T Ca ratios ($p = 0.5$) and those of the ambulatory control group ($p = 0.55$). Although the total calcium levels of the supine control group were similar to the pre dialysis levels, the ultrafilterable calcium and UF/T Ca ratios were significantly lower than the pre dialysis levels of the patient group ($p = 0.001$). While the pre dialysis ultrafilterable calcium levels were

slightly lower than those of the normal ambulatory controls their significance is questionable because blood samples were obtained from the patients in the supine position.

Total and ultrafilterable magnesium levels were elevated in both the pre and post dialysis samples and were approximately 1.5 times higher than those of the normal ambulatory group. The ultrafilterable/total magnesium ratios of the pre dialysis samples were also significantly higher than those of the normal controls. There was a decrease in both total and ultrafilterable magnesium during dialysis but the ultrafilterable/total magnesium ratios did not change significantly ($p = 0.2$). As all the patients had increased serum parathyroid hormone levels it follows that ionic magnesium in higher than normal concentrations does not suppress parathyroid hormone secretion in vivo as was suggested by the work of Targovnich et al (22) who found that ionic magnesium was as effective as ionic calcium in suppressing parathyroid hormone secretion in isolated parathyroid glands.

Serum parathyroid hormone levels appear to be a significant indicator of bone disease in that the patients with clinical and radiological evidence of osteodystrophy had serum parathyroid hormone levels in excess of 10 000 pg ml^{-1} while patients without evidence of osteodystrophy had parathyroid hormone levels significantly lower than this.

Although comparison of the ultrafilterable/total calcium ratios with parathyroid hormone levels by linear regression analysis indicated an inverse relationship between them, the number of observations were too small for any definite conclusions to be made.

Comparison of serum parathyroid hormone levels with inorganic phosphate (Figure 5), ultrafilterable calcium (Figure 1), ultrafilterable/total calcium ratios (Figure 2), ultrafilterable magnesium (Figure 4) and ultrafilterable/total magnesium ratios (Figure 5) by linear regression analyses did not reveal any significant relationships.

The results of this study have shown that the regimen of five hours dialysis every three days is adequate to maintain electrolyte and acid/base balance and that there is no evidence to suggest that fluctuations in electrolyte levels between dialyses play any part in the pathogenesis of osteodystrophy.

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