angle of the inclined screen test, and physical strength was evaluated by the time to survive in water (forced swimming method). GAA and CRT concentrations in serum, muscle and other organs were significantly decreased in CRF rats. GAA administrations significantly increased CRT content in muscle, and improved muscle capabilities dose-dependently.

| | muscle power | physical strength |
|---|---|---|
| | sliding angle; ° | swimming time; min |
| Sham | 55.2 ± 0.9 | 52.0 ± 3.8 |
| CRF(GAA 0) | 42.3 ± 1.9* | 21.9 ± 1.2* |
| CRF(GAA 10 mg) | 47.4 ± 1.3*$ | 25.5 ± 1.1*$ |
| CRF(GAA 100 mg) | 48.8 ± 2.1*$ | 29.0 ± 3.3*$ |

*; $p < 0.05$ vs Sham $; $p < 0.05$ vs GAA 0

In conclusion, we demonstrated a deficiency of GAA and CRT, and muscle weekness in CRF rats. However, oral GAA supplementation could recover muscle content of CRT and muscle capabilities in these rats.

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**IMPACT OF IDPN ON KT/V**

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The administration of intradialytic parenteral nutrition (IDPN) and the impact it has on Kt/V has not been thoroughly examined. The available literature addressing the influence of IDPN on Kt/V is limited and the observations are conflicting. Some studies have observed little or no significant change with IDPN administration. In contrast, one randomized, crossover study by McCann et al. of patients receiving IDPN observed a significant decrease. When amino acid (AA), dextrose and lipid components were administered a significant reduction in mean Kt/V resulted. When solutions containing only AA were administered, Kt/V was significantly less then when IDPN was withheld Administration of IDPN without AA resulted in a mean Kt/V not significantly different from the mean Kt/V when IDPN was withheld. AA containing IDPN reduced mean Kt/V by 18–23%. By personal communication with McCann, it was thought the amount of carbohydrate (CHO) determined whether the Kt/V was impacted when they studied further (unpublished). The authors found that by increasing the CHO, the Kt/V was not impacted (lower) and theorized that inadequate CHO resulted in catabolized protein generating urea. To investigate this further, a retrospective analysis was performed of the Pentec Health IDPN internal data base consisting of 489 patients that met inclusion and exclusion criteria. For this group: the age was 66.23+13.38 years; BMI was 25.79+56; 239 males (49%) and 250 females (51%); 304 patients with DM (62%); 430 patients with HTN (88%). Baseline albumin was 2.98+38 g/dL and baseline creatinine was 6.03+2.10 mg/dL. Results are below Table

| Kt/V (mean + SD) | | | |
|---|---|---|---|
| Baseline | Month 1 | Month 2 | Month 3 |
| 1.65+.34 | 1.44+.36 | 1.43+.35 | 1.44+.34 |

A repeated measures ANOVA indicated a statistically significant difference ($p < 0.0001$). A Tukey's adjustment resulted in non-significant differences between month 1 and month 2 ($p = 0.995$), month 1 and month 3 ($p = 0.987$), and month 2 and month 3 ($p = 0.936$). In conclusion, the initial decrease in Kt/V was followed by a period of stabilization, and the mean Kt/V never dropped below the KDOQI recommendation of 1.2.

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**A FULL COLOR VISUAL EDUCATING TOOL TO IMPROVE RENAL DIET COMPLIANCE**

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The objective is to simplify Renal diet education of a newly diagnosed, illiterate/non-English speaking CKD patient by using a 5 step concept that is explained by a fully illustrated color manual. The 5 steps to be followed are as follows:

Step 1. What foods are eaten daily? The renal dietitian is faced with many challenges when taking a diet history & educating a newly diagnosed CKD patient. It is of utmost importance to tailor education to specific patient's needs & to adapt the diet prescription to the patient's literacy level, culture, socio-economic circumstances & eating habits.

Step 2. How kidneys work. The damaged kidney is portrayed as a sieve with very fine mesh that does not allow normal kidney functions. Once the patient understands that his uremic symptoms are related to diet & damaged kidneys, he is willing to change his old eating habits. RRT is explained visually.

Step 3. What to eat. The calculated, technical diet prescription is explained in an understandable way by a visual tool called "The renal plate" that was specifically developed. The Renal plate is based on adjusting the Basic 3 Food Groups by making use of the South African Renal exchange lists.

Step 4. What not to eat. Foods to avoid due to high sodium, phosphate & potassium are presented by illustrations.

Step 5. How much to eat. Portion sizes are addressed through visuals since "The renal plate" only focuses on types of foods in a specific food group, not on quantity of each food group to be eaten.

Compliance to a renal diet is an integral part of patient care. If understandable renal diet education is linked to uremic symptoms & starts as soon as the patient is diagnosed, there is far better future diet compliance & disease outcome.

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**HIGH CIRCULATING LEVEL OF FIBROBLAST GROWHT FACTOR-23 PROMOTES RENAL EXCRETION OF PHOSPHATE IN HEMODIALYSIS PATIENTS WITH RESIDUAL RENAL FUNCTION**

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Fibroblast growth factor-23 (FGF-23) regulates phosphate metabolism and elevated levels occur in patients with kidney disease and are associated with smortality in maintenance hemodialysis (MHD) patients. Residual renal function (RRF) presumably improves phosphate metabolism in MHD patients. We investigated the role of circulating FGF-23 on urinary phosphate excretion and phosphate balance in 134 MHD patients. Demographics, laboratory data, and excretion capacity of phosphate were recorded. We used multivariable regression to analyze the relationship of serum phosphate with other factors and of the tubular reabsorption rate of phosphate with other factors. Patients with high urinary output ( > 200 mL/day) had lower serum phosphate, calcium, iPTH, and FGF-23 than patients with low urinary output ( < 200 mL/day). The independent risk factors for elevated serum phosphate were nPNA, iPTH, and FGF-23 in patients with low urinary output, and female gender and GFR in patients with high urinary output. In high urinary output patients, the weekly phosphate excretion was 300 to 1500 mg, the renal contribution to weekly phosphate elimination was about 15% when GFR < 2 mL/min, 25% when 2 ≤ GFR < 5 mL/min, and 40% when GFR ≥ 5 mL/min. The tubular reabsorption of phosphate (44% ± 19%) was nearly 50% of the normal level, much lower than that of sodium, chlorine, and calcium which ranged from 85% to 99%. Elevated circulating FGF-23 was significantly associated with decreased tubular phosphate reabsorption after adjusting for GFR and serum phosphate ($\beta = -0.147$, $p = 0.003$). In conclusion, RRF is associated with significant capacity to excrete phosphate and high levels of FGF-23 promote phosphate excretion in remnant nephrons.

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**IMPACT OF NLRP3 INFLAMMASOME ON THE DEVELOPMENT OF HYPERTENSION AND RENAL AND CARDIAC HYPERTROPHY IN 2K1C AND DOCA/SALT MICE**

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NLRP3 inflammasome is formed by NLRP3, the adaptor ASC, and caspase-1, functions as a sensor of danger signals and triggers processing and release of interleukin-1beta (IL-1β). Recent animal studies demonstrate that NLRP3 inflammasome promotes renal inflammation. However, little is known on the role of NLRP3 in hypertension. We have investigated the effect of NLRP3 inflammasome on blood pressure, plasma renin activity and concentration