Evolution of the low-protein diet in chronic uremia: with special emphasis on the electrodialyzed whey diet

James L. Casey
University of Nebraska Medical Center

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EVOLUTION OF THE LOW-PROTEIN DIET IN CHRONIC UREMIA WITH SPECIAL EMPHASIS ON THE ELECTRODIALYZED WHEY DIET

By:

J. LYNN CASEY

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## TABLE OF CONTENTS

- Introduction ........................................................................... 1
- Definitions ............................................................................. 1
- Some Early History of the Low-Protein Diet ............................. 3
- The Low-Protein Diet in the 20th Century ................................. 4
- Results of Low-Protein Diets .................................................... 13
- Complications of the Low-Protein Diet .................................... 16
- Case Presentations .................................................................... 19
- Discussion .............................................................................. 26
- Summary ................................................................................. 31
- Conclusion ............................................................................... 31
- Bibliography ........................................................................... 33
- Appendix ............................................................................... 35

**EVOLUTION OF THE LOW-PROTEIN DIET**

**IN CHRONIC UREMIA WITH SPECIAL EMPHASIS**

**ON THE ELECTRODIALYZED WHEY DIET**
Introduction

Progressive and irreversible renal diseases produce in late stages a clinical syndrome that is called "uremia". The chemical characteristic of uremia is azotemia. In this condition, nonprotein nitrogen (NPN) is increased, mainly due to retained blood urea nitrogen (BUN). However, creatinine, uric acid, certain conjugated amino acids, and some products of amino acid intermediary metabolism also contribute to azotemia (1).

This paper will be concerned with the evolution of the low-protein diet in the treatment of uremia. Before beginning this discussion, several commonly used terms and concepts will be defined.

Definitions

An organism is in nitrogen balance when the processes of catabolism, or breaking down of tissues, and anabolism, or building up of tissues, are in equilibrium. Thus, dietary nitrogen intake equals combined nitrogen excretion in feces, skin and urine. Positive nitrogen balance exists when, as in a growing animal, anabolic processes are greater than catabolic, and consequently more protein is taken in than excreted. Conversely, in negative nitrogen balance, catabolism exceeds anabolism. Thus, as in old age, starvation, and wasting diseases, more nitrogen is excreted than is ingested (2).

Uremia due to chronic renal insufficiency is a condition of chronic negative nitrogen balance. Since there is an incurable underlying lesion, the principles of management involve re-establishment of the best possible nitrogen balance.

Biologic value of protein is a ratio of the amount of food absorbed to the amount eliminated (2). This can also be thought of as the ratio of the amount
of protein ingested to the amount utilized in the synthesis of body protein (3).

The eight essential amino acids are lysine, leucine, isoleucine, valine, threonine, tryptophan, phenylalanine, and methionine. The other 14 amino acids can be synthetized by the body and are thus "non-essential" (2, 3). If a protein source has a high percentage of amino acid content and contains all the essential amino acids, and the concentration of each essential amino acid is roughly proportional to its minimal daily requirements, it is said to be of high biologic value (3).

Rose, in 1956, established the minimal daily requirement of essential amino acids. At the same time, he suggested a daily caloric intake of 35-50 cal/kg per day to insure maximum protein utilization. He showed that if adequate calories were not ingested, both dietary and body protein will be used as caloric sources and nitrogen balance will be negative (4).

The work of Rose and Dekker, in 1956, gave impetus to the use of urea sparing in the treatment of uremia. They labeled urea with $^{15}$N and fed it to the rats. The diet excluded non-essential amino acids. They demonstrated up-take of the isotope in non-essential amino acids of the tissues. This proved that, in rats, urea could be utilized for metabolic needs, when non-essential amino acids were excluded from their diets (5).

Acidosis, anemia, and azotemia are the cardinal characteristics of uremia. Acidosis results from the impaired ability of the kidney to excrete acid and retain bicarbonate. The anemia, although complicated and incompletely understood, is in general the result of decreased erythropoiesis. With the greatly reduced glomerular filtration rate in severe renal insufficiency, azotemia occurs (1).
Toxic symptoms result from this azotemia, and may be due to retained urea nitrogen or another unremoved constituent (Vide infra). The signs and symptoms of uremia can appear in any system of the body. Gastrointestinal symptoms may predominate. The uremic patient may experience nausea, vomiting, anorexia, diarrhea, melena, and severe malnutrition. Lassitude and mental depression, coma, psychosis, convulsive disorders and tetany may occur. Uremic deposits in the skin and anemia may be responsible for a sallow complexion. Purpura, petechiae, and easy bruising may be noted. Hypertension, encephalopathy, papilledema, congestive heart failure, edema, pericarditis, and neuropathy are other manifestations of uremia (1,6).

Some Early History of the Low-Protein Diet

Several important advances, both investigative and clinical, have led to the present status of the "art and science" of management of this condition of chronic renal wasting. The story began in 1827, when Dr. Richard Bright wrote a treatise on the kidney in "dropsical effusion". He collected and studied 24 cases of "anasarca with coaguable urine" over a twelve year period. This symptom complex has come to be known as Bright's disease (7).

In Guy's hospital reports, 1836, Dr. Bright wrote the following about the dietary treatment of renal disease:

"A great deal still further depends upon diet. Where milk is grateful, if it sits easily on the stomach, and is freely digested, I believe it to be one of the best aliments which can be taken. Light animal food frequently agrees; tea should be avoided; all badly cooked vegetables, and all fruits, will often be found to be injurious. The great rule is to avoid everything which obviously deranges the stomach; and to take tonic and nutritive, but not stimulating food. The less wine and spirituous liquors is taken, the better." (8)

As early as 1845, some practitioners were treating chronic renal insufficiency with low "azote" diets, as observed by Thomas Watson:
"The diet in the chronic forms of the disease should be nutritive but unstimulating. M. Solon suggests that if, in the renal cases, urea be detected in the blood, the patients should be restrained from too animalized a diet. Dr. Budd has had the same thought, and has put to the test, I believe in the Hospitalship Dread Nought, the utility of withholding all articles of food that contain azote. I have found this restriction entirely useless in one painful case, in which it was fairly enforced. In fact, the principal of such restriction appears to be wrong: the urea if furnished to the blood, not in the primary assimilative process, but in that which is secondary and destructive." (9).

The Low-Protein Diet in the 20th Century

Sir William Osler noted in 1909 that the most important element in treating Bright's disease was care in food and drink. The patient was to be warned not to eat meat more than once a day (10).

These then were the most important clinical advances at the turn of the century. "How much" and "what kind" of protein to be used in the treatment of uremia became the important questions that nephrologists began to consider. The following studies came from both clinical and laboratory investigations, and are presented in chronological order.

Smith, in 1927, stated that nitrogen retention should be treated by a daily diet of less protein that could be excreted by the kidney in 24 hours (11). Using this principle, he sustained a boy with chronic nephritis and a non-protein nitrogen level of 166 mg/100 ml for six months. Symptoms were ameliorated and the NPN was reduced to 66 mg/100 ml on a diet of 0.26 g/Kg/day. After the diet was liberalized in protein (40 g/d), the NPN rose, and the patient became nauseated with vomiting, lethargy, and convulsions preceeding his death (12).

Van Slyke and others in 1928 treated a patient for one month with a 40 g protein/d diet. His BUN dropped from 141 to 57 mg/100 ml although his blood urea clearance did not improve (13).

Farr and Smadel, in 1937, made rats nephritic by injecting them with anti-
kidney serum. They divided the nephritic rats into three groups: low-protein (5% by weight), normal-protein diet (18%), and high-protein diet (40%). The latter group had more abnormal urinary sediment, more proteinuria, a greater decrease in glomerular filtration rate, and a very high mortality rate (14/16). (The two survivors were nephritic at the conclusion of the experiment.)

Results of work by Farr and Smadel (14):

<table>
<thead>
<tr>
<th></th>
<th>Low Protein</th>
<th>Normal Protein</th>
<th>High Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Animals</td>
<td>16</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Deaths during 1st month</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Renal failure death 2-11 mo</td>
<td>0</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Death other than renal fail</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Survivors with nephritis</td>
<td>2</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Recovered</td>
<td>11</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Kempner, in 1944, showed that a low-sodium, low-fat diet designed for the treatment of hypertension was effective in treating renal failure. This "rice diet" contained mainly rice, fruits, and fruit juices, and was very low in protein content (20 g/d) (15, 16). However, rice protein is of poor biologic value and the high potassium content made the diet hazardous to use. In addition, the low fat content made it very unpalatable, and therefore patient acceptance was poor (17).

Fishberg, in "Hypertension and Nephritis" in 1944 advocated a "low-protein, high-carbohydrate, medium-fat diet." The fat and carbohydrate were said to have a "protein-sparing" effect; the fat intake was moderate because of decreased ability of some nephritics to absorb it. He stated that "there is no convincing evidence of any difference between the various protein foods as regards nephrotoxic action or liability to produce uremic symptoms in a patient with impaired renal function" (18).

Addis did much to cement many of the current ideas about protein administration in renal failure. In 1949, he compared renal failure to heart failure,
i.e., the "work" of urea clearance done by the renal parenchyma being similar to the effect of exercise or "work", on a decompensated heart. He showed that after a 75% nephrectomy on rats, high protein diets produced a hypertrophy of the remaining renal tissue, just as cardiac failure produced hypertrophy of cardiac muscle. Following the 75% nephrectomy, the rats fed high-protein diets had a high mortality rate with persistent proteinuria, hematuria, cylinduria, and severe azotemia. The low-protein group had less azotemia and a better survival rate (19).

Despite the hypertrophy of the kidney, patients do better clinically on the low-protein diet (19).

In a junctural sense, the important work of Rose and Dekker should be re-mentioned here. Rose established the minimal daily requirements of the essential amino acids. Working together, they proved that urea could be utilized as a source of protein for catabolism in rats if non-essential amino acids are withheld (4,5).

Herndon and co-workers, in 1959, performed an experiment with rather startling results. They studied six patients, five with chronic renal failure and one normal volunteer, on a metabolic ward. It was demonstrated that the daily requirement of protein for the patients with renal failure was 0.54-0.70 gm/Kg/d, while that of the normal subject was indeed less than this, i.e., 0.49 g/Kg/d.

The reason that the normal person needed less basal protein than the nephrectics was not clear, but they postulated that either a general reduction in the effectiveness of protein utilization or perhaps an increased requirement for a given amino acid was responsible (20).
Carmelo Giordano expanded the urea-sparing principle to human use in 1963. On a metabolic ward, he followed eight patients with severe renal disease (six were uremic) and one normal volunteer. He showed by meticulous nitrogen balance studies that an essential amino acid diet of two g/d was not capable of producing nitrogen balance, in any subject. However, when ammonium salt, glycine, glutamic acid, or urea was added to their diet, positive nitrogen balance or equilibrium was obtained. The importance of this was that urea, whether fed (exogenous) or the result of altered excretion (endogenous), could be utilized for the synthesis of non-essential amino acids if the diet is adequate in calories and the minimum daily requirement of amino acids is provided.

Using himself as a control, he demonstrated that a condition of negative nitrogen balance existed when following a diet of essential amino acids plus glycine. This reverted to nitrogen equilibrium or positive nitrogen balance when either tribasic ammonium citrate or urea (both oral and subcutaneous infusion) was added to the essential amino acid regimen. This indicated that the use of exogenous ammonium and urea for production of non-essential amino acids and consequent nitrogen equilibrium was feasible in humans.

The uremic patients, fed only essential amino acids, had a decrease in symptoms, and in five of six, the nitrogen balance changed from negative to positive. This indicated use of endogenous urea for production of non-essential amino acids (21).

To determine that the urea, and not the ammonium, was being utilized, an ingenious experiment was devised. It was known that urease produced by the bacteria of the intestine decomposed urea to ammonium (22). The two non-
uremic patients were given paromomycin, an oral antibiotic which "sterilized" the bowel, and therefore eliminated the bacterial source of urease. Stool cultures were negative, which proved that no urease-containing bacteria were present. One of the patients never-the-less maintained positive nitrogen balance on an amino acid and urea diet. This experiment proved that urease was not necessary for urea utilization (21).

Giovannetti and Maggiore, in 1964, published a new diet for the dietary treatment of severe chronic uremia. They postulated that the difficult requirements to be met in treating renal failure were first to lower the production of protein catabolites and secondly to prevent body protein wastage. They began treatment with a period of "priming", i.e., a diet almost totally deficient in protein. This, they felt, depleted the amino acid pool within the body and enhanced subsequent protein anabolism from endogenous urea.

The "basal protein-deficient diet", as they called it, contained 1.0-1.5 g of nitrogen per day. 2000-3000 calories per day were supplied to provide "urea sparing". A "bread substitute" was designed from deglutenized maize (wheat) starch, palm oil, and water, and since this could be used to make spaghetti so dear to the hearts of their Italian patients, the term "spaghetti diet" was used to describe the diet.

In this initial priming period, (usually 20 days), blood urea nitrogen levels fell and toxic symptoms were eliminated. However, their patients were now in a state of negative nitrogen balance, and so a 20 g/d essential amino acid supplement was offered. This converted the nitrogen balance to positive, and the BUN remained constant. Furthermore, uremic symptoms did not recur and five of the eight patients improved enough to return to work.
After a period of time (60 to 90 days) with the essential amino acid supplement diet, the addition of one or two eggs per day, because of their high biologic value, was allowed. Again the BUN remained stable and toxic symptoms were not observed (23).

Shaw, et al, from Manchester, in 1964, modified the Giovanetti diet to suit the tastes of 20 English patients. Their diet contained approximately 18 g protein/d, (0.26 g/Kg/d) with adequate essential amino acids (except methionine, which was added). The bread allowed on the diet was the type given to phenylketonurics.

The English workers used only one diet, unlike the "spaghetti diet" which had "basic protein-deficient" and "protein-containing" phases. The patients began on the low-protein diet immediately, with no priming, because co-operation was better.

These clinicians noted a gradual fall in blood urea, the level of the final stable value being inversely proportional to the glomerular filtration rate. The clinical response was so remarkable that five patients with glomerular filtration rate of less than 3 ml/min returned to work. The BUN becomes stable after a period of time on the diet, and even those with stable BUN's as high as 200 mg/100 ml were asymptomatic while on the diet (24).

Berlyne, Shaw, and Nilwarangkur, in 1965, published a follow-up of the 20 patients just mentioned and added six more. They noted that in those patients with a glomerular filtration rate greater than 1.5 ml/min who were able to take the entire diet there was some beneficial response, especially a loss of gastrointestinal symptoms, and a longer life. In some patients, hemodialysis
was needed to ameliorate nausea and vomiting in order to commence the diet.

Although their renal function did not improve, those participants with glomerular filtration rates of over 3 ml/min obtained relief from all symptoms of uremia. These patients had an improvement in the anemia and, if transfusions were required, the frequency was less (25).

Symptomatic hyperkalemia and acidosis were noted in both studies. In the later paper a terminal clinical syndrome of epistaxis, bleeding, agitation, and no gastrointestinal symptoms was described.

Berlyne, in 1966, reported on 50 patients with up to two years of treatment with the Anglo-Saxon form of the Giovanetti diet. He listed the following indications for beginning this diet: uremic symptoms, BUN greater than 200 mg/100 ml or glomerular filtration rate of 5 ml/min or less. Although documentation is poor, the survival rate seemed to be much increased. He listed "polycystic disease" as having the best prognosis in his experience, and "hypertension with chronic failure" as having the poorest prognosis.

In all cases, the BUN decreased and, if glomerular filtration rate was greater than 3 ml/min, the BUN could return to normal values. Serum creatinine did not decrease. (This was recognized as a measure of endogenous metabolism and therefore not influenced by diet.) A serum creatinine level of greater than 20 mg/100ml usually preceded death by a matter of one to two weeks (26).

Workers in the United States lagged behind the Europeans. In 1967, Franklin et al reported on a 21-month study on 34 patients. He adopted the dietary principles of the Italians and English and applied them to American tastes. His patients were managed on a 20 g protein/d; 14 g from eggs and
the remainder from low biologic value vegetables. High caloric intake was demanded of the patients part of which was guaranteed by a bread made of protein-free starch. PKU bread was sometimes substituted.

Seven patients were unable to follow the diet because of unpallatability. 23 of the 27 remaining had relief of uremic symptoms. Three who failed had urea clearances of less than 1 ml/min, and the fourth had intractable heart failure. Eleven returned to work, and three resumed household duties. Serum urea nitrogen levels dropped an average of 50%. Urine volume decreased from 1,725 cc to 1,475 cc presumably because of decreased osmotic load delivery to the kidney due to the fall in serum urea nitrogen. As the glomerular filtration rate continued to decrease, because of the progression of renal damage, the urinary output fell further and uremic symptoms recurred after a fall below 1000 ml/d.

In general, those who did not obtain a fair amount of relief either had a urea nitrogen clearance of less than 1 ml/min or could not adhere to the diet. Those who achieved a good response had urea nitrogen clearance above 1.5 ml/min and were able to follow the diet well (27).

An anuric teenage Negro boy with hereditary nephritis was reported in September 1967 by Levin and Winkelstein who kept him alive for 15 months with the use of a special low-protein "electrodialyzed whey." Whey is a by-product of the cheese industry and heretofore a waste material usually fed to swine. By decaseinating whey, Wyeth laboratory workers prepared a powder composed of lactalbumin with a biologic value of 100. Dialysis made it low in electrolyte content.
The diet has four parts: a low-protein, low-electrolyte, high-caloric beverage made of whey; a low-protein, low-electrolyte, high-caloric exchange list; "free" high-fat and high-carbohydrate foods; and controlled volume of fluids.

The beverage is composed of 100 gm of electrodialyzed whey, 75 gm vegetable oil, 150 gm Dextri-Maltose (a high carbohydrate powder used in some baby formulas) and 300 gm water. The whey provided 26 gm of protein a day. This mixture was blended with artificial flavors to be served as a frozen custard or milk shake. It provided about 1650 calories per day, as the main "bulk" of the diet (see Appendix).

The exchange diet (see Appendix) provided variety. The patient was instructed to select exchanges not to exceed 7 gm protein, 600 ml water, 7 mEq sodium, and 25 mEq potassium.

The "free" food list contained high-carbohydrate or high-fat snacks or additions to the meals. The patient was to choose freely from this list (see Appendix).

The "controlled extra fluid" was calculated to allow water or electrolyte- and protein-free fluids according to the patient's fluid needs and activity. Corrected to allow for water or oxidation in the anuric case, it amounted to 45 ml/100 calories.

Prior to treatment with the diet, the patient was very ill with anasarca, acidosis, a steadily rising BUN, and a decreasing urinary output. He had undergone ten transfusions in the six months prior to beginning the diet. One month before the diet was started, he was admitted to the hospital with oli-
guria, hypertensive encephalopathy, congestive heart failure, and uremia. Two peritoneal dialyses were done. Just after he was started on the diet, he became anuric.

In the subsequent 15 months, the boy was dialyzed electively, first at 14 day and later 21 day intervals. Hypertension was controlled without the use of medications and he required only seven more transfusions. Muscle development and nutritional status improved and blood PH returned to normal. The most remarkable point is that although anuric he progressively rehabilitated himself, finished high school, and went to work in his father's cotton fields (28).

In October, 1967, a conference on the *Nutritional Aspects of Uremia* was held in Scottsdale, Arizona. Dr. Levin reported that eight patients were on the whey diet with seven being dialyzed every 21 days and one, whose creatinine clearance was 3-4 ml/min, being maintained by diet alone. All had been failures on conventional low-protein diets. The average duration of treatment was 7 1/2 months.

Four other patients died while on the diet. Three had malignant hypertension, uncontrollable by diet or drugs. The forth had severe hypertension, generalized arteriosclerosis, and hypertensive cardiovascular disease. Three of these were unable to adher to the diet and none had been rehabilitated. Two patients were on the diet 6 and 3 months, and after discontinuance, died in 10 days and 6 weeks, respectively (29).

**Results of Low-Protein Diets**

Assessing the results and effectiveness of the various low-protein diets is
complicated, and there are many aspects to consider. Adherence, longevity, and clinical and chemical changes will be considered briefly.

**Adherence** to the various diets has been difficult for some patients because of the unpalatability or monotony of the diets. Franklin noted a 22% incidence of inability to follow his diet, in most cases because of a dislike for the bread used (27). Analysis of Giovanetti, Berlyne, and Shaw's groups show 10-25% inability to accept the diet (23, 24, 25). Levin noted that four of sixteen patients could not tolerate the whey diet (29).

The reasons people cannot accept these diets are many and has not been studied in depth. Abram noted, in a subjective study of the problem, that the chronic renal failure patient is frequently observed to have rather marked personality changes and even psychotic symptoms. He suggested the need for psychiatric evaluation before long-term management was undertaken. He felt that closer psychiatric management might produce better results from the standpoint of strict adherence to dietary control (30).

**Biochemically,** every investigator has shown the blood urea nitrogen to decrease on the low-protein diets. Creatinine level does not decrease on the diets. Creatinine production is endogenous and cannot be influenced by dietary management (2). A serum creatinine level greater than 20 mg/100 ml is usually a harbinger of death, often within one to two weeks (27).

Retained urea per se is not entirely responsible for the toxic symptoms of uremia. Uremic patients undergoing dialysis have improved symptomatically whether the BUN is lowered or not (6). Perhaps another of the "azotes" is responsible for the symptoms. As Merrill says:
"Protein intake should be limited not because of the urea moiety alone, but because of other nitrogenous metabolites, and particularly because of the acid residues which cannot be excreted." (6).

However, as Merrill and other workers have shown, the low-protein diets do relieve many of the complaints of uremia. They also lower the urea nitrogen. They may also promote a reduction of the moiety (or moieties) that is responsible for this symptom complex. So far, the BUN is the measureable ingredient which is used to assess efficacy of the diet, and the relief of symptoms may be a fortunate happenstance (6).

Symptoms lessen and may disappear while on a low-protein diet. Berlyne's 50 patients showed a marked decrease in anorexia, nausea, vomiting, singultus, diarrhea, drowsiness, and apathy, and felt better. In general, if the glomerular filtration rate was greater than 2 ml/min, they went back to work. All symptoms disappeared with glomerular filtration rate greater than 3 ml/min (26). Of Franklin's 27 patients eleven went back to work and three resumed household duties. Again, as in Berlyne's case, the higher the glomerular filtration rate, the better the clinical result (27).

Many workers stress the necessity for intermittent peritoneal dialysis or hemodialysis when symptoms such as nausea and vomiting make it impossible to eat. Good results on the diets require ingestion of the entire amount prescribed (27, 31).

The effect of low-protein diets on longevity is difficult to evaluate. From Smith in 1927 to Levin in 1967, no reporter has failed to express the opinion that patients "do better clinically" while on a low-protein diet. Toxic symptoms decrease and a sense of well-being is established. Many subjects returned to a normal life (24, 32). However, what effect the diet may have on increasing
the length of life awaits a well-controlled prospective study. At any rate, the diet is merely a form of management, and can do nothing to combat progressive renal deterioration (27).

However, one point is worth mentioning. Chronic hemodialysis programs are increasing in number (33). Recent advances in the immunological and surgical aspects of renal transplantation are making this a realistic answer to the problem. It makes good sense to adhere to the best management of the uremic syndrome possible while a patient awaits placement on such a program (6,27).

Complications of the Low-Protein Diet

There are many complications of renal failure which are well known, e.g., secondary hyperparathyroidism, neuropathy, metastatic calcification, encephalopathy, pericarditis, and renal osteodystrophy (3). These will not be dealt with here. However, there are several complications that have been noted since the advent of the Giordano-Giovenetti diet. It is not known whether these are secondary to the low-protein diet or emerge with the possible lengthening of life with better management. The discussion that follows will be limited to some of these problems.

Berlyne was impressed that patients on the Giordano-Giovanetti diet showed signs of agitation and bleeding, but had no gastrointestinal symptoms. Death could be expected within two weeks after this phase developed. He called this "modified terminal renal failure syndrome" (25). Franklin, however, found this not to be true in his 34 patients (27).

Shaw (24) and Berlyne (25) noted a tendency for patients on their low-protein diets to develop severe acidosis. Franklin, however, did not encounter this
problem. He may have avoided it by prophylactic supplementation of the diet with calcium carbonate (27).

Anemia has been mentioned earlier. Most patients had either a decrease in anemia or a decreased frequency of transfusions. As Franklin noted, the better the renal function, the better the response of the anemia. With creatinine clearances exceeding 1.9 ml/min, he found the anemia improved on his diet (27). Shaw and Giovanetti had similar results (24,23).

Hyperkalemia did occur in both Berlyne and Franklin's patients both with and without acidosis respectively. This was treated with oral sodium polystyrene sulphanate (Kayexalate), a sodium-for-potassium exchange resin (27,28). However, Berlyne reported three cases in which heart failure, edema, and severe hypertension developed secondary to sodium overloading from the use of Kayexalate (34). He suggested the use of a calcium-exchange resin instead, and noted no problems with hypercalcemia (35).

A few patients with chronic renal failure who are not diabetic may show elevations in blood sugar. Morgan suggested in 1967 that these patients should be given a diet high in carbohydrates and low in protein even if insulin is required (17).

Marked proteinuria, in renal failure, even patients who are not nephrotic, may produce severe protein wasting. Berlyne and later Franklin had a practical answer to this. In any patient with a greater than 3 g/d urinary protein loss, \( \frac{1}{2} \) egg per day was added to the diet for each 3 g protein lost (25,27).

Edema despite modest salt intake was noted by Franklin. This may have been due to decreased solute load secondary to the fall in BUN attributed to the diet which might allow greater sodium reabsorption. However, the edema ap-
peared only when urea clearance was less than 1.9 ml/min, so there may be a critical glomerular filtration rate below which salt and water are retained (27).

All of the diets are low in vitamins. Any good maintenance multiple vitamin should remedy this. Maddock felt that Levin's whey diet was low in L-phenylalanine and DL-methionine, and has added this to the formula (35).

Berlyne stated in 1966, that several of his patients were suffering from "red eyes". This was due to "ocular metastatic calcification." The high serum phosphate levels common to renal failure were complicated by the high serum calcium levels of secondary hyperparathyroidism with an increased calcium:phosphorus ratio. This was remedied by the administration of aluminum hydroxide gel, which lowered the serum phosphorus levels (26).
CASE PRESENTATIONS

Case I, W.C., a white female who was 36 years old when first seen at U.N.H. on May 20, 1957, for hypertension, albuminuria, edema and vomiting in the 8th month of her ninth pregnancy. She had never consulted a physician prior to this pregnancy. She stated that no problems had been encountered in any previous pregnancies, although her husband recalled that during the last two or three there had been some periorbital edema. No other history suggesting renal disease could be elicited.

She was treated with bed rest and felt better, although her blood pressure increased; her urinary protein content was 4.9 grams/24 hrs. The labor began on May 26th. Oliguria, gross hematuria, and the absence of fetal heart tones were reported on the 27th, when an amniotomy was done. Extreme electrolyte imbalance was noted (Na 114 mEq/L; K 4.6 mEq/L; CO2 4.8 mEq/L; Cl 102 mEq/L; BUN 58 mg/100 ml). She was confused and then comatose and a stillborn male infant was delivered. Partial placental abruption was found.

The electrolyte problems were treated and she improved. On dismissal, the urine output was normal; her blood pressure was normal; PSP 26%; NPN 58 mg/100 ml; BUN 36 mg/100 ml.

She was readmitted on 11/11/58, for tubal ligation. At that time, the blood pressure was 150/98 mm Hg; BUN 25.5 mg/100 ml; NPN 45 mg/100 ml; serum creatinine 2.3 mg/100 ml; Hb 13.4 gm/100 ml; serum K, Cl, Na, CO2, chest x-ray, and ECG were within normal limits.

She was discharged to the care of her referring physician on a low-salt, low-protein diet.
Her third admission, at age 47, was on June 19, 1967, after vomiting for one week. Adult-onset diabetes had been diagnosed seven years previously. She was not on medication for this. Her blood pressure was 150/102 mm Hg and she had a sallow appearance with darkened skin. The thyroid gland was palpable but not enlarged. Grade IV/VI systolic blowing murmur was best heard at the apex. There was no cardiomegaly or edema and the remaining physical findings were normal. Urinalysis showed Sp. Gr. of 1.008 and 0-2 WBC/HPF. Hb was 8.3 gm/100 ml; Hct 25%; BUN 153 mg/100 ml; FBS 118 mg/100 ml; serum creatinine 16 mg/100 ml; CO2 17 mEq/L; pH 7.31; Na 134 mEq/L; K 4.1 mEq/L; Cl 94 mEq/L; Ca 4.8 mEq/L. Her chest X-ray showed no active lung or heart disease, but there was generalized bony demineralization and collapse of T11. ECG findings were 1° AV block; LVH; and non-specific T wave changes consistent with myocardial ischemia, pericarditis, or myocarditis. Her creatinine clearance was 4 ml/min. Proteinuria was 2.7 g/24 h. Urinary Na and K were 99 mEq and 29 mEq/24H.

She was given two units of blood with a resultant rise of Hb to 10 gm/100 ml, and fall in BUN to 85 mg/100 ml and serum creatinine to 10.8 mg/100 ml. Inability to concentrate urine was noted. She was dismissed on a 30 g/d protein diet and 6 g/d salt restriction on 6/29/67.

Her fourth admission was 7/18/67, for weight gain and edema, and possible dialysis. The physical exam showed a blood pressure of 210/120 mm Hg, 3+ ankle edema, and ascites. Lab tests included: Hb 6.4 gm/100 ml; Hct 21%; FBS 84 mg/100 ml; BUN 108 mg/100 ml; creatinine 12 mg/100 ml; uric acid 8.4 mg/100 ml; CO2 16 mEq/L; PH 7.22; Na 138 mEq/L; K 5.2 mEq/L; Cl
104 mEq/L; Ca 4.5 mEq/L; PO₄ 8.2 mEq/L. Creatinine clearance was 2.6 ml/min. 24 H urine outputs of Na, K and protein were 110 mEq, 39 mEq, and 3.1 g respectively. Dialysis was not done.

The chest X-ray revealed cardiomegaly, prominent pulmonary vasculature, and probable slight bilateral pulmonary effusion. She lost 2.7 Kg at bedrest and left the hospital on 8/2/67 again on a 30 g/d protein diet. She was digitalized on an outpatient basis.

Her fifth admission was on 8/17/67, for evaluation of hypertension, transfusion, and probable dialysis. Blood pressure was 200/120 mm Hg. Abnormalities on physical examination included; scattered ecchymotic areas on the skin, a puffy face, uremic odor to the breath, "half and half" nails, cervical vein distension at 30°, increased ascitic fluid, palpable liver, 2+ ankle edema, an unsteady gait, and increasing mental dullness. The laboratory reported: Hb 5.6 gm/100 ml; pH 7.22; CO₂ 14 mEq/L; Na 138 mEq/L; K 5.6 mEq/L; Cl 103 mEq/L; BUN 138 mg/100 ml; creatinine 13.5 mg/100 ml.

Two transfusions raised her Hb to 7.8 gm/100 ml and the BUN rose to 145 mg/100 ml. Digitalis, methyldopa, furosemide, spironolactone, allopurinal, sodium bicarbonate, and sodium polystyrene sulfonate were used. Diuresis did not occur so for the first time peritoneal dialysis was done. After this, the BUN was 65 mg/100 ml; pH 7.39; Hb 7.5 gm/100 ml; diuresis and decreased nausea were achieved and she was discharged on 9/3/67.

Twenty-four hours after dismissal she returned with extreme weakness in her legs and arms, dizziness, and urinary frequency (every three minutes). She told of a neighborhood "welcome home" feast of meat, sweet corn, tomatoes,
and watermelon. Neurological exam revealed hyperreflexia and tetany. The serum K was 7.4 mEq/L; BUN 62.5 mg/100 ml; creatinine 9 mg/100 ml and the ECG revealed peaked T waves. Sodium polystyrene sulfonate and naso-gastric suction decreased the serum K to 4.5 mEq/L with rapid amelioration of symptoms and she was discharged on 9/6/67.

The seventh admission was from 9/18/67 through 10/7/67 for nausea, vomiting blood, and abdominal pain. Physical examination and chest x-ray revealed congestive heart failure. Her blood pressure was 190/90 mm Hg; Hb 7.1 gm/100 ml; BUN 135, 148 mg/100 ml; creatinine 15 mg/100 ml; pH 7.27; Na 137 mEq/L; K 6.3 mEq/L; Cl 100 mEq/L; Ca 4.8 mEq/L; uric acid 7.0 mg/100 ml. Peritoneal dialysis was started and continued for 52 exchanges as a Pseudomonas species was cultured from several early exchanges. Later peritoneal fluid and blood cultures were negative and she remained afebrile throughout the dialysis. The BUN dropped to 20 mg/100 ml at one time, but rose to 67 mg/100 ml. On termination of dialysis the creatinine was 12.2 mg/100 ml. She was started on the whey diet on 10/7/67, the day of dismissal. There was no salt restriction. Her medications included methyldopa 250 mg, three times daily, a multiple vitamin, one tablet daily, calcium gluconate 2 g three times daily, sodium polystyrene sulfonate 15 g daily, and propoxyphene hydrochloride for pain.

She was followed in the Renal Research Clinic from 10/12/67 to 11/30/67. She was a docile lady, and readily followed the diet, in fact stating that she "liked the whey". Her blood pressures were better controlled than previously being 130/75, 144/110, 170/106, 158/100, 141/94, and 160/90 mm Hg on weekly to biweekly clinic visits. Her former complaints of nausea, vomiting, twitching, dizziness, and blurring of vision did not recur and she was able to
do some housework. Serial BUN's were 90, 95, 97.5, and 108 mg/100 ml. Uric acid's were 9.4, 8.4, 7.6, 7.4, mg/100 ml, as she was started on allopurinal 100 mg twice daily. The Hb dropped from 9.5 to 7.0 gm/100 ml. Despite a fluid intake limited to 550 ml orally per day, she progressively gained 17 ½ pounds; although not symptomatic for congestive heart failure.

Her **eighth admission** (12/7/67 to 12/12/67) was for re-evaluation of renal status and possible dialysis. She was symptom free but had edematous eyelids and 2+ pretibial edema. The neurological examination was normal. Hb was 5.6 gm/100 ml; creatinine 34 mg/100 ml (probably erroneous); pH 7.28; CO₂ 20 mEq/L; Na 134 mEq/L; K 4.5 mEq/L; Cl 94 mEq/L; Ca 4.8 mEq/L; uric acid 10.8 mg/100 ml; creatinine clearance 1.5 cc/min; 24 H urinary Na and K were respectively 74 mEq and 9 mEq. She received four units of blood with an increase in Hb to 8.5 gm/100 ml and decrease in BUN to 97.5 mg/100 ml. The ECG was normal. The azotemia was felt to have a pre-renal element and no dialysis was done. After initial loss in weight, she gained 2 ½ pounds prior to discharge.

Her **ninth admission** (12/16/67 to 12/21/67) was for dyspnea, increased ankle edema, and ascites. The blood pressure was 210/130 mm Hg; Hb 10.5 gm/100 ml; BUN 113 mg/100 ml; WBC 16,100/cc; creatinine 15.5 mg/100 ml. Although she had pneumonitis, antibiotics were withheld because of her nearly absent renal function. Peritoneal dialysis was performed, with BUN dropping to 52.5 mg/100 ml and creatinine to 8.7 mg/100 ml. She lost five pounds, improved clinically, and had a normal white count on dismissal.

The **tenth and last admission** was 12/24/67. She died on 12/30/67. She complained of dyspnea for three days, left chest pain, cough, 7.5 pound weight
gain, scanty urine, and fever. Her temperature was 37.4°C. Her pulse was paradoxical from 60 to 100. Blood pressure was 98/60 mmHg, with systolic paradoxes from 130 to 98. There was dullness on percussion of the left side of her chest. The PMI was in the left 5th intercostal space at the anterior axillary line. The abdominal was markedly distended, and massive pitting edema was present. The Hb was 11 gm/100 ml; WBC 10,000/cc; BUN 97.5 mg/100 ml; Pro Time 12.8 sec (control 11.4); bleeding time 2 minutes; clotting time, ten minutes. The electrolytes were remarkably normal, except K of 3.6 mEq/L. The ECG revealed ST-T depression and inversion compatible with acidosis, digitalis effect, pericarditis or ischemia. The chest X-ray suggested pericardial effusion. She was put on continuous O₂, but dyspnea persisted. Tamponade was suspected so pericardiocentesis was done and 140 cc of bloody fluid was obtained. Her blood pressure rose to 130 mm Hg with 30 mm of paradox.

She improved clinically, but serial urine outputs (per 24 H) were 178, 104, 150, 115, 159 ml. On the 29th, a repeat pericardiocentesis returned 300-400 cc, and the blood pressure rose to 144 mm Hg, still with 30 mm of paradox. The BUN was 97.5, 105, 118, 139, and 145 mg/100 ml (26th through 30th).

On the 30, peritoneal dialysis was begun with a somewhat bloody fluid returning immediately from the catheter. In 20 minutes, the pulse and blood pressure were unobtainable and she was unresponsive. Resuscitation affects were unsuccessful.

Significant gross autopsy findings were hemoperitoneum of 2500 cc., puncture wound of the superior mesenteric vein, hemopericardium (150 cc) and pericarditis, bilateral pleural effusions (500 cc), and double pelvis of right kidney. The heart weighed 420 grams. The right ventricle was 7 mm thick and the left
15-17 mm thick. Both kidneys were shrunken, and the capsules stripped only with difficulty. The right kidney weighed 52 gms and the left 15 gms. The renal cortex was shrunken bilaterally. Many subcapsular cysts were noted on the right kidney. Both right and the left renal pelves were normal. Amyloidosis of the kidney, spleen, liver, heart, and adrenals was noted. Microscopic diagnosis showed chronic pyelonephritis. Both kidneys showed only a few hyalinized glomeruli present. These were also areas of chronic interstitial fibrosis and chronic inflammatory infiltrate.

The cause of death was hemorrhagic shock secondary to hemoperitoneum due to laceration of the superior mesenteric vein during the attempted peritoneal dialysis. The chronic renal failure was thought due to chronic pyelonephritis, but this was difficult to ascertain because of the marked degree of hyalinazation of the glomeruli. Amyloidosis (probably primary) was also a contributing factor.

Case II, W.M., was a 75 year old Caucasian male with chronic renal failure probably due to chronic pyelonephritis. He was started on the electrodialyzed whey diet two weeks prior to his last admission, after one peritoneal dialysis. He had previously been unable to follow a standard low-protein diet.

His final admission on 12/21/67 for progressive weakness, confusion, and lethargy. The admission blood pressure was 190/80 mm Hg and the pulse was 80/min. He was a sallow, lethargic, and nearly deaf, elderly male with Grade I Keith-Waggener-Barker retinopathy. His chest was normal on percussion and auscultation. The heart examination revealed a PMI 3-4 cm lateral to the left midclavicular line and a sinus rhythm without murmurs. The neurological exam was normal.
Admission lab values: RBC 2.35/cc; Hct 27%; Hb 8.6 gm/100 ml; BUN 126 mg/100 ml; serum creatinine 15 mg/100 ml; CO₂ 16.0 mEq/L; Cl 96 mEq/L; Na 128 mEq/L; K 4.8 mEq/L; pH 7.230; urinary Na and K were 32 and 47 mEq/L, respectively. The urine sediment contained 20-25 RBC/HPF, 40-45 WBC/HPF and was packed with bacteria. The serum calcium, phosphorus, and uric acid were normal.

He was given NaCl supplement for his hyponatremia and cephalothin for an Aerobacter urinary tract infection. He underwent a peritoneal dialysis for progressive acidosis, azotemia, edema, and near oliguria (600 ml/d). The serum creatinine climbed to 19 mg/100 ml. He was noted to have a bilateral pleural effusion on chest x-ray although he was asymptomatic. He had one episode of posterior pharyngeal bleeding, estimated at 75 cc. He became depressed and told his attendents he was dying, and despite two more peritoneal dialyses and two units of blood, suddenly became tetanic. Despite 30 mEq NaHCO₃ and 5 gm calcium gluconate, he lapsed into coma and died. Postmortem examination was not done.

Discussion:

Graphs of the two cases presented are included in the Appendix.

Case I was in the hospital one-half the time for the five months prior to starting the whey diet. Once on the diet, she spent two symptom-free, useful months at home. Fatigue, nausea, vomiting, somnolence, twitching, and lability, frequent complaints earlier in the disease, disappeared for these two months.

In Case I, BUN values were around 140 mg/100 ml predialysis and 60 mg/
100 ml postdialysis prior to starting the diet. After starting on the diet, the rise in BUN was slower, being "stable" at 90-110 mg/100 ml. Hypertension was more easily controlled while on the diet (see Case Presentation). She was easily controlled on methyldopa 250 mg, three times daily. Previously, control had been difficult, as many as four agents at once failing to achieve satisfactory blood pressures. Transfusion frequency was reduced, although four units were required in December.

Renal osteodystrophy was observed on chest X-ray (demineralization and collapse of T11—see Case Presentation.) However, calcium and phosphorus imbalance was not observed while on the electrodialyzed whey diet. Calcium gluconate administration may have prevented this.

Hyperkalemia was a problem, although symptoms were only noted on one occasion (after the "welcome home" dietary indiscretion). Daily administration of sodium polystyrene sulfonate was felt to be responsible for maintenance of reasonable potassium balance.

This patient was a "salt-loser" (urinary losses of sodium approximately 100 mEq/d) and was not restricted in salt consumption. Normal serum sodium values were maintained. This "freedom at the salt-shaker" may have contributed to the edema fluid, although azotemia and albuminuria also were undoubtably important (37).

Pericarditis occurs in approximately half of the cases of long-standing uremia; effusion is common and may rarely produce tamponade, as in this case (1). Levin noted that two of his eight patients exhibited myocarditis with "toxic myocardopathy" but this reverted in both cases to normal with dialysis (29). More cases are necessary to determine if pericardial effusion and tamponade are more common
with this diet.

Renal collapse in this case was heralded by severe oliguria and rapidly increasing azotemia. One might suspect that the tamponade contributed to these although the autopsy findings of almost absent glomeruli probably is an adequate explanation.

Case II is not as promising. Azotemia could not be controlled by the electrodialyzed whey diet, even with dialysis. Perhaps the serum creatinine of 19 mg% and the agitation and bleeding this patient displayed were indeed "harbingers of death" (as earlier noted by Franklin and Berlyne) (27,25)

Four other patients were started on this diet. Two of these could not maintain the diet. One expressed a desire for more "solid" food, the other simply found the beverage unpalatable. Both patients had severe renal disease and died shortly. The two other patients presently on the diet total less than one month experience and are therefore not included.

A constant diet of any kind indeed would become monotonous. They whey is difficult to emulsify, and an electric blender is necessary. The Dextri-Maltose imparts an "overlysweet" taste to the beverage. The author substituted dextrose hydrous for the Dextri-Maltose with some decrease of sweetness and increased palatability resulting.

One patient also heated the beverage to make a hot pudding and by alternating with the cold servings, eliminated some tedium of the dietary routine.

One unexperienced with the use of this diet will undoubtedly spend hours calculating correct allowances. Perhaps the following discussion will help expedite these calculations.
Calculation of the diet

To facilitate calculation of the dietary requirements for each patient, the author prepared a "fill-in-the-blank" chart. A sample of this chart for Case I is found in the Appendix.

First, one determines the "ideal weight" from the patient's height and body build (46 Kg in this example). The ideal body weight was used so that positive metabolic balance could be attained; allowances should be made for obese patients so that a state of starvation does not exist.

Next, using this weight, one calculates the daily protein requirements based on an estimation of 0.5-1.0 gm/Kg. In Case I, this was 23-46 gm/day. The mean value, 35 g, was used. There are ten gms in the "exchange diet and extra fluids", so this is subtracted from the total daily requirement (i.e. 35-10 = 25.) This amount will be provided in the whey formula.

3½ ounces of whey (dry measure) equals 100 gms and this supplies 26 gms of protein. Therefore, when it is determined how much protein the patient needs, one can calculate his daily amount of whey. In this case, 25 gms of protein was needed, so the patient was instructed to mix 3½ ounces of whey into the recipe for each day's allowance of formula.

In calculation of the protein requirement for their cases on the electrodialyzed whey diet, Levin and Winkelstein used Herndon's established requirements for patients in chronic renal failure as was noted in the text of this review (20).

The caloric requirement is based on 50-75 cal/Kg. This was 2300-3450 calories per day in the case presented. 1650 calories are supplied by the formula and 650-1800 calories in the exchange diet and extra fluids, which would provide 2300-3350 calories per day. Should the caloric requirement be greater than can
be provided by the calculation, one simply allows more exchanges (at the expense of increasing low biologic value protein intake), encourages "free foods", or increases the Dextri-Maltose in the formula.

Water requirements were based on the calculations of Darrow and Pratt (38). This was 45 ml/100 cal. Using the calculated caloric requirement, this amount was 1030-1550 ml for the sample case. Arbitrarily, the arithmetic mean was used, i.e. 1290 ml/day. There are 300 ml in the formula and 600 in the exchange diet, leaving 390 ml in "extra fluids" per day.

Weight gain, urinary output, and edema are charted at the patients return visits. Such variables as activity, fever, and perspiration are taken into consideration, and the fluid intake is modified accordingly.

Sodium and potassium were calculated in a similar manner, based on caloric intake. The sodium intake was not excessive, but 15-25 mEq of potassium was provided in the exchange diet. This high intake is considered a necessary evil and the serum potassium was reduced with the judicious use of sodium polystyrene sulfonate (34).

The amount of sodium lost by the kidney will vary in each individual. Occasional patients lose a great deal of salt. The term "salt-losing nephritis" has been coined to describe this phenomenon. Whether this represents a specific tubular lesion or an exaggeration of the usual contingency with decrease in renal mass is not known (37). Therefore, some patients need salt restrictions while others do not. The case in point did not require salt restriction, because of "salt-losing". Management of sodium can be delicate, especially in cases of concomitant congestive heart failure or hypertension.
Patients may need extensive studies of sodium excretion to determine the correct amount of salt restriction (34).

Summary and Conclusion

The first portion of this thesis contains a review of the literature concerning the evolution of the low-protein diet as used for chronic uremia. Some physicians prior to 1900 restricted protein, because it was thought that if urea was retained in the blood, it could be reduced by restricting it from the diet.

"How much" and "what kind" of protein to be used were the questions the 20th Century investigators set out to answer. At present, this still is not entirely known, but the work of Smith, Rose, Dekker, Giordano, Giovannetti, Shaw, Berlyne, Franklin, and Levin cited here as well as that of many others has given more insight into this complex matter. Sections on results of the various diets and complications peculiar to the low-protein diet follow. The final pages of the first section explains the electrodialyzed whey diet of Levin and Winkelstein.

The second portion of the paper reports on two patients who were maintained on the diet. In Case I, presented in detail, beneficial results were achieved. In the second case, the results were not as encouraging. There follows a brief commentary on certain practiced aspects of the use of the electrodialyzed whey diet.

Data are presented in support of the low-protein diet for use in chronic renal failure. It appears that an ideal diet is palatable, offers variety, and is easy for the patient and the physician to understand. It should be of high biologic value, and therefore, non-essential amino acids should be restricted. Calories should be adequate. Sodium, potassium, and other electrolytes should not be
excessive. Water content should be low.

The ideal quantity of protein needed is still not known. Some workers use 0.50-1.00 gm/kg/d, which roughly correlates with Herndon's measurements. The exact answer awaits further investigation.

Most authors have the clinical impression that the diets presented allow terminal uremic patients to live longer. However, a controlled prospective study is needed to determine the exact effectiveness of the diet.

The electrodialyzed whey diet seems to offer some advantages. It offers the patient greater variety than other diets. The beverage, while not a connoisseur's delight, is palatable. Improvements in taste can be made with experimentation. The exchange system makes the diet easy for patient (and physician) to understand. The whey diet has a protein biologic value of 100. It contains a few non-essential amino acids in the exchange lists. Calories are adequate. Sodium is low, but potassium is high and therefore sodium polystyrene sulfonate or another potassium-lowering agent must be used. Water content is adequate to excessive, in the author's opinion.

It is felt that the low-protein diet definitely is efficacious in the management of chronic uremia. It is also felt that the electrodialyzed whey diet has great promise as a tool in the hands of the physician who managing renal failure cases.
BIBLIOGRAPHY


APPENDIX

Comparison of BUN Levels on 30 gld Protein and Electrodialyzed Whey Diets:
Case I ----------------------------- 36

Comparison of BUN Levels on 20 gld Protein and Electrodialyzed Whey Diets:
Case II------------------------------- 37

Graphic Presentation of First Case of Electrodialyzed Whey Diet Treatment:-------------------------- 38

Calculation of Dietary Needs:
Case I ------------------------------- 39

Calculation of Dietary Needs:
Sample ---------------------------------- 40

Electrodialyzed Whey Diet:
Food Exchanges --------------------------- 41

Electrodialyzed Whey Diet:
Sample of Diet for One Day ------------------------ 42

Electrodialyzed Whey Diet:
Instruction Sheet ------------------------- 43

Giovannetti Diet ------------------------ 44

Kempner Rice Diet ---------------------- 45
Comparison of BUN levels on 30 g/d protein and low diets

01/31     5/31     6/30     7/30     8/31     9/30     10/31     11/30     12/31

KEY:

- Peritoneal Dialyses
- Hospital admissions
- Transfusions

CASE I
Comparison of Blood Levels on 36.2% Protein and EDW Diets

KEY:

Perineal Diatheses
Hospital Admissions
Transfusions

CASE II
GRAPHIC PRESENTATION OF
LEVIN AND WINKELSTEIN'S
INITIAL CASE REPORT OF PATIENT
ON ELECTRODIALYZED WHEY DIET
(NEW ENGLAND JOURNAL OF MEDICINE, SEPT. 21, 1967)

![Graph](image)

**Figure 1.** Serum Chemical Determinations, Urine Output and Weight of the Patient.
*The vertical lines represent periods of peritoneal dialysis, and transfusions are indicated by the arrows.*
RENAL RESEARCH:
ELECTRODIALYZED WHEY DIET

NAME: Case 1, W.C.  
DATE:  
AGE: 47  
BODY BUILD: Medium  
PRESENT WEIGHT:  
IDEAL WEIGHT: 46 Kg.  
HEIGHT: 4'10"  

DIAGNOSIS:  
CALCULATION OF DIETARY NEEDS:

<table>
<thead>
<tr>
<th>Dietary Component</th>
<th>Estimated Requirement/day</th>
<th>Calculated Requirement/day</th>
<th>Amount Provided to Patient/Day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>0.5-1.0 gm/kg</td>
<td>23-46 gm</td>
<td>25</td>
</tr>
<tr>
<td>Calories</td>
<td>50-75 Cal/kg</td>
<td>2300-3450</td>
<td>1450</td>
</tr>
<tr>
<td>Water</td>
<td>45 ml/100 Cal</td>
<td>105-165 ml</td>
<td>300</td>
</tr>
<tr>
<td>Sodium</td>
<td>0.2 mEq/100 Cal</td>
<td>1.0-6.9 mEq</td>
<td>1.0 mEq</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.4 mEq/100 Cal</td>
<td>9.2-13.8 mEq</td>
<td>0.6 mEq</td>
</tr>
</tbody>
</table>

To calculate Extra Fluids: Mean Calculated Water Requirement MINUS 900 ml is equal to the extra fluids per day. 
Then: 240 ml Water equals one cup. 
\[ \frac{240 \text{ ml}}{1 \text{ cup}} \approx \frac{240}{200} = 1 \text{ cup} \]

To calculate Whey per day: Mean Calculated Protein Requirement MINUS 10 (in Exchange Fluids) and interpolate to determine total protein and total whey per day. 
That is, 26 gm protein=100 gm Whey = 3 1/3 ounces. 
\[ \frac{35-10 = 25}{3} \approx 100 \text{ gm Whey} = \frac{3}{3} \text{ ounces} \]

Indicate on Patient's Instruction Sheet:
1. Number of ounces of whey to be used per day.
2. Number of cups of water allowed as extra fluids per day.
3. Whether the exchanges may or may not be salted.
Renal Research:
Electrodialyzed Whey Diet

Name: 
Age: 
Present Weight: 
Height: 
Date: 
Body Build: 
Ideal Weight: 

Diagnosis: 
Calculation of Dietary Needs:

<table>
<thead>
<tr>
<th>Dietary Component</th>
<th>Estimated Requirement/day</th>
<th>Calculated Requirement/day</th>
<th>Amount Provided to Patient/Day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>In Formula</td>
<td>In Exchange Diet &amp; Extra Fluids</td>
<td>Total</td>
</tr>
<tr>
<td>Protein</td>
<td>0.5-1.0 g/m²</td>
<td>10 g</td>
<td></td>
</tr>
<tr>
<td>Calories</td>
<td>50-75 kcal/kg</td>
<td>1450</td>
<td>650-1800</td>
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<tr>
<td>Water</td>
<td>45 ml/100 Cal.</td>
<td>300</td>
<td>600 +</td>
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<tr>
<td>Sodium</td>
<td>0.2 mEq/100 Cal.</td>
<td>1.0 mEq</td>
<td>3.0-5.0</td>
</tr>
<tr>
<td>Potassium</td>
<td>0.4 mEq/100 Cal.</td>
<td>0.5 mEq</td>
<td>15-25</td>
</tr>
</tbody>
</table>

To calculate Extra Fluids: Mean Calculated Water Requirement MINUS 900 ml is equal to the extra fluids per day. 
Then: 240 ml Water equals one cup.

To calculate Whey per day: Mean Calculated Protein Requirement MINUS 10 (in Exchange Fluids) and interpolate to determine total protein and total whey per day. 
That is, 26 gm protein=100 gm Whey = 3 1/3 ounces.

Indicate on Patient's Instruction Sheet:
1. Number of ounces of Whey to be used per day.
2. Number of cups of Water allowed as Extra Fluids per day.
3. Whether the Exchanges may or may not be salted.
ELECTRODIALYZED WHEY DIET:

FOOD EXCHANGES

Bread and Cereal List

Group I

Bread - one slice
Crackers - six saltines
Popped corn - ½ cup
Puffed Rice - one cup
Puffed Wheat - one cup
Shredded Wheat - two small biscuits
Sugar Cookie - three

Group II

Corn - 1/3 cup
Cooked Grits
Cooked Macaroni - 1/3 cup
Cooked Rice - 1/2 cup
Cooked Spaghetti - 1/3 cup
Cooked Oatmeal - 1/2 cup
Peas - 1/4 cup
Baked Potato - 1 small
Boiled Potato - 1 small or 1/3 cup
French Fried Potato - 1/4 cup
Mashed Potato - 1/3 cup

Vegetable List

Asparagus - 1/4 cup tips
Green Beans - 1/2 cup
Beets - 1/4 cup
Broccoli - 1/4 cup chopped
Brussel Sprouts - 1/4 cup
Cabbage - 1/2 cup
Carrots - 1/4 cup
Cauliflower - 1/2 cup
Celery - 1/3 cup
Cucumber - 1/3 whole
    (use only occasionally)
Onions - 1/3 cup
Lettuce - 1/4 cup
Spinach - 1/4 cup
Squash - 1/2 cup
Tomatoes - 1/2 cup
Tomato Puree - 1/2 cup

Fruit List

Apple - one cup
Applesauce - one cup
Apricots - 4 medium halves
Banana - 1/2 small
Cantaloupe - 1/4 to 1/3 cup
Cherries - 8 large
Fruit Cocktail - 1/4 cup
Grapes - 15 grapes
Grapefruit - 1/2 small
Lemon - 1/3 cup
Orange - 1/3 orange
Peach - two halves
Pear - two medium or 4 halves
Pineapple - one cup
Plum - two medium
Rhubarb - 1/2 cup
Raisins - 2 1/2 Tbsp.
Strawberry - 3/4 cup
Tangerine - 1 small
Watermelon - 1/3 cup
ELECTRODIALYZED WHEY DIET:
SAMPLE OF DIET FOR ONE DAY

BREAKFAST:
Toast (Bread and Cereal List, Group I)
Butter (Free Food List)
Jelly (Free Food List)
Applesauce (Fruit List)
Whey Formula

LUNCH:
Mashed Potatoes (Bread and Cereal List, Group II)
Butter (Free Food List)
Green Beans (Vegetable List)
Canned Peaches (Fruit List)
Whey Formula

Afternoon Snack: Jelly Beans (Free Food List)

SUPPER:
Cooked Spaghetti (Bread and Cereal List, Group II)
Tomato Puree (Vegetable List)
Raw Carrots (Vegetable List)
Butter (Free Food List)
Whey Formula

Evening Snack: Strawberries (Fruit List)
ELECTRODIALYZED WHEY DIET:
INSTRUCTION SHEET

I. Daily Whey Requirement

Mix ___ ounces of whey, 1 1/2 cups of distilled water, five Tablespoons of vegetable oil, and five ounces of Dextri Maltose (#2). This can be mixed with an electric mixer or a blender. Artificial flavoring (chocolate, vanilla extract, strawberry, Kool Aide) can be added as desired.

The mixture can be taken either cold, as a milk shake, or frozen, as a custard. It should be eaten in three equal servings, one for breakfast, one for lunch, and one for supper.

II. Food Exchanges

From the list of food exchanges provided, select the following each day:

One Exchange for Bread and Cereal List, Group I
Two Exchanges from Bread and Cereal List, Group II
Three Exchanges from Vegetable List
Three Exchanges from Fruit List

These Exchanges may - may not be salted.

III. Free Foods

Eat as much of the following each day as desired:
Sugar
Butter
Mayonnaise
Lard
Jelly
Plain Hard Candy
Plain Fondant Candy
Plain Gumdrops
Chewing Gum
Jelly Beans
Starch Jelly Pieces

IV. Extra Fluids

You may have as many as ___ cups of water each day.
Giovannetti’s Diet for Chronic Renal Failure
(Contains ess. amino acids, Low Na, Low K, Low Pro)
Lancet, 1964 Vol. 1, p. 1000-1003

Breakfast
2 boiled eggs
2 pats unsalted butter
2 servings pears
coffee or tea (distilled)

Lunch
1 boiled egg
1 pat unsalted butter
1/2 c. cooked carrots
lettuce wedge w/ lemon wedge
1 serving special cornstarch pudding
1 fresh orange
coffee or tea (distilled)
vegetable oil
sugar
honey

Supper
1 boiled egg
1 sliced tomato with lettuce salad
1/2 c. cooked carrots
lettuce wedge w/ lemon wedge
1 serving special cornstarch pudding
1 serving canned peaches
coffee or tea

Supper
1 serving special cornstarch pudding
with apple juice

Content:

Calories: 2166
Protein: 29.5 gm.
Fat: 133.0 gm.
K+: 961.9 mg.
Na: 729.7 mg.

Foods allowed in diet:

1. Eggs (4)
2. Fruits: Pears, apples, peaches, oranges, and plums
   We used orange juice and apple juice also
3. Vegetables: Tomatoes, carrots, pumpkins, and lettuce
4. Special Cornstarch Pudding
5. Coffee or Tea, sweetened (distilled)
### Kempner Rice Diet

<table>
<thead>
<tr>
<th>Meal</th>
<th>CHO (gm.)</th>
<th>PRO (gm.)</th>
<th>Fat (gm.)</th>
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</thead>
<tbody>
<tr>
<td><strong>Breakfast</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>200 gm. cooked Rice</td>
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<tr>
<td>8 oz. Fruit Juice</td>
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<tr>
<td>2 servings Fruit</td>
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<tr>
<td>Beverage with Sugar</td>
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<td><strong>Mid-morning Snack</strong></td>
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<tr>
<td>1-2 servings Fruit</td>
<td>20-40</td>
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<tr>
<td>Beverage with Sugar</td>
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<tr>
<td><strong>Lunch</strong></td>
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<tr>
<td>200 gm. cooked Rice</td>
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<tr>
<td>Beverage with Sugar</td>
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<tr>
<td><strong>Mid-afternoon Snack</strong></td>
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<tr>
<td>1-2 servings Fruit</td>
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<tr>
<td>Beverage with Sugar</td>
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<tr>
<td><strong>Dinner</strong></td>
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<tr>
<td>200 gm. cooked Rice</td>
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<tr>
<td>2 servings Fruit</td>
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<tr>
<td>8 oz. Fruit Juice</td>
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</table>

*No sugars, hard candies, jams, jelly without*