

SYMPTOMATOLOGICAL OBSERVATIONS OF LOW
OSMOLALITY BATH IN ESRD PATIENTS

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DEDICATION

This paper is dedicated to Gesine A. Franke, M.A., R.N. for without her encouragement and guidance during my undergraduate work at Texas Woman's University, I may never have completed my pursuit of a nursing career.

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CHAPTER 1

INTRODUCTION

The physiological causes, signs, and exact nature of intoxicating symptoms of chronic renal failure have not been specifically identified to date, neither through exhaustive research projects nor extensive solution-producing procedures. The research measures have, however, produced treatment procedures whereby the blood of renal patients can be purified in an artificial kidney. Hemodialysis procedures provide a life-saving crutch but do not cure the patient's uremia and often produce symptomatological complications. Life on hemodialysis is difficult.

As many as 30,000 to 45,000 persons in the United States are hemodialysis maintained each year (Gutch & Stoner, 1979), and each of these patients must face complicated problems created by their man-made machines, especially the possibility of human error or machine failure. In order to deliver the best machine-produced benefits for hemodialysis patients, every effort should be made to identify the first signs of machine-caused symptoms and the error should be traced immediately.

This study sets forth as its goal to identify specific symptoms exhibited by patients while in the process of hemodialysis and to correlate those symptoms to machine malfunction or maladjustments.

Problem Statement

The problem of this study was to answer the following questions: Is there a relationship between symptomatological observations and low osmolality bath in end-stage renal disease patients?

1. Is there a level of osmolality related to headaches in hemodialysis patient subjects?

2. Is there a relationship between body osmolality level and blood pressure in hemodialysis patient subjects?

3. Is there a relationship between individual electrolyte levels of sodium, potassium, or chloride, and the incidence of muscle cramps in hemodialysis patients?

Justification of Problem

The justification of the study was centered on mechanical and human error and ethical issues associated with hemodialysis. An artificial kidney is the main method of the treatment for chronic renal failure

patients. Nurses caring for patients receiving hemodialysis treatment share with physicians the responsibility of monitoring the effect of hemodialysis and concomitant physiological interactions.

Henson (1972) indicated that one of the most important difficulties of hemodialysis is a combination of mechanical and human error. Recently, Rozas and Rutt (1978) pointed out that progressive dialysis encephalopathy was related to aluminum contents in the tap water dialysate of the hemodialysis bath. Most dialysis centers now utilize deionized water to prevent dialysis encephalopathy.

Orranger and Mattern (1976) discovered that formaldehyde residue left on equipment after cleaning, induced hemolysis during chronic hemodialysis. Formaldehyde is used as a preservative for dialysis equipment, and the practice is widespread. Because of this discovery, nurses now use clinitest tablets to analyze the dialysis bath prior to use to detect the presence of formaldehyde. Another factor causing hemolysis during hemodialysis is overheated dialysate. Berkea, Kahen, Chazan, and Garell (1975) observed overheated dialysate (50 C) for 20 minutes. A normal dialysate temperature is the same as

the body temperature (36.5°). Evidence of delayed and protracted hemolysis was gathered.

Hemodialysis is an external life support system which has definite benefits and which can extend the life of persons who, a few years ago, would have died of uremia. Overall mortality for chronic hemodialysis patients is presently on the order of 9% per year (Thomas, 1978). The use of hemodialysis to extend life, however, does not speak clearly to the very important issue of what the quality of hemodialysis life may be.

Talley (1972) pointed out that some symptomatological complications of hemodialysis affect the quality of patient life and can even result in the ultimate complication--death. Mechanical and human errors can be prevented with adequate understanding of their pathogenesis. Therefore, there should be no incidence of mortality associated with any preventable complication (Byer, 1972). Patients can adapt and do well, be rehabilitated to some level of premorbid activity, and resume gainful employment (Talley, 1972).

Another point of this study's justification involves ethical issues. A study exposing patients to hypo-osmolality is not possible under real circumstances on human subjects. This is because rapid decreased plasma

osmolality induces the dialysis disequilibrium syndrome, which is a well-known complication of hemodialysis (Gutch & Stoner, 1979). The symptoms of the disequilibrium syndrome are characterized by headache, nausea, vomiting, restlessness, agitation, muscle cramps, hypotension, hypertension, convulsion, confusion, stupor, coma, and sometimes death. Since this investigator and other health team members are aware of these complications, it is contrary to the ethics of human experimentation as determined in 1964 in the Helsinki Declaration. The Declaration of Helsinki set forth criteria for clinical research involving human beings. Included in the criteria are the requirements of consent from human subjects and the recommendation of the minimal standards of ethical conduct for clinical investigation. Use of this declaration as a guideline for clinical research promotes conformity to the moral and scientific principles which justify medical research (Bishop, 1979).

Bishop (1979) also indicated that nurses must change the focus of their interest to the field of research aimed at the improvement of nursing technology. Many variables, such as those associated with hemodialysis, can be easily monitored. Medical devices used today

give nurses the opportunity to broaden the scope and knowledge of subjects of interest to the profession. Nurses have begun to be more involved in clinical research. This action requires that nurse researchers accept greater responsibility for patient care, both practically and ethically.

The patients in this researcher's investigation received dialysis three times per week for 4 hours duration each time. The hemodialysis unit used a batch-system, in which a given volume of concentrated dialysate was mixed with tap water and checked prior to use in the hemodialysis bath.

Travenol hemodialysis machines with plastic tanks were the dialysing apparatus. The plastic tanks holding the bath expanded without the knowledge of the staff. Therefore, the hemodialysis nurses prepared the tank solution with 10% more tap water than under normal circumstances. This occurred because water was added to a specified mark. The diameter of the tank was increased thereby increasing the volume above the therapeutic level. Thomas (1978) stated that the disadvantage of batch-system dialysis machines is that large reservoirs must accompany the machine. These

large reservoirs diminish portability and are extremely susceptible to human error.

As a result of the change in concentrate values of dialysate while the travenol machine was being used, patients complained of severe headache, muscle cramping, and hypotension during and after dialysis. It is the responsibility of the nurse working with these highly sophisticated hemodialysis machines to constantly observe, assess, and monitor the patient's condition. The nurse must also observe the mechanical accuracy of calibrations to ensure patient safety (Henson, 1972). These observations also include checking for signs and symptoms the patient may exhibit when there is low osmolality dialysate. The significant symptoms are headache (usually the first symptom to occur), muscle cramping, and hypotension. The nurse must differentiate these symptoms from among the multiple health problems exhibited in many end-stage renal disease (ESRD) patients because all symptoms manifested are not necessarily related to chronic renal failure. On the other hand, symptoms of low osmolality bath can be obscured by known health problems such as fluid and electrolyte imbalance and uremia (Gutch & Stoner, 1979).

The nurse's investigation of the relationship between low osmolality dialysate and hypotension, muscle cramps, and headache could contribute to quality patient care. The nurse's utilization of this knowledge and dedication to increase the quality of patient care during dialysis and other adaptations to illness is important. Observation of symptoms and signs exhibited by a patient can be utilized to enhance the quality of nursing practice. To accomplish this, the nurse must be prepared to recognize sets of symptoms and signs whenever similar incidences occur. If low osmolality during dialysis under certain conditions produces a set of symptoms and signs which is of diagnostic value, this should be identified in the opinion of this investigator. Such knowledge could be utilized by the nurse in decision making concerning the hemodialysis patient.

As a result of this study, a set of symptoms and signs under the condition of low osmolality level of hemodialysis could be recognized and determined. Only by such research methods can the nursing profession rely on assessment of symptoms and signs to validate the effectiveness of its actions, improve its function, and reach its ultimate goal of providing better quality of patient care.

Conceptual Framework

The "unphysiology of dialysis" concept (Kjellstrand & Evans, 1975) encompasses three parameters accompanying the complications of hemodialysis: cycling changes in blood chemistries, changes in osmolality, and body water distribution. The cycling of the blood chemistries produces changes in major hormones which regulate body water and electrolyte metabolism. Dialysis-induced alterations in these parameters are thought to contribute to the immediate problems of headache, nausea, vomiting, hypotension, and muscle cramping during dialysis. In the normal process of dialysis, osmolality falls due to loss of the majority of waste products, urea, and creatinine (Kjellstrand, Rosa, Shideman, Rodrigo, Davin, & Lynch, 1978). The factor causing the unphysiology of dialysis is the overload of water, potassium, sodium, and other metabolic products which causes hormonal changes in the body. Changes in hormones, mainly those effecting membrane transport, cause additional significant side effects (Kjellstrand et al., 1978).

Several studies (Bergstrom, 1978; Henrick, Woodard, Blachley, Govez-Sanchez, Pettinger, & Cronin, 1980; Kjellstrand et al., 1978; Rodrigo, Shideman, McHugh, Buselmeier, & Kjellstrand, 1977) tested in clinical

experiments the changes in plasma osmolality and ultrafiltration that occur during hemodialysis. Patients who received concurrent mannitol infusions showed fewer side effects. In addition, ultrafiltration without dialysis was associated with efficient removal of fluid in an isotonic fashion. Patients undergoing mannitol infusions experienced no change in plasma osmolality but a marked reduction in total body fluid. In spite of significant fluid removal, fewer side effects were experienced. It may be concluded that fluid removal is tolerated better when osmotic shifts are not present. Since both mannitol infusion during dialysis to keep patients' osmolality from falling and isosmotic ultrafiltration without dialysis resulted in similar reductions in symptoms, it is conceivable that rapid decreases in plasma osmolality, which would cause water to shift into cells, might be responsible for the symptoms of headache, nausea, muscle cramps, and hypotension. The inadvertent use of hypotonic dialysate would, thus, aggravate the tendency to manifest these symptoms. In this study, existing serum osmolality level data were related to symptomatological observations of patients on hemodialysis.

Assumptions

The assumptions relevant to this study were

1. Individuals receiving low osmolality dialysis bath (below 270 mosm/Kg) experience headache, hypotension, and muscle cramping.
2. Monitoring the patient's safety and mechanical accuracy of calibrations is part of the nurse's function.

Hypotheses

The hypotheses for the study were

1. There will be no difference in the incidence of headache reported by ESRD patients during hemodialysis among three groups classified by effective plasma osmolality (osmolality level: ≤ 265 , 266-279, ≥ 280).
2. There will be no correlation between blood pressure and effective plasma osmolality level of the hemodialysis patients.
3. There is no difference in the individual electrolyte concentrations of sodium, potassium, or chloride and the incidence of muscle cramps during hemodialysis treatment in patients with ESRD.

Definition of Terms

For the purposes of this study, the following definitions were applicable.

1. End-stage renal disease (ESRD) patient--a male or female receiving hemodialysis treatment in compensation for permanently nonfunctioning kidneys.

2. Headache--a painful sensation directly associated with the dialysis procedure as a new event of throbbing in the head. The headache was measured by notes on the patient's charts.

3. Muscle cramps--muscle cramps which are directly associated only with the dialysis procedure and include muscle spasm. The muscle cramps can be measured by notes of subject's complaints on the charts.

4. Blood pressure--diastolic and systolic blood pressure; the cardiac cycle consists of a period of relaxation pressure called diastolic pressure followed by a period of contraction pressure called systolic pressure. Hypotension is a diastolic blood pressure below 50 mmHg and systolic blood pressure below 90 mmHg on the sphygmomanometer reading and is noted on the patients' charts (Guyton, 1971).

5. Osmolality--normal effective plasma osmolality level is 285 mosm/Kg which controls water movement and distribution between and within the body fluid compartments. Low effective plasma osmolality level in the study is below 270 mosm/Kg (Rose, 1977).

6. Electrolytes--sodium, potassium, and chloride related individually; normal extracellular sodium level is 140 mEq/L, potassium level is 4 mEq/L, and chloride level is 104 mEq/L. These findings came from laboratory sheets.

Limitations

The limitations of this study were:

1. No control for recognizing incidents of charting errors and omissions.
2. Individual differences in tolerance of low osmolality.
3. Rate of expansion of faulty tank unknown.
4. No control of dialyzer and pressure of hemodialysis machine.
5. No control of infusing isotonic or hypertonic solution during hemodialysis.

Summary

This study was undertaken to determine a set of symptoms and signs under the condition of low osmolality level of hemodialysis patients. These symptoms and signs included headache, muscle cramps, and hypotension during hemodialysis.

CHAPTER 2

REVIEW OF LITERATURE

The literature reviewed was related to symptomatological observation and low osmolality level in end-stage renal patients. The research articles were organized into four areas: (a) end-stage renal disease; (b) osmolality--hypoosmolality and hyperosmolality; (c) osmolality-related symptoms--muscle cramps, hypotension, and headache; and (d) nurse and hemodialysis.

End-Stage Renal Disease (ESRD)

Kidney function depends upon the normal interrelated action of the cardiovascular, pulmonary, nervous, endocrine, metabolic, and urinary-collecting systems (Brundage, 1980; Leaf & Cotran, 1980). All of these vital systems are likely to suffer when the kidneys are progressively destroyed by a disease process. Any disease process that damages kidney tissue can result in chronic renal failure. Thus, chronic renal failure is largely independent of any one specific disease process. As chronic renal failure progresses, further damage is sustained and converted to end-stage renal disease (Leaf & Cotran, 1980). ESRD patients develop

an uremic condition due to impairment of homeostatic functions such as inability of concentrating urine and excreting waste products and increasing urea concentration in the body systems.

ESRD is treated effectively by extracorporeal hemodialysis. Symptoms that occur during treatment with hemodialysis are unpleasant facts of life when the patient is maintained by the artificial kidney (Henderson, 1980). During hemodialysis, the common clinical pattern consists of dizziness, malaise, nausea, headache, and cramps accompanied by a fall in blood pressure (Henderson, 1980). These symptoms are identified by Kjellstrand and Evans (1975) as being related to low serum osmolality level.

Approximately 25% of the patients have experienced these distressing symptoms during hemodialysis (Henderson, 1980). These symptoms often become bothersome stumbling blocks to the patient's successful adaptation to life with dialysis (Levandro & Davis, 1979).

Osmolality--Hypoosmolality
and Hyperosmolality

Osmolality

The concept of osmolality as described by Guyton (1971) is the total number of dissolved particles

(solutes) within body water or as the number of solutes per kilogram of water. Osmolality controls water movement and distribution between and within body fluid compartments. This movement is an important function of osmolality, as the exchange of materials between major fluid compartments is essential for normal cellular function (Rose, 1977).

In the theory of osmosis, water molecules exhibit random motion and can move across a membrane by a mechanism similar to that for diffusion of solutes. When solutes are added to water, the random movement of the water molecules is reduced. Water molecules then move more purposefully from an area of relative low osmolality to one of higher osmolality (Rose, 1977).

In order to determine osmolality disorders in a patient, the plasma osmolality and sodium level are the important diagnostic values. Rose (1977) explained precisely that the osmolality of the plasma (P_{osm}) is equal to the sum of the osmolalities of the individual solutes in the plasma. Most of the plasma osmoles are sodium salts, with lesser contributions from ions, glucose, and urea. The osmotic effect of the plasma ions can be estimated by multiplying plasma sodium concentration times two. The resultant plasma sodium

(Na^-) concentration is equal to the approximate osmotic pressure of serum as generated by potassium (K^+), calcium (Ca^{2+}), and magnesium (Mg^{2+}) salts (Rose, 1977).

The osmotic contributions of glucose and urea, both of which are measured in milligrams per deciliter are based on their molecular weight. The molecular weight of glucose is 180. The two nitrogen atoms in urea equal a molecular weight of 28. Urea is measured as blood urea nitrogen (BUN) and is used in the calculation of osmolality.

$$\text{mosmol/Kg of glucose} = \frac{\text{glucose} \times 10}{180} = \frac{\text{glucose}}{18}$$

$$\text{mosmol/Kg of urea nitrogen} = \frac{\text{BUN} \times 10}{28} = \frac{\text{BUN}}{2.8}$$

The plasma osmolality then, can be estimated from the following equation.

$$P_{\text{Osm}} = 2 \times \text{plasma } (\text{Na}^-) + \frac{(\text{glucose})}{18} + \frac{\text{BUN}}{2.8}$$

Although urea contributes to the absolute value of the P_{Osm} , it does not effect distribution of water between the cells and the extracellular fluid. Because of this, it is an ineffective osmole, and is instead related to

membrane permeability (Guyton, 1971). Effective P_{osm} does not include the BUN. The normal human values for plasma osmolality are 290 to 300 mosmol/Kg and effective P_{osm} is 285 mosmol/Kg (Rose, 1977).

Regulation of extracellular osmolality and total body water is the osmoreceptor-antidiuretic hormone (ADH) system. Guyton (1971) pointed out that an increase in osmolality of the extracellular fluids excites the osmoreceptors: this promotes ADH secretion and causes marked reabsorption of water by the renal tubules while solutes continue to be lost into the urine. As a consequence, the extracellular fluid becomes diluted and its concentrations of ions and other solutes move toward normal. On the other hand, low osmolality of the extracellular fluid decreases the activity of the osmoreceptors, thus decreasing ADH secretion, and large amounts of water are lost into the urine until the extracellular fluid osmolality again returns to normal.

Hypoosmolality--Hypoosmolar Imbalance

Luckmann and Sorensen (1980) defined hypoosmolality as an increase in water relative to solute concentration or a decrease in solute relative to water. Dilution of extracellular fluid (ECF) toxicity is a potential cause

of significant central nervous system dysfunction. Acute decreases in ECF osmolality can produce cellular over-hydration (swollen cells) ultimately leading to cerebral edema, seizures, and coma. Frequently, hypoosmolality is detected as a significant fall in the serum sodium concentration on routine laboratory screening. However, not all instances of hyponatremia are associated with hypoosmolality. Important discrepancies between sodium concentration and osmolality exist whenever there is a substantial rise in the plasma concentration of osmotically-active substances such as glucose or mannitol (Weitzman, 1980).

A number of researchers have asserted that the etiology of hypoosmolality and hyponatremia with minimal ECF expansion can be attributed to certain malignancies, drugs, central nervous system disorders, pulmonary disease, and myxedema (Schrier & Berl, 1976). As pointed out by Hendrick et al. (1980), Ogden and Cohen (1978), and Rodrigo et al. (1977), hypoosmolality and hyponatremia occur during hemodialysis with ultrafiltration. The reduction of osmolality leads to clinical signs and symptoms associated with dialysis disequilibrium. The symptoms and signs include nausea and vomiting, muscle cramps, hypotension, and headache.

The prevention of hyposmolality during hemodialysis has been attempted through various methods: (a) high sodium concentration in the dialysate (Ogden & Cohen, 1978; Port, Johnson, & Klass, 1973; Vanstone, Meyer, Murrin, & Cook, 1979); (b) constant decrease of dialysate osmolality (Chen, Ing, Dangirdes, Humayun, Brescia, Gandi, Hano, & Kheirbeck, 1979); (c) infusion of intravenous hypertonic sodium (Acchiando, 1975); (d) administration of slow-release oral sodium chloride (Catto, 1973); and (e) infusion of intravenous mannitol and 50% glucose (Hagstram, Lingerard, & Tibbling, 1969; Milutinovich, Graefe, Follette, & Scribner, 1979; Rodrigo et al., 1977).

Ogden et al. (1978) concluded that hypotonic or heavy solute solution greatly reduced dialysis disequilibrium symptoms. Ogden et al. indicated that the advantage of the high sodium dialysate method was that it modified cardiocirculatory responses such that greater ultrafiltration was achieved without concurrent hypotension. It was also a simple and cost-effective method. A disadvantage of the method was that it slightly aggravated the problems of interdialytic weight gain and predialysis hypotension.

Hyperosmolality--Hyperosmolar Imbalance

Luckmann and Sorensen (1980) defined hyperosmolality as decrease in water relative to solute concentration or increase in solutes relative to water. Both water deficit and solute excess cause hyperosmolality, shrinking of cells, and dehydration. Rose (1977) explained that the progression of hyperosmolality was exhibited as the primary symptom of dehydration. In dehydration, resulting from decreased water intake, excessive water output, or heavy solute load, the ECF becomes hypertonic, water then leaves the cells and passes into the extracellular fluid. The cells become dehydrated and shrunken as dehydration progresses. Finally, water becomes reduced in all body compartments (Rose, 1977).

Luckmann and Sorensen (1980) stated that the etiology factors related to hyperosmolality are cerebral injury (impaired thirst), coma, diabetes insipidus, diabetic acidosis, excessive infusion solution or heavy solute load glucose (D50W), protein, sodium bicarbonate, and mannitol. Acute increases in ECF osmolality can produce cellular dehydration ultimately leading to kidney failure, shock, fever, and coma (Weitzman, 1980).

In chronic hemodialysis patients, the clinical value of osmolality is higher due to elevated BUN; however, urea is an ineffective osmole. Therefore, the effective P_{Osm} actually is reduced because of a decrease in plasma sodium concentration. The patient may then demonstrate symptoms of hypoosmolality, rather than hyperosmolality (Rose, 1977). Presenting symptoms of this state include lethargy and confusion, hyponatremia (110 ± 8.1 meq/L) and serum hyperosmolality (340 ± 36.6 mosm/Kg H_2O). There are not many problems of hyperosmolality in chronic hemodialysis patients reported in the literature. Borges, Hochs, and Kjellstrand (1979) reported that eight patients had severe mannitol intoxication after infusing mannitol (304 gm) for prevention of renal failure. Six patients were hemodialyzed for 6 hours and one peritoneally dialyzed for 15 hours. One died before institution of therapy. All others recovered from their intoxication. During hemodialysis, there was a slow steady fall in serum osmolality as mannitol was removed by dialysis and then sodium was infused. Osmolality initially rose during peritoneal dialysis only to fall later. At 48 hours, there was no mannitol remaining in the seven treated patients.

Borges et al. (1979) concluded that intracellular idiogenic osmoles were best treated by hemodialysis. Hemodialysis rapidly corrects biochemical abnormalities and leads to clinical improvement.

Osmolality-Related Symptoms

Muscle Cramps

Muscle cramps are relatively common in patients on chronic hemodialysis. Muscle cramps are painful. They interfere with the patient's already compromised sense of well-being (Levandro & Davis, 1979). Unfortunately, the cause and treatment of muscle cramps has not merited much attention in the literature.

Several theories have been advanced to explain the origin of the more common variety of dialysis-related muscle cramps. Chillar and Desfonges (1972) developed a metabolic theory to explain muscle cramps. Muscle anoxia could be the major causative component. The theorists supported this by noting that after each dialysis (a) arterial blood pH rises considerably from acidotic to alkalotic levels; (b) serum inorganic phosphate levels fall; (c) 2,3-diphosphoglycerate red blood cell levels (RBC 2,3-DPG) also fall; and (d) aside from decreased levels of serum potassium and inorganic phosphate, electrolyte levels remain relatively stable before and

after dialysis. In uremia there is an increase in RBC, 2,3-DPG and metabolic acidosis. This decreases the hemoglobin affinity for oxygen and makes more oxygen available for tissue use. Dialysis reverses these changes. In effect, the hemoglobin regains its previous fondness for oxygen and anoxic muscle cramping results.

Jenkins and Dreher (1975) proposed a hypothesis relating to the electrochemical changes that might influence muscle cramps. They explained that the electrochemistry of calcium, sodium, and proteins affected muscle membrane excitability, calcium fluxes within the cell, and properties of the contractile proteins.

The majority of studies related muscle cramping to (a) decreased plasma volume due to ultrafiltration, (b) hypoosmolality, or (c) hyponatremia with ion flux across the muscle cell membrane (Jenkins & Dreher, 1975; Stewart, Fleming, & Mannel, 1972). Recently Neal, Resnikoff, and Unger (1981) supported the plasma volume depletion and hypoosmolality hypothesis. This study demonstrated that volume changes, ultrafiltration rate, and weight loss were greater in those patients who had cramps than in those who did not. This study also correlated the relief of muscle cramping with the

infusion of salt poor albumin, a substance which has a trivial effect on osmotic concentration while it substantially increases blood volume. The blood pressure changes observed in patients treated with D50W (50% glucose) were consistent with changes anticipated by the expansion of an actually depleted blood volume. An equal volume of D5W (5% glucose) did not significantly change blood pressure (Neal et al., 1981).

These investigators (Neal et al., 1981) concluded that if dialysis-related muscle cramps were a result of hyponatremia, D50W would not have relieved the cramping. Administration of D50W exaggerates hyponatremia as a result of fluid shifts. Like hypertonic sodium, hypertonic dextrose increases plasma osmolality and volumes and serves to decrease the intracellular volume.

The study by Milutinovich et al. (1979) was based on the same hypotheses that high ultrafiltration rates induce plasma and extracellular volume contraction. Rapid and excessive extracellular volume removal or hypoosmolality has been considered as the primary cause of cramps, and thus extracellular volume expanders were suggested for their relief. Additional amounts of sodium chloride or mannitol may partially cancel the

intended effect of ultrafiltration, which is the normalization of the patient's extracellular volume.

The effect of an injection of hypertonic glucose was evaluated for the same transitory effect on the extracellular volume without the administration of any extra sodium chloride or a nonmetabolized substance such as mannitol. In chronic uremic and nondiabetic patients, a total of 44 cramping episodes was observed. In a double-blind trial either 50 ml of hypertonic glucose or physiologic saline solution was injected, and the therapeutic response was evaluated. Of a total of 44 episodes of cramps, 26 were treated with hypertonic glucose and 18 with normal saline. Treatment with hypertonic glucose relieved 17 of the 26 episodes. In contrast only 5 of the 18 episodes were relieved with 50 ml of normal saline. No complications related to hypertonic glucose injections were identified (Milutinovich et al., 1979).

The last group of muscle-cramping studies correlate the cramping to hyponatremia (Stewart et al., 1972). The results of these studies were in opposition to the findings of Milutinovich et al. (1979). Stewart et al. (1972) increased the sodium concentration of the dialysate to 145 meq per liter and found statistically significant reduction

in the frequency of cramping. When the cramps did occur, usually during aggressive ultrafiltration, they responded to the usual treatment of boluses of normal saline (Stewart et al., 1972).

Based on these findings, Stewart et al. (1972) expanded their theory to say that muscle cramps during hemodialysis is the combined result of excessive reduction in plasma volume, coupled with cellular overhydration and sodium ion depletion. Plasma volume is reduced directly by ultrafiltration and, indirectly, by an intracellular solute lag, which allows water to move into the intracellular spaces causing overhydration.

Levandro and Davis (1979) pointed out there were two life-threatening causes of muscle cramping during dialysis. They are infrequent but require immediate attention should they occur. Cramps may result from (a) incorrectly prepared dialysate (overly concentrated or overly dilute) and (b) hemodialysis following an incompatible blood transfusion. These critical incidents can be prevented by verifying dialysate concentration and evaluating recent transfusions.

Hypotension

Hypotension is a common problem during hemodialysis. Approximately 25% of all hemodialysis patients have

symptomatic hypotension (Henderson, 1980). Symptomatic hypotension occurs so frequently that the maintaining of dialysis is seriously hampered. When the blood pressure drops significantly, the patient experiences nausea, vomiting, excessive perspiration, restlessness, cramping, diarrhea, confusion; and then goes into shock (Lancaster, 1979a).

Symptomatic hypotension during hemodialysis is multifactorial. Bergstrom (1978) and Rodrigo et al. (1977) recently called attention to the importance of osmolality changes occurring during dialysis, saying these osmolar changes are responsible for hypotension. They demonstrated that intradialytic hypotension was avoided with isolated ultrafiltration followed by hemodialysis with a parallel flow dialyzer. This finding was confirmed again by Bergstrom in 1978. Two years later, Henderson (1980) explained Bergstrom's (1978) study in the pathophysiological scheme.

Consider the common circumstance where 1 to 3 liters of plasma water are removed over a 4 hour period. During the course of that removal, plasma proteins are concentrated, providing an enhanced oncotic force to recruit extravascular fluid into the vascular space. This force, when quantitated in terms of milliosmoles per liter, would seem to be trivial at best, as it would provide less than 1/2 mosm (that is, 10 mmHg) driving gradient for capillary water reabsorption. This, however, is quite sufficient

to unbalance the Starling forces of the micro-circulation in favor of reabsorption of extravascular fluid. Consider further, that the small and more osmotically active solutes, urea and creatinine, move from the blood into the dialysis bath. Urea and creatinine at steady state are presumed to be in diffusion equilibrium with intracellular water. With the abrupt reduction of plasma water concentration in these solutes during dialysis and the lag in equilibration across biological membranes, an additional osmotic driving gradient for cell uptake of water would occur, leaving the extracellular space to be refilled from the already diminished intracellular space. For a given ultrafiltration rate, the magnitude of this volume depletion would clearly be a direct function of the rate of fall in plasma urea concentration with dialysis. (p. 571)

High efficiency dialyzers, especially those used in the clinical setting, promote a high plasma urea nitrogen level and put the patient at a greater risk for symptomatic hypotension than would less efficient dialyzers or peritoneal dialyzers where the urea clearance rate is less. In 1978, Rouby, Rotembourg, Durande, Basset, and Legrain indicated that dialyzers with large surface areas offer the potential for short treatment periods. The fluid removal required to render the patient dry would be accomplished in a shorter treatment time. Pathophysiologic speculation anticipates the accentuation of symptomatic hypotension. Recent tests have found the speculation to be valid (Henderson, 1980).

Osmolar changes are now associated with hypotension. Empirical therapies are being suggested for symptomatic hypotension. These include hypertonic mannitol, isotonic and hypertonic saline and plasminate (albumin). The mannitol, saline, and albumin provide extracellular solute particles with osmotic or oncotic capability to recruit vascular volume (Henderson, 1980).

Reports by Raja, Henriquez, Kramer, and Rosenbaum in 1979 suggested that acetate influx during hemodialysis is responsible for hypotension. They compared standard dialysate with bicarbonate dialysate to determine which influenced more hypotension. They found both factors (acetate and bicarbonate) may have an important role in the etiology of intradialytic hypotension. They also found that the incidence of hypotension decreased but was not prevented when osmolar changes were minimized with mannitol infusion or when bicarbonate dialysate was utilized with the Redy sorbent system. Further, hypotension episodes could not be avoided by prevention of osmolar changes or with bicarbonate dialysate alone.

Prevention of hypotension during isolated ultrafiltration may be due to both the lack of osmolar shifts and acetate influx. Raja et al. (1979) concluded that the incidence of hypotension is more efficiently decreased

with sequential ultrafiltration/hemodialysis than with hemodialysis alone, in spite of a relatively equal weight loss and osmolar shift with both methods.

Hendrick et al. (1980) also demonstrated the same findings as in the previous study of Raja et al. (1979) (the mechanisms involved in the stability of blood pressure during ultrafiltration alone vs. regular dialysis). In addition, they examined the importance of changes with serum potassium, osmolality, and plasma norepinephrine during several dialysis maneuvers based on the autonomic neuropathy hypothesis (Ewing & Winney, 1975; Lilley, Gordon, & Stone, 1976).

Hendrick and his associates (1980) selected six stable, normotensive chronic dialysis patients. During the 2 hours of each dialysis maneuver, the subjects exhibited a uniform 2% to 3% increase in body weight. They found that supine to upright mean blood pressure was decreased 90 to 75 mmHg in three patients. The patients became symptomatic after weight loss during regular hemodialysis, but orthostatic blood pressure was stable (80 to 86 mmHg), and the patients were asymptomatic after ultrafiltration and weight loss. Hendrick et al. (1980) concluded that the ultrafiltration procedure affords orthostatic blood pressure stability during rapid weight

removal when compared with regular dialysis. Maintenance of constant plasma osmolality appears to be the major protective reason for this stability, via an effect on peripheral vascular resistance.

The acute changes in blood pressure that occur in dialysis may be associated with acute changes in serum potassium concentration. Although plasma norepinephrine concentrations were high in dialysis patients, the magnitude of further increases in norepinephrine concentrations observed in these studies would appear to be insufficient to protect blood pressure during the stress of rapid weight removal and erect body position. Henderick et al. (1980) concluded from the preceding findings that acute vasculopathy or autonomic neuropathy is mediated by an osmotic fluid shift, which is the result of failure of both afferent and efferent resistances of the microvasculature to constrict appropriately when fluid is removed from the vascular space.

Hemodialysis-Related Headache

Headache is one of the most common symptoms experienced by people and has a recorded history dating back several thousand years. Karger and Base (1978) quoted Plato's advice: "for the part can never be well unless the whole is well, let no one persuade you to cure the

head until he has first given you his soul to be cured" (p. 1). Although headache is a common phenomena in many countries of the world, Newland, Illis, Robinson, Batchelor, and Waters (1978) noted that there has been little research until recently on headaches. There also are few dialysis-related headache studies noted in research literature and no description of them in the major nephrology or other medical textbooks. Perhaps because headache per se does not kill, it does not attract much research attention.

In 1972 new observations about a "dialysis headache" were made by Bana, Yap, and Graham (1972). They considered the headache problem as different from post-dialysis disequilibrium syndrome. Before Bana et al's. research, the dialysis-related headaches usually described by the patient were represented as an integral part of the so-called postdialysis disequilibrium syndrome. The researchers clinically compared headaches which the patient experienced before and after undergoing dialysis. The investigations found that the muscle contraction of tension headache was not associated with the dysequilibrium syndrome. Bana et al. postulated that the occurrence of headache correlated with many factors, for instance, biochemical and physiologic changes during dialysis, type

of dialyzer, or the patient's previous history of headache.

The results of the investigation by Bana and colleagues (1972) demonstrated that 70% of chronic dialysis patients developed headaches during hemodialysis. They also found that (a) all patients with signs of significant headache history before entering a dialysis program suffered headache during dialysis; (b) the headaches were more severe headaches during dialysis and lasted for longer intervals between dialysis; (c) the place of dialysis and type of dialyzer did not affect the headache pattern; (d) significant hypertension, emotional upsets, and excessive sodium intake increased the chances of severe headaches occurring; and (e) nephrectomies, transplants, and immunosuppressant drugs were found to be positively related to headache occurrence.

The studies done by Bana and Graham (1976) and Verniory, Povleige, Van Greertrugden, and Toussaint (1972) demonstrated that headaches during dialysis are related to decreased sodium and osmolality or negative pressure. Headache occurs at a time of falling blood pressure, and it is worse with the incidence of a large blood pressure drop. Headache occurs with the onset of

transplant rejection and the concomitant rise in plasma renin.

Bana and Graham (1976) based their study on the renin-angiotensin-aldosterone system as it relates to the dialysis headaches. Activation of the renin angiotension system results in vasoconstriction causing a rise in blood pressure. This, in turn, initiates the secretion by the kidney of vasodilating substances of the post-aglandin type which causes the vascular headache. Bana and Graham derived this line of reasoning from the work of Lee, Gougoutas, Takman, Daniel, Grostic, Pike, Nihman, and Muirhead (1966).

Bana and Graham (1976) concluded that (a) low renin level might be the result of excessive secretion of aldosterone or (b) it might be related to damage to the juxta-glomerular apparatus with consequent inability to secrete renin. Bana et al. (1972) found controversy resulting from their previous study report that low aldosterone level, low serum osmolality level, and low serum sodium level were associated significantly with increased occurrence of headache in chronic hemodialysis patients. Therefore, they suggested that alteration in the level of specific chemicals in the dialysate bath can decrease headaches.

Bana and Graham (1978) postulated another hormone, similar to a mineralocorticoid, may be responsible for hypertension and possibly headache. They studied plasma 18-hydroxy-11-deoxycorticosterone (18-OHDOC) levels. This hormone is synthesized from the zona fasciculata of the adrenal gland and to a small extent from zona glomerulosa. It is under pituitary adrenocorticotrophic hormone control. A trend toward low plasma 18-OHDOC and aldosterone levels in dialysis patients with headaches was noted. Previous researchers (Melby, Dale, & Wilson, 1971; Woods, Liddle, & Strant, 1969) indicated a number of low renin, low aldosterone essential hypertension patients have elevated blood 18-OHDOC. The assumption was that the plasma volume was too overloaded to suppress plasma renin and subsequent 18-OHDOC secretion. Adrenal damage, presumed secondary to the initial renal insult, results in hypersecretion of adrenal corticotrophic hormone and the elevation of 18-OHDOC.

Bana and Graham (1978) concluded from this study that the chronic hemodialysis procedure can lead to big shifts in blood volume and renal hemodynamic changes. This, in turn, can result in altered 18-OHDOC (prostaglandin, renal medullary hormone, vasodilator secretion) by one of the above mechanisms. The ultimate result

could be altered renin-angiotensin-aldosterone-prostaglandin balance. Additionally, their findings demonstrated no difference in BUN, creatinine, sodium, potassium, osmolality, or hematocrit, values between the groups of hemodialysis patients with headache and the group without headache.

The Nurse and Hemodialysis

The hemodialysis nurse can be defined as one who has knowledge and expertise in delivering care to the patient and family at any stage on the renal disease and dialysis continuum (Lancaster, 1979a). The nurse must be aware of the process of pathophysiological aspects of hemodialysis-related symptoms. Although appropriate medical intervention is warranted, the nurse's utilization of knowledge and dedication to increase the quality of patient care during dialysis is important. Observation of symptoms and signs that a patient exhibits can be utilized to enhance the quality of nursing practice (Henson, 1972).

Lancaster (1979b) pointed out that the nurse must be aware of (a) delivering quality care on a daily and long-term basis, (b) evaluating that care, and (c) accounting for the quality of care the nurse provides.

Quality nursing care begins by utilizing the nursing process to provide individualized care for each patient. An integral part of this process is the setting of realistic short- and long-term goals based on the dialysis nurse's greater depth of knowledge.

The nurse working with highly-sophisticated hemodialysis machines constantly observes, assesses, and monitors the patient's condition and also observes the mechanical accuracy of calibrations to ensure patient safety. Therefore, the nurse's observation of symptoms and signs is extremely important to hemodialysis patients (Henson, 1972).

With an adequate understanding of the pathogenesis, the nurse can better assess subtle changes in a patient's physical pattern which may effect the success of the hemodialysis treatment. When the hemodialysis nurse is confronted by symptoms, the nurse must be ready to tap all knowledge resources to provide the necessary intervention and emotional support. Thus, the nurse should have competencies based on knowledge and demonstrate the ability to implement the requisite quality of nursing care (Lancaster, 1979b).

Nursing care needed by the hemodialysis patients is the kind of care which must be measured periodically

by an objective assessment tool. A set of standards incorporated in an objective assessment tool is needed for ongoing evaluation of patient care. The set of standards helps the nurse to explore possible treatment for dialysis-related symptoms in a methodological way. This provides each patient the benefit of current available information (Lancaster, 1979b).

Summary

The literature on symptoms of hypoosmolality has been selectively reviewed. In general, most research supports a positive relationship between occurrence of symptoms and incidence of low osmolality. The osmolality-related symptoms have been studied simultaneously since 1972 by various researchers. Research has shown that hypotension and muscle cramps are associated with hypovolemia due to rapid ultrafiltration and subsequent osmolar shifts (hypoosmolality). Hypoosmolality and hyponatremia are related to headache as covariates of the aldosterone level. The cluster of phenomena observed by Kjellstrand and Evans (1975) is the cycling of blood chemistries, osmolality, and body water. Generally, this accounts for the associated symptoms of hemodialysis.

With an adequate understanding of these pathogeneses, the hemodialysis nurse can better assess subtle changes in a patient's physical pattern, which may affect the success of the hemodialysis treatment. The hemodialysis nurse's observation of symptoms and signs is extremely important for hemodialysis patients, as indicated by the old adage, "Nurse, know thy patient."

CHAPTER 3

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

The study utilized the ex-post facto correlational research design. The aim of this type of study is to determine which factors influence, effect, or relate to variables of interest (Polit & Hungler, 1979). A data sheet developed for this study was used to gather data from the patient's chart in order to relate the symptomatological observations to low osmolality bath in the hospitalized ESRD patients.

Setting

The setting for this study was a nonprofit county hospital in a metropolitan area in a southwestern city. The hospital is a 1,000-bed, acute care facility. The hospital has a six-bed hemodialysis unit. In addition, patients in various intensive care units are dialyzed with portable dialysis machines. The hemodialysis unit uses batch-system dialysis machines in which a large volume of dialysis solution is premixed and then circulated through a dialyzer. Regular staff of the unit consists of six registered nurses.

Population and Sample

The population included hospitalized males and females who were diagnosed as having ESRD. The convenience method of sampling (Treece & Treece, 1977) was used to select subjects until the minimum number of 30 was reached. This number was necessary to provide 10 subjects for the three osmolality levels. This included patients whose charts had shown that they met the study delimitations. All patients' charts were available in the hemodialysis unit file cabinet in this hospital. The delimitations were:

1. Charts which recorded hemodialysis therapy between January, 1979 and September, 1979.
2. Charts which belonged to males or females experiencing chronic hemodialysis and who were between the ages of 18 and 65 years and received hemodialysis in the hemodialysis unit.
3. Charts which belonged to the patients who were hospitalized and had documented chronic hemodialysis for over 3 months.
3. Charts which belonged to patients who were dialyzed with a Travenol I hemodialysis machine which had an expanded plastic dialysate solution tank.

5. Charts which belonged to the patients who were on 2 gm sodium diet and received no medications 6 hours prior to dialysis.

6. Charts which belonged to the patients who had blood pressure of 90/50 or above prior to dialysis.

7. Charts which documented no complaints of headache or muscle cramps prior to admission.

8. Charts which documented routine serum electrolytes and effective osmolality pre- and post-dialysis.

9. Charts excluded from the study were those which documented evidence of disorientation, uncontrolled hypertension, cerebral edema, fluid overload, metabolic imbalance, or mental psychosis.

Protection of Human Subjects

Prior to collection of data, permission to conduct this study was obtained from the Human Research Review Committee of Texas Woman's University (see Appendix A). Authorization from the hospital involved was also obtained according to procedures of Texas Woman's University and the institution's policies (see Appendix B). Patients' names were protected by coding the patient's chart with a number instead of noting the

patient's name on the data sheet. The investigator kept the original coded paper which contained the patient's name and chart number in order to verify the data during this study. Following completion of the study, the list was destroyed.

Instrument

The instrument used for this study was a data sheet developed specifically by this investigator. This type of instrument (Polit & Hungler, 1979) was selected because the data sheet format has been widely used to record data in physiological research as well as by other disciplines. The data sheet guided the systematic, orderly, and inclusive collection of desired data (see Appendix C). The categories for data collection were determined from the theoretical framework (Treece & Treece, 1977). These categories included body weight, sodium, potassium, chloride, effective osmolality, hypotension, headache, and cramps.

Data Collection

Collection of data was from September 1, 1981 to September 15, 1981 and was obtained from patients' charts. The investigator reviewed records at the hemodialysis unit supply room at a time when the hemodialysis

staff would not be disturbed. The data were assigned to groups according to previously identified osmolality levels.

Treatment of Data

All data were collected and coded onto the data sheets. Descriptive statistics were used to describe the population. For Hypothesis 1, a chi-square test for independent samples was performed. The patients were placed into one of the prescribed six cells, according to the presence or absence of headache and osmolality value.

For Hypothesis 2, the osmolality and diastolic blood pressure for each patient was correlated using the Pearson product-moment correlation coefficient. Hypothesis 3 was treated using the t-test for independent samples. Patients were categorized into two groups, depending on whether muscle cramps were present or not. Three t-tests were performed, one each using the potassium level, sodium level, and chloride level.

CHAPTER 4

ANALYSIS OF DATA

This study was conducted to determine the relationship between low osmolality bath and symptomatological observations in end-stage renal disease patients. The ESRD patients' charts were reviewed in an ex-post facto correlational research design. Data were collected from patients' charts. The findings related to analysis of the data are presented in this chapter.

Description of Sample

The total sample consisted of 33 ESRD patients' charts. These patients received hemodialysis in the hospital during 1979. Ages of the subjects ranged from 18 to 65 years, with a mean age of 43 years. Sixty percent of the sample were Black, 30% were White, and 10% were Spanish-American. Male patients comprised 42%, and female patients comprised 58% of the sample. Eighty percent of the sample received below 280 mosm/kg dialysate bath, and 20% of the sample received above 280 mosm/kg osmolality level of dialysate bath.

The data were collected from all eligible charts. The desired distribution of 10 units per osmolality level

could not be obtained from the charts. The groups related to osmolality levels was as follows: (a) osmolality below 265 group was 9 subjects, (b) osmolality 266-279 group was 17 subjects, and (c) osmolality above 280 group was 7 subjects (see Table 1).

Table 1

Number of Subjects in Each Osmolality Group

Osmolality Group	No. of Subjects
265	9
266-279	17
280	<u>7</u>
Total	33

Findings

The first hypothesis was: There will be no difference in the incidence of headache reported by patients during hemodialysis among three groups classified by posteffective plasma osmolality (effective osmolality: ≤ 265 , 266-279, ≥ 280). Table 2 demonstrates the distribution of the groups having headaches. The hypothesis was tested by utilizing the chi-square test to determine the independence of the samples for headache. The hypothesis could not be rejected ($\chi^2 = .82$, $p = .67$).

Table 2
Osmolality/Headache Findings

Osmolality Group	No Headache	Headache
265	7	2
266-279	11	6
280	4	3

N = 33.

The second hypothesis was: There will be no correlation between blood pressure and effective plasma osmolality level of the hemodialysis patients. The statistical technique used to test the hypothesis for Pearson's product-moment correlation coefficient for significance (r). Pearson product-moment correlation coefficients were used--one with systolic blood pressure and posteffective plasma osmolality and the second with the diastolic blood pressure and posteffective plasma osmolality. There was no significant relationship found between systolic blood pressure and posteffective osmolality (r = -.22, p = .10). There was no significant relationship found between diastolic blood pressure and posteffective osmolality (r = -.31, p = .06).

The third hypothesis was: There is no difference in the individual electrolyte concentrations of sodium, potassium, or chloride and the incidence of muscle cramps during hemodialysis treatment patients. Hypothesis 3 was treated by t-tests for independent samples. The hypothesis could not be rejected as no difference was found in the values of each of the three electrolytes' concentration and the incidence of muscle cramps. These findings are demonstrated in Table 3.

Table 3
Electrolytes/Cramps Findings

	<u>N</u>	Sodium	Chloride	Potassium
Cramps (Mean) (SD)	23	131 (4.98)	95.5 (5.08)	4.1 (1.06)
No cramps (Mean) (SD)	10	131.1 (2.80)	96.3 (2.50)	3.68 (0.76)
Significance		p = .43	p = .67	p = .29

N = 33.

Summary of Findings

The following summarizes the findings of the study.

1. No relationship was found between the incidence of headache and three levels of posteffective plasma osmolality (effective osmolality: ≤ 265 , 266-279, ≥ 280).

2. No relationship was evidenced between blood pressure and posteffective plasma osmolality.

3. No relationship was observed between muscle cramps and individual electrolyte concentrations (sodium, chloride, and potassium).

CHAPTER 5

SUMMARY OF THE STUDY

This chapter is a summary of the study. The conclusions were drawn from data obtained through statistical testing. Implications of the conclusions are stated, followed by recommendations for further study.

Summary

This investigation was an ex-post facto correlational study implemented in a 1,000-bed county hospital in a metropolitan area in a southwestern city. The study was conducted on data obtained under real circumstances of subjects accidentally being exposed to low osmolality bath during hemodialysis due to mechanical and human error. The study investigated posteffective plasma osmolality and symptomatological observations recorded on charts of 33 hospitalized male and female ESRD patients who received hemodialysis. All data were collected from the patients' charts. The data sheet was developed for this investigation. This data sheet guided the systematic, orderly, and inclusive collection of desired data. The categories for data collection were determined

from the theoretical framework. Data were analyzed by chi-square, Pearson product-moment correlation coefficient, and t-test in order to study the relationship of low effective plasma osmolality and the incidence of specific symptoms.

Discussion of Findings

The findings of the study were that no relationship was found between (a) the incidence of headaches and low osmolality, (b) muscle cramps and individual electrolyte concentrations (sodium, potassium, and chloride), and (c) blood pressure and low osmolality level.

Previous studies indicated a high correlation between low osmolality levels and the symptoms of headache, hypotension, and muscle cramps. It is postulated that the results of this study, which demonstrated no significant correlations between low osmolality level and symptoms, were possibly due to the following reasons: (a) the small sample size; (b) data utilized were obtained from patients' charts and may not have been recorded with rigorous precision, resulting in possible omissions and errors; (c) data collected were posthemodialysis effective osmolality blood samples instead of blood samples obtained during the occurrence of hemodialysis-related symptoms; (d) the infusion of isotonic or

hypertonic solutions given during hemodialysis was possibly associated with changes in postosmolality level; and (e) the absence of accommodation for differing dialysis machines' idiosyncracies.

Conclusion and Implication

Based upon the findings and the limitations of this study, the following conclusion was drawn: low osmolality bath does not generate diagnostically determinative symptoms. Implications for nursing care of the ESRD patients cannot be explicated from this study. Nurses cannot predict from laboratory values which patients will experience headache, muscle cramps, or hypotension. Also, the nurse cannot evaluate osmolality of bath from symptoms. Observable symptoms and osmolality of the dialysis bath are not predictive of each other. Therefore, it is important to rely on the nurse's assessment skills and deliver appropriate nursing care to the individual hemodialysis patient as the symptoms develop.

Recommendations for Further Study

Recommendations for further study are a repeat study to include data collection of the following: (a) type of dialyzer, (b) type of pressure of hemodialysis machine, and (c) the concurrent infusion of isotonic or

hypertonic solutions during hemodialysis. The apparatus and mechanics of dialysis equipment differ between the coil dialyzer and the hollow fiber dialyzer. Passage of the blood through either dialyzer is responsible in some way for the onset of symptoms. Therefore, a comparison needs to be made between the incidence of symptoms occurring in the patients on the coil or the hollow fiber dialyzer. Also the type of pressure of hemodialysis machine is responsible for the incidence of symptoms (Bana & Graham, 1976). Another suggestion for data collection is inclusion of the infusion of isotonic or hypertonic solutions given during hemodialysis. This is recommended because of its association with changes in posteffective plasma osmolality level.

APPENDIX A

-2-

1. Brief description of the study (use additional pages or attachments, if desired, and include the approximate number and ages of participants, and where they will be obtained).

The study will be ex-post facto correlational research design and derived its population from hospitalized men and women who are diagnosed LBD (between the ages of 18 and 65). The sample will be chosen by the convenience method, which will include 30 subjects. A data sheet developed for this study will be used to gather the data from patients' charts. The treatment of data will use a chi-square test in order to analyze the symptomatological observations of low osmolality bath.

2. What are the potential risks to the human subjects involved in this research or investigation? "Risk" includes the possibility of public embarrassment and improper release of data. Even seemingly nonsignificant risks should be stated and the protective procedures described in #3 below.

There are no potential risks in this study. Patients' names will be protected by coded patients' chart number instead of the noted patients' name on the data sheet. Therefore, the investigator will keep the original coded paper that has the patients' name and patients' chart number in order to verify the data during this study. Following completion of the study the list will be destroyed.

3. Outline the steps to be taken to protect the rights and welfare of the individuals involved.

Same as #2

4. Outline the method for obtaining informed consent from the subjects or from the person legally responsible for the subjects. Attach documents, i.e., a specimen informed consent form. These may be properly executed through completion of either (a) the written description form, or (b) the oral description form. Specimen copies are available from departmental chairmen. Other forms which provide the same information may be acceptable. A written description of what is orally told to the subject must accompany the oral in the application.

There are no human subjects involved in this study. The investigator will obtain agency permission in order to review the patient's chart.

- 5. If the proposed study includes the administration of personality tests, inventories, or questionnaires, indicate how the subjects are given the opportunity to express their willingness to participate. If the subjects are less than the age of legal consent, or mentally incapacitated, indicate how consent of parents, guardians, or other qualified representatives will be obtained.

NA

Signed *Lois Blough* Date 23 - June 81
 Program Director

Signed *Howard* Date 6-22-81
 Graduate Student

Signed _____ Date _____
 Dean, Department Head, Director

Date received by committee chairman: *E. Kurtz 6/23/81*

APPENDIX B

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Parkland Memorial Hospital

GRANTS TO Ho Soon Lee Cho

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem.

Symptomatological Observations of Low Osmolality Bath
in End-Stage Renal Patients

The conditions mutually agreed upon are as follows:

1. The agency (may) (~~may not~~) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (~~may not~~) be identified in the final report.
3. The agency (~~wants~~) (~~does not want~~) a conference with the student when the report is completed.
4. The agency is (willing) (~~unwilling~~) to allow the completed report to be circulated through interlibrary loan.
5. Other Student represents a copy of the report

Date: 7-6-81

[Signature] Dd. 7163
Signature of Agency Personnel

[Signature]
Signature of Student

[Signature]
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows:
Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

APPENDIX C

DATA SHEET

NUMBER	HEMODIALYSIS DATE			
SEX				
AGE				
DIAGNOSIS				
PREDIALYSIS	WT			BP
POSTDIALYSIS	WT			BP
PREDIALYSIS	NA	K	CL	EFF OSMOLALITY
POSTDIALYSIS	NA	K	CL	EFF OSMOLALITY
DURATION OF DIALYSIS				
SYMPTOMATIC	HYPOTENSION		YES	NO
	CRAMPS		YES	NO
	HEADACHE		YES	NO

REFERENCE LIST

- Acchiando, S. R. Management of muscle cramps in hemodialysis patients: Controlled prospective study. Proceedings of the Dialysis Transplant Forum, 1975, 6, 6.
- Bana, D. S., & Graham, J. R. Renin response during hemodialysis headache. Headache, 1976, 16, 168-172.
- Bana, D. S., & Graham, J. R. Plasma 18-Hydroxy-11-Deoxy corticosterone in dialysis patients with headache. Headache, 1978, 18, 23-25.
- Bana, D. S., Yap, A. U., & Graham, J. R. Headache during hemodialysis. Headache, 1972, 12, 1-14.
- Bergstrom, J. Ultrafiltration without dialysis for removal of fluid and solutes in uremia. Clinical Nephrology, 1978, 9, 156-164.
- Berkes, S. L., Kahen, S. I., Chazan, J. A., & Garell, A. S. Prolonged hemolysis from overheated dialysate. Annals of Internal Medicine, 1975, 83, 363-364.
- Bishop, V. Taking a stand on ethics: Nurses ethical responsibilities. Nurses' Mirror, 1979, 149, 22-23.
- Borges, H., Hochs, J., & Kjellstrand, C. Severe mannitol intoxication. The American Society of Nephrology, 1979, 1, 112A.
- Brundage, D. J. Nursing management of renal problems. St. Louis: The C. V. Mosby Co., 1980.
- Byer, B. J. Nursing care in dialysis. In G. L. Henson (Ed.), Caring for patients with chronic renal disease. New York: J. B. Lippincott Co., 1972.
- Catto, G. R. Treatment of muscle cramps during hemodialysis. British Medical Journal, 1973, 3, 389.

- Chen, W. T., Ing, T. S., Dangirdes, J. T., Humayun, O. J., Brescia, V. C., Gandi, J. E., Hano, V., & Kheirbeck, A. D. Hydrostatic ultrafiltration during hemodialysis with a constantly decreasing dialysate osmolality. The American Society of Nephrology, 1979, 1, 115A.
- Chillar, R. K., & Desforges, J. F. Muscular cramps during maintenance hemodialysis. The Lancet, 1972, 8, 285.
- Ewing, D. J., & Winney, R. Automatic function in patients with chronic renal failure on intermittent hemodialysis. Nephron, 1975, 15, 424-429.
- Gutch, C. F., & Stoner, M. H. Review of hemodialysis for nurses and dialysis personnel. St. Louis: The C. V. Mosby Co., 1979.
- Guyton, A. C. Basic human physiology. Philadelphia: W. B. Saunders Co., 1971.
- Hagstam, K. E., Lingerard, B., & Tibbling, G. Mannitol infusion in regular hemodialysis treatment for chronic renal insufficiency. Scandinavian Journal of Urology and Nephrology, 1969, 3, 257-263.
- Henderson, L. W. Symptomatic hypotension during hemodialysis. Kidney International, 1980, 17, 571-576.
- Henrick, W. L., Woard, T. D., Blachley, J. D., Gowe-Sanchez, C., Pettinger, W., & Cronin, R. E. Role of osmolality in blood pressure stability after dialysis and ultrafiltration. Kidney International, 1980, 18, 480-488.
- Henson, G. L. (Ed.). Caring for patients with chronic renal disease. New York: J. B. Lippincott Co., 1972.
- Jenkins, P. G., & Dreher, W. H. Dialysis induced muscle cramp. Treatment with hypertonic saline and theory as an etiology. Transactions of American Society of Artificial Internal Organ, 1975, 21, 479-481.
- Karger, S., & Base, I. Research and clinical studies in headache. Basel: Gasser & Cie, 1978.
- Kjellstrand, C. M., & Evans, R. L. Considerations of new dialysis schedules: Theoretical evaluation and review of literature. Opuscula Medico--Technica Lundensia, 1975, 16, 26-37.

- Kjellstrand, C. M., Rosa, A. A., Shideman, J. R., Rodrigo, F., Davin, T., & Lynch, R. E. Optimal dialysis frequency and duration, the unphysiology hypothesis. Kidney International, 1978, 13(8), 120-124.
- Lancaster, L. E. The patient with end stage renal disease. New York: John Wiley & Sons, 1979. (a)
- Lancaster, L. E. Where do we want to be tomorrow? Nephrology Nurse, 1979, 1, 6-10. (b)
- Leaf, A., & Cotran, R. S. Renal pathophysiology. New York: Oxford University Press, 1980.
- Lee, J. B., Gougoutas, J. Z., Takman, B. H., Daniel, E. G., Grostic, M. F., Pike, J. E., Nihman, J. W., & Muirhead, E. G. Vasodepressor and antihypertensive prostaglandine (PGE) of PGE type with emphasis on the identification of medullin PGE~217. Journal of Clinical Investigation, 1966, 45, 1036.
- Levandro, R., & Davis, V. Dialysis-related muscle cramps, still the patient's bugbear. Nephrology Nurse, 1979, 1, 25-35.
- Lilley, J. J., Gordon, J., & Stone, R. A. Adrenergic regulation of blood pressure in chronic renal failure. Journal of Clinical Investigation, 1976, 57, 1190-1200.
- Luckmann, J. & Sorensen, K. C. Medical-surgical nursing: A psychophysiologic approach. Philadelphia: W. B. Saunders Co., 1980.
- Melby, J. C., Dale, S. L., & Wilson, T. E. 18-Hydroxy-Deoxycorticosterone in human hypertension. Circulation Research, 1971, 28(Supp. II), 143-152.
- Milutinovich, J., Graefe, U., Follette, W. C., & Scribner, B. H. Effect of hypertonic glucose on the muscular cramps of hemodialysis. Annals of Internal Medicine, 1979, 90, 926-928.
- Neal, C. R., Resnikoff, E., & Unger, A. M. Treatment of dialysis-related muscle cramps with hypertonic dextrose. Archives of Internal Medicine, 1981, 141, 171-173.

- Newland, C. A., Illis, L. S., Robinson, P. K., Batchelor, B. G., & Waters, W. E. A survey of headache in an English city. In S. Karger (Ed.), Research and clinical studies in headache. Basel: Glasser & Cie, 1978.
- Ogden, D. A., & Cohen, I. M. A double blind crossover comparison of high and low sodium dialysis. Proceedings of the Dialysis Transplant Forum, 1978, 15, 158-165.
- Orranger, E. P., & Mattern, W. D. Formaldehyde-induced hemolysis during chronic hemodialysis. New England Journal of Medicine, 1976, 294(26), 1416-1420.
- Polit, D., & Hungler, B. Nursing research principles and methods. New York: J. B. Lippincott Co., 1979.
- Port, F. K., Johnson, W. J., & Klass, D. W. Prevention of dialysis disequilibrium syndrome by use of high sodium concentration in the dialysate. Kidney International, 1973, 3, 327-333.
- Raja, R., Henriquez, M., Kramer, M., & Rosenbaum, J. L. Intradialytic hypotension--role of osmolar changes and acetate influx. Transactions of American Society of Artificial Internal Organ, 1979, 15, 419-421.
- Rodrigo, F., Shideman, J. R., McHugh, R., Buselmeier, T. J., & Kjellstrand, C. M. Osmolality changes during hemodialysis natural history, clinical correlations and influence of dialysate glucose and intravenous mannitol. Annals of Internal Medicine, 1977, 86, 554-561.
- Rose, B. D. Clinical physiology of acid-base and electrolyte disorders. New York: McGraw-Hill Book Co., 1977.
- Rouby, J. J., Rotembourg, J., Durande, J. P., Basset, J. Y., & Legrain, M. Importance of the plasma refilling rate in the genesis of hypovolemic hypotension during regular dialysis and controlled sequential ultrafiltration-hemodialysis. Proceedings of the European Daily Transplant Association, 1978, 15, 239.

- Rozas, V. V., & Rutt, W. M. Progressive dialysis encephalopathy from dialysate aluminum. Archives of Internal Medicine, 1978, 138, 1375-1377.
- Schrier, R. W., & Berl, T. Disorders of water metabolism. In R. W. Schrier (Ed.), Renal and electrolyte disorders. Boston: Little Brown, 1976.
- Stewart, W. K., Fleming, L. W., & Mannel, M. A. Muscle cramps during maintenance of hemodialysis. The Lancet, 1972, 1, 1049.
- Talley, T. E. Complication of chronic hemodialysis. In G. Hansen (Ed.), Caring for patients with chronic renal disease. New York: J. B. Lippincott Co., 1972.
- Thomas, M. Maintenance hemodialysis. In E. A. Friedman (Ed.), Strategy in renal failure. New York: John Wiley & Sons, 1978.
- Treece, E. W., & Treece, J. W. Elements of research in nursing. St. Louis: The C. V. Mosby Co., 1977.
- Vanstone, J. C., Meyer, R., Murrin, C., & Cook, J. Hemodialysis with glycerol dialysate. Trans. American Society of Artificial Internal Organs, 1979, 15, 354-356.
- Verniory, A., Povliege, P., Van Greertrugden, J. J., & Toussaint, C. Renin and control of arterial blood pressure during terminal renal failure treated by hemodialysis and transplantation. Clinical Science, 1972, 42, 685-700.
- Weitzman, R. E. Disorders of water and electrolyte metabolism. In J. H. Stein (Ed.), Nephrology. New York: Grune & Stratton, 1980.
- Woods, J. W., Liddle, G. W., & Strant, E. G. Effect of an adrenal inhibition in hypertensive patients with suppressed plasma renin. Archives of Internal Medicine, 1969, 123, 366-370.