

DIET AND DIALYSIS IN ACUTE RENAL SHUTDOWN

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by

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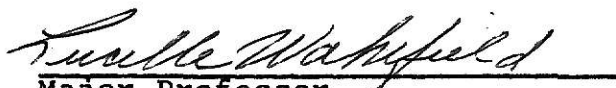
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INTRODUCTION

Kidney diseases are hard to diagnose (1). Statistically, there is more known about mortality rate than morbidity in kidney diseases. The incidence of kidney diseases as a major U.S. health problem can be expressed as a ratio of 9.49 persons per 10,000 population. Of this ratio, 55,000 deaths a year are caused by renal failure.

The detection of kidney diseases are masked by complications of other diseases (1). Cardiovascular diseases are the number one health problem in the United States. Kidney malfunctions are often associated with cardiovascular diseases, since both may be present in the same individual.

In 1972, in Vietnam, 63% of the American patients diagnosed as having renal failure died (2). Before dialysis, the overall mortality rate in patients with acute renal shutdown was 69%. In the military however, it was as high as 91%.

In general, emphasis has been placed on therapeutic rather than preventative measures (1). It is estimated that a 20% reduction in deaths and in acute conditions would accrue from an improved diet. The diet is critical to the maintenance of life in impaired kidney functions. The kidneys function to excrete the end products of body metabolism and to control the concentrations of most of the body's fluids (3). Therefore, it can be seen the kidney is closely related to nutrient metabolism; and the role of the diet and nutrition in preventing or modifying the severity of kidney diseases is advantageous (1).

If kidney diseases progress to the point of no return, kidney dialysis is employed as an alternative to diet or to transplantation. The scope of this paper recognizes the numerous diseases of the kidneys. Among these, only the most terminal cases requiring the use of kidney dialysis will be discussed. These can be grouped under the general title of acute renal shutdown. It includes: acute and chronic glomerulonephritis and tubular necrosis caused by nephrotoxins, crush syndrome and transfusion reaction (3-6).

A basic understanding of the normal function of the kidney is essential in understanding the diseased state and the preventative and therapeutic measures taken to return the kidney back to a normal state of functioning.

The purposes of this paper are to a) review normal kidney function; b) indicate kidney malfunction in acute renal shutdown; and c) discuss diet and dialysis as treatment for the disease condition.

NORMAL KIDNEY FUNCTION

Basically, the kidney refers to two organs (7). They are identical "paired, bean-shaped organs" situated behind the peritoneum on either side of the lumbar spine (1, 4, 7, 8). The kidneys lie against the back muscles in the upper abdomen under the diaphragm (7). They are protected by ribs and cartilage, but are not considered to be in the peritoneal cavity. The kidneys are referred to as being in the retroperitoneal space.

A layer of fat, called the adipose capsule, surrounds the kidneys and acts as the chief supporting structure (7). The kidneys are further enclosed in a membranous capsule made of fibrous connective tissue to serve as a framework.

The functions of the kidneys are threefold. A primary function is to excrete urea, a nitrogenous waste material from body metabolism, and salts from blood plasma (3, 7). The kidneys also function as an aid in the maintenance of water balance. This is done by balancing the oral intake of water with the excretion of water (7). Lastly, they regulate the acid-base balance in the body. These will be covered in more depth later in the paper. Of concern now is a closer look at the kidneys themselves.

The kidneys are divided into two major portions, the cortex and the medulla (9). Each kidney contains approximately one million nephrons (3). A nephron is composed of a complex network of small tubes. Each nephron is capable of individual work in filtering blood. The tubes are divided into two groups: the glomeruli, which collect fluid and the convoluted tubules, that basically reabsorb fluids (3, 7). The glomeruli, proximal and distal tubules are located in the cortex (9). However, the loop of Henle and a portion of the collecting tubules pass through the medulla. The nephron, shown as seen by Best and Taylor (10) is in figure 1.

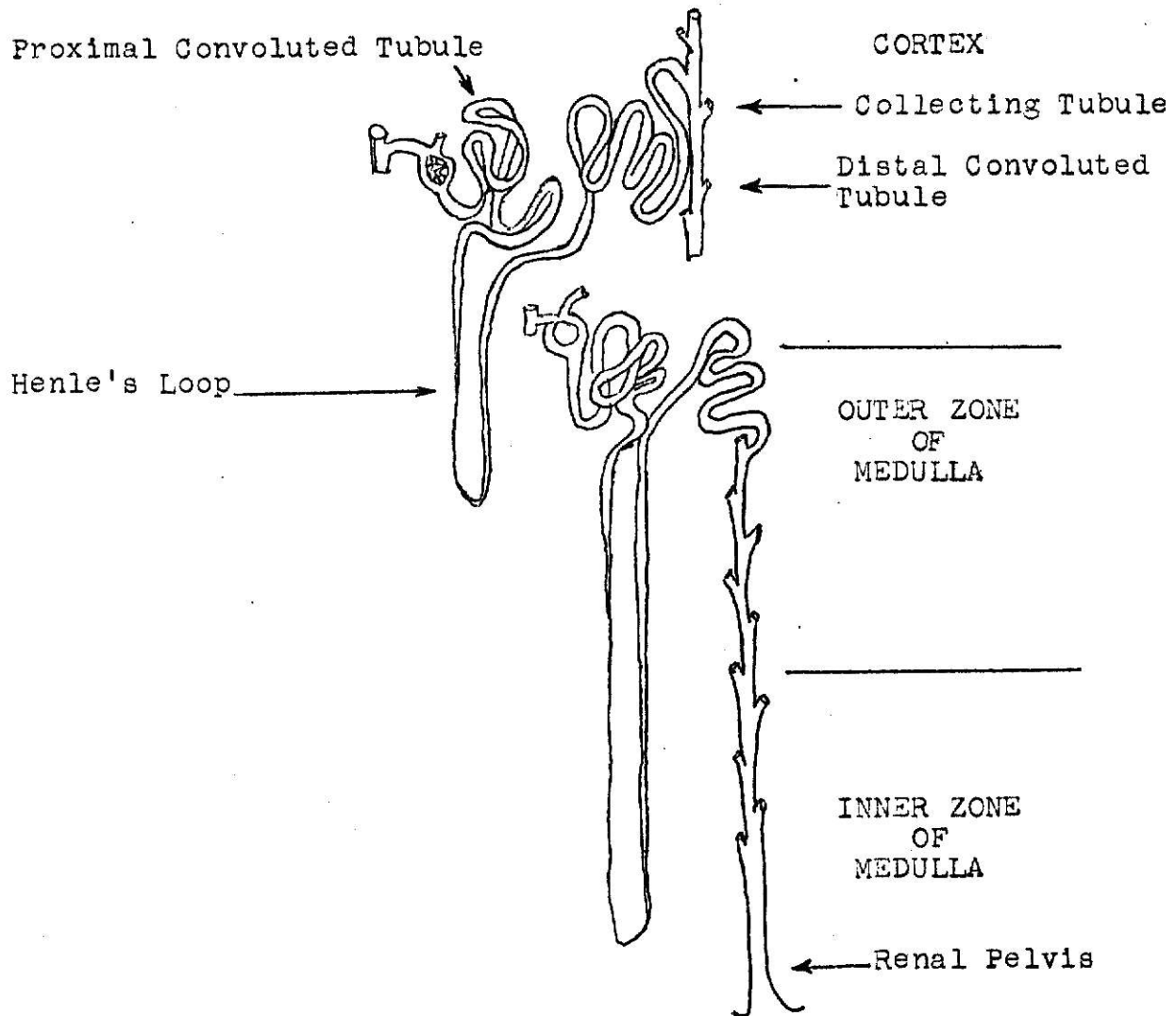


Fig. 1 The nephron within the kidney (10).

Pathway of Urinary Flow Through the Kidneys

The beginning of the urinary pathway, within the kidney, occurs in the cortex (3). The outer zone of the medulla, the inner zone of the medulla and the papilla are encountered later, as the urine is filtered through the nephron and empties into the pelvis (3, 10).

Blood enters the nephron by way of the afferent arteriole (3). From there it flows into the glomerulus. Two things occur in the glomerulus. The blood is first filtered through the glomerulus and passes out again by way of the efferent arteriole, through a capillary network called the peritubular capillary network. The network surrounds the kidney and goes back into the veins. A second part of the fluid is filtered and collected in Bowman's capsule.

Purpose of the Structures

The fluid that has been collected by Bowman's capsule flows through a series of tubes beginning with the proximal tubule. About 87% of the water, along with glucose, amino acids, protein and aceto-acetic acid are reabsorbed. However, there are also other variable secretions. These consist of hydrogen and potassium ions. All are regulated by electrolyte and osmotic concentrations in the extracellular fluids. Near the end of the distal tubule is the juxtaglomerular apparatus. The juxtaglomerular apparatus is the passing point of the collecting tubule meeting the afferent arteriole. It is a structure believed to

have either an endocrine function or other function relating to the control of blood through the afferent arteriole. The last point in the kidney, still within the cortex, is the collecting tubule. However, part of the collecting tubule passes through the medulla again before emptying into the renal pelvis. The paratubular capillaries in the medulla are arranged in long loops called the vasa recta. This serves as a mechanism in concentrating the urine (9). It will be discussed later in relation to osmolarity. The urine formed from the waste products of metabolism and excess water are passed into the tubules at the rate of approximately one ml per minute (3, 10).

The individual nephrons empty into the outer zone of the medulla of the kidney (7, 10). The urea goes to the renal pelvis, in the inner medulla, and passes through calices into the ureter and down to the bladder (3, 10). From the bladder, the urea is excreted from the body by the uretra (7).

The flow of blood through the kidneys have relevance in determinations affecting kidney capacities at given periods (3). Normally, in the reference man weighing 70 kg, the rate of blood flow would be 1,200 ml per minute, with a variance of 500 to 1,500 ml per minute. This is determined by using the renal fraction.

The renal fraction is the portion of cardiac output that passes through the kidneys per minute. This means, the basal cardiac output of the reference man equals 5,000 ml per minute blood flow. Of the 5,000 ml per minute blood flow from the

heart, 24% is filtered per minute by the kidneys, with a variance of 10 to 30%.

The afferent and efferent arterials regulate the blood flow through the kidneys. The walls of the arterials are lined with smooth muscles which can constrict to lower blood flow through the kidneys when stimulated by sympathetic nerve impulses (3).

The Glomerulus and Filtration

Taking an even closer look at the nephron, it can be seen the nephron is the functioning unit of the kidney (3, 5). It consists of the glomerulus and the tubules (5). First consideration is given to the glomerulus. An important part of the glomerulus is the membrane (3). It consists of three layers. The endothelial membrane is on the inner surface of the glomerular capillary. The epithelial cells are in the middle and the basement membrane is on the outer edge of the glomerular capillary. The membrane differs from other membranes in the body by being twenty-five times more porous. This allows for a more rapid filtration of water and particles (3).

Filtration of water and particles occurs by "hydrostatic pressure" inside the glomerular membrane. It is the pushing of water and particles through capillary pores into Bowman's capsule. The opposing force comes from "colloid osmotic pressure" in the blood. The pressure in the glomerular capillaries is estimated to be 70 mm of mercury; whereas, the pressure in Bowman's capsule is estimated to be 20 mm of mercury.

The rate of glomerular filtration depends on filtration tissue pressure. Afferent and efferent arterioles work together to stabilize the filtration rate. The afferent arteriole constricts, to decrease blood flow into the glomerulus. This also acts to decrease the resistance to blood outflow from the glomeruli. In this way the rate of blood flow can be altered. The arterioles are stimulated by the sympathetic nerves. The reverse occurs depending on the nerve impulses (3).

Overall, an increase in arterial pressure increases glomerular pressure; which in turn, increases glomerular filtration rate. However, when arterial pressure falls to 60 mm of mercury, as in severe hypotension, the filtration pressure becomes zero and the output of urine also becomes zero. The result is anuria (3, 5). A decrease in protein, such as in hypoproteinemia, would increase filtration pressure and filtration rate (5).

Increases or decreases in protein plasma are inversely proportional to changes in the colloid osmotic pressure (3). The higher the colloidal osmotic pressure, the less the filtration rate and vice versa. For example, if a person drank much water, the colloidal osmotic pressure would decrease and the glomerular filtration rate would increase. The filtration rate may also be influenced by renal blood flow (5). The volume of blood or plasma which contains the same amount of substance that is excreted in the urine in one minute is known as "clearance" of a substance (3).

Filtration can be measured by the renal plasma clearance of inulin, a polysaccharide. However, mannitol can be used (3, 5). Neither of the substances are secreted or reabsorbed, yet are filtered. Therefore, the filtered inulin, which has the same concentration as plasma, may be used as an indicator of the amount of plasma water filtered by glomeruli each minute (5). This is also known as the renal plasma clearance of inulin (3). The volume or rate of glomerular filtration can be estimated when the amount and the concentration are known (5). The normal glomerular filtration rate is 125 to 130 ml per minute (3, 5). The filtration fraction, or the renal plasma that becomes glomerular filtrate is 19%. The glomerular filtration rate is:

$$\frac{125 \text{ ml/minute glomerular filtration rate}}{650 \text{ ml/minute plasma flow}} = 19\%$$

Renal Tubules

In the tubules of the nephron, as in the glomeruli, an important part is the membrane (10). The inside of the tubule sides are composed of a brush border. Epithelial cells are in the middle and a basement membrane is on the outside of the tubule membranes. The tubule membranes perform two basic functions (5). They secrete organic electrolytes and passively and actively reabsorb filtrate (3, 10).

Passive reabsorption is transport of solutes from the tubules entirely by diffusion (3). The process of diffusion works through a pressure gradient. The pressure in Bowman's capsule is approximately 20 mm of mercury whereas, the pressure

in the renal pelvis is only about 2 mm of mercury. About 87% of the fluid is reabsorbed in the proximal tubules and 12% in the distal tubules. All the water and 1/3 to 1/2 of all the electrolytes are reabsorbed by diffusion. However, passive reabsorption occurs secondarily to active reabsorption.

Active reabsorption. Active reabsorption of substances involves a series of steps. A substance (S) from the tubules diffuses into the brush border of the epithelial cell. Here it combines with a carrier (C). The combination is promoted by a specific enzyme in the epithelial cell. The SC combination diffuses to the opposite end of the cell where another enzyme catalyzes the combination to separate. The substance (S) is pushed into the interstitial fluids and the carrier (C) rediffuses to the luminal pole to begin the process again.

Active reabsorption, in contrast to passive reabsorption, can occur against a concentration gradient. Therefore, energy is required for transport. The amount of energy depends on two factors. One is the quantity of substance being reabsorbed and the other is the concentration gradient encountered. The energy needed can be calculated by the force needed to move the substance times the distance the substance is moved, equals the energy necessary to move the substance. This may be expressed as: $F \times D = E$.

The metabolically important substances that are actively reabsorbed include: monosaccharides, especially glucose, all the amino acids, aceto acetic acid and protein. Protein is

transferred by a process called athrocytosis. Proteins are engulfed by the brush border, and transported into the epithelial cell. Within the cell, the protein is broken down into its individual amino acids. Then the amino acids are transported into the interstitial fluids. Sodium, potassium, calcium, magnesium, chloride, bicarbonate, phosphate and sulfate ions are reabsorbed and excreted in variable quantities in addition to the metabolically important substances (4).

On the other hand, urea is reabsorbed only slightly. Only 40% urea is reabsorbed through the tubules, in comparison to 99.4% reabsorption of water. Water is reabsorbed approximately ten or more times better than uric acid. Cretinine however, is not reabsorbed at all and is passed into the urine. The tubules of the nephron not only reabsorb and excrete substances, they also secrete hydrogen ions, potassium ions and certain drugs. The process is catalyzed by specific enzymes within the epithelial cell.

Active secretion. The only difference between active reabsorption and active secretion is the direction of the transport mechanism. The secretion comes from the interstitial fluid to the epithelial cell and goes to the inside of the tubule (3).

Hydrogen ions are secreted in large quantities into the distal tubules to help maintain acid-base balance. Acid-base balance will be discussed in more detail later in the paper.

Potassium ions, like hydrogen ions, are secreted into the distal tubules (3). The potassium ions are not regulated, but are in a direct proportion with the sodium and water concentrations. That is, if the potassium in the extracellular fluid concentration is too high, potassium is automatically secreted into the distal tubules of the kidney.

Concentrating power of the kidney. The concentrating power of the kidney is the ability of the kidney to selectively discharge some substances and keep others (3). The individual constituents in the concentrating power of the kidney can be seen by referring to table 1 devised by Guyton (3).

Plasma and blood flow through the kidneys can be estimated by para-aminohippurate (PAH), hippuran, and diodrast (3, 5). These substances traverse the carrier system and are completely removed from the tubules (5). Normal figures for renal plasma flow is 650 cc per minute with a blood flow of 1,200 cc per minute. These figures can be used to express renal function.

Tubular secretory function can be measured by an intravenous injection of PAH in the concentration of 50 mg per 100 cc. This would make the tubules work at their maximum capacity. However, this amount constitutes an overload and the tubules cannot secrete all of the PAH. Therefore, a measurement of the tubular capacity for maximum secretion is useful in measuring tubular damage (5).

TABLE 1

Relative concentrations of substances in the urine (3)

Substance	Urine (1 ml/min)	
	Quantity mEq/min	Concentration mEq/l
Na+	0.128	128.0
K+	0.06	60.0
Ca++	0.0048	4.8
Mg++	0.015	15.0
Cl-	0.134	134.0
HCO ₃ ⁻	0.014	14.0
H ₂ PO ₄ ⁻ HPO ₄ ⁻⁻	0.05	50.0
SO ₄ ⁻⁻	0.033	33.0
Urea	18.2	1820.0
Uric acid	0.42	42.0
Cretinine	1.96	196.0
Glucose	0.00 mg %	0.00 mg %

Maintenance of Water Balance

A second function of the kidney is to aid in the maintenance of water balance (3, 5, 7, 10). The kidneys help to regulate electrolyte concentrations, osmolarity (the ratio of water to dissolved substances) and the total volume of extracellular fluids.

Electrolyte concentrations. The regulation of electrolytic solutions involves the balance between the cations: sodium, potassium, calcium and magnesium; and the anions: bicarbonate, chloride, phosphate, sulfate, organic acids and proteinate. The total cations must equal the total anions expressed in mEq per liter to be in balance (5). The potassium concentration increases or decreases the electrical potential across the membranes of the nerve and muscle fibers (3). A low potassium concentration increases the electrical potential across the membranes. A sodium deficiency has a similar effect. Calcium ions regulate the permeability of the cellular membranes. When the concentration is high, the permeability is decreased and vice versa. The function of magnesium is not known, but high levels have been found to depress the nervous system. The anions bicarbonate, chloride, phosphate, sulfate, organic acids and proteinate do not need to be regulated as much as the cations (3, 5).

Sodium. Sodium accounts for more than 90% of the total cations with a normal concentration of 142 mEq per liter of plasma (8). It is the single most important ion that needs to

be regulated (3). Sodium controls the distribution of water throughout the body (8). The sodium ion concentration is controlled in the kidneys and in the adrenal cortex (3). A decrease within the kidney in sodium stimulates the pituitary gland to secrete antidiuretic hormone (ADH) (8). Antidiuretic hormone stimulates reabsorption of water from the distal tubule and collecting duct of the kidneys. As the blood becomes more dilute, less ADH is released and the urine becomes more diluted. An increase in sodium however, stimulates water retention.

The adrenal cortex secretes a mineralocorticoid, aldosterone to control the rate of sodium lost from the extracellular fluid (3). Aldosterone acts specifically on the renal tubules to increase the rate of sodium absorption. The mechanism of action is not known. A lack of aldosterone however, causes much sodium to appear in the urine. The reverse also occurs. Sodium concentration in the extracellular fluids is controlled by a feedback mechanism relating to the concentration of sodium in the extracellular fluids, and operates in a cyclic fashion. Additionally, renin, a hormone, is secreted into the blood, by the kidneys, when arterial pressure falls below normal (9). The renin combines with angiotensin, a plasma protein. The blood vessels constrict and increase arterial pressure. It also increases aldosterone secretion from the adrenal glands. Aldosterone acts on the kidneys to retain salt and water.

Recently, experiments have shown glomerulotropic hormone is secreted from the diencephalon region of the brain to the

adrenal glands to promote aldosterone secretion (3). When sodium is lost or gained, water is also lost or gained in direct proportion (8). The normal adult contains 1.09 g sodium per kg fat free body weight. In other words an average man contains 65 g or 2,700 to 2,800 mEq of sodium.

Potassium. Potassium ion concentrations in the extracellular fluids are not regulated as much as sodium (3). However, when aldosterone increases sodium reabsorption, potassium reabsorption from the kidneys decrease. It has been postulated, but not proven, that aldosterone is as important in regulating the sodium-to-potassium ratio as it is in regulating sodium ion concentrations. When potassium is lost, sodium enters the cell (8). The kidneys are unable to conserve potassium and may flush it out by diuresis. Most of the potassium is in the cells and has a concentration of about 150 mEq per kg of water. Worth noting is an inverse relationship of bicarbonate-to-potassium.

Calcium. Calcium provides a framework for bones and teeth and is involved in neuromuscular irritability. The calcium ion concentration is controlled by the parathyroid glands. A low level of extracellular calcium stimulates the parathyroid hormone to increase reabsorption of calcium from the bones to maintain an extracellular fluid balance (3). When extracellular calcium is high, calcium is lost in the urine. The adult contains approximately 20.1 g of calcium per kg of fat free body weight (8).

Magnesium. The function of magnesium is not completely understood (3, 8). It has a role in enzyme activity (8). It is important as a coenzyme in carbohydrate and protein metabolism as well as being involved in neuromuscular irritability. Magnesium is in the highest concentration in the cerebrospinal fluid. It is believed the same factors controlling calcium absorption also influence magnesium absorption. Decreased magnesium in the extracellular fluids increase tubule reabsorption and vice versa (3). The kidneys regulate magnesium extracellular levels the same as they regulate potassium concentrations. Both function primarily as intracellular ions. The adult contains about 0.36 g of magnesium per kg fat free body weight (8).

Anions. The anions are regulated secondarily to the cations (3). When cations are absorbed from the tubules an electropositive state is formed in the interstitial fluids. This creates electronegativity in the lumen of the tubules. Anions diffuse across the membrane and establish an electrostatic balance. This happens especially when aldosterone causes an increase in sodium reabsorption from the kidneys. In turn, there is an increase in the absorption of chloride, which composes about three fourths of the anions.

Calcium and chloride ions in the extracellular fluids indicate increased phosphate ions by virtue of the action of the parathyroid glands. The parathyroid glands cause the reabsorption of phosphate along with calcium from the bones. The other

anions primarily serve as acid-base regulators and will be discussed later in the paper.

Osmolarity. The extracellular fluid concentration is also controlled by its osmotic activity (8). Osmotic activity refers to the number of solute particles in a solution. The particles may be electrolytes or non-ionized particles, such as glucose and urea. The concentration of the particles is measured in milliosmols (1/1,000 of an osmol) since the concentrations are small. When the extracellular fluid is hypertonic, too much sodium is present. However, when the extracellular fluid has a below normal concentration of electrolytes, it is referred to as being hypotonic. Cells take up water in a hypotonic state and vice versa.

The osmolarity of extracellular fluids is controlled by the secretion of antidiuretic hormone (ADH) into the blood from the supraoptico-hypophyseal axis of the hypothalamus and neurohypophysis (3). When anti-diuretic hormone is released, increased amounts of water are reabsorbed from the distal tubule and the collection ducts of the kidney, and added to the extracellular fluid. This means as more hormone is released, the urine becomes more concentrated, indicating the occurrence of more reabsorption or conservation of water. In the extracellular fluid, as more ADH is released, the hypertonicity becomes an isotonic solution (8). Normal basal conditions show a small, moderately concentrated urine (10). However, when antidiuretic

hormone is absent, large volumes of water are released into the urine (3).

The loop of Henle and distal tubule reabsorb sodium chloride and other electrolytes. Therefore, a conservation of electrolytes occurs even though there is a large excretion of water in the urine.

Osmoreceptors, in ganglion cells, located in the anterior hypothalamus, function to control antidiuretic hormone. Each osmoreceptor, containing intracellular fluid, continually sends out nerve impulses. When the extracellular fluid volume is low, osmosis adds more water to the chamber of intracellular fluid. The increase causes the nerve impulses to become less rapid. This decreases the antidiuretic hormone secretion and causes the extracellular fluids to become more concentrated until normalcy is reached. The reverse also happens.

Total volume of extracellular fluids. Extracellular fluid is controlled by the electrolytic concentrations, by osmolarity, and by the total volume of extracellular fluid. The total volume of extracellular fluid depends on the net effect of the intake and the output of water per day.

About $1/2$ to $2/3$ of all the fluid intake is in the form of pure water. The rest is obtained from food (3). A small amount occurs from oxidation of hydrogen in the metabolism of food. The average intake of water per day is approximately 2,400 ml.

Water loss occurs primarily through the urine. During a normal temperature day (68°F), approximately 1,400 ml of urine

is lost. Seven hundred milliliters per day of insensible water is lost through the skin and lungs each day. The feces and perspiration account for the remaining water loss from the body.

The more water and sodium chloride taken in, the higher the arterial pressure becomes. This increases the volume of extracellular fluid. As a control, the glomerular pressure increases, resulting in more filtration and loss of water through the kidneys. The increased filtration helps maintain a balanced state between water and electrolytic intake and output. This mechanism is important but not a major source of regulation of urinary output.

The total volume of extracellular fluids is controlled additionally by pressoreceptors. They are in the walls of arteries. When extracellular fluid volume increases, blood volume increases. As increased blood volume causes increased arterial pressure, the pressoreceptors decrease sympathetic nerve impulses. This in turn, causes more urinary output to help reduce extracellular fluid volume. The arterial pressure, then returns to normal.

Additionally, volume receptors or stretch receptors located in the walls of the left atrium of the heart help control extracellular fluid volume. The receptors are thought to cause reflexes from the hypothalamus that cause a decrease in the secretion of aldosterone and antidiuretic hormone. It can be recalled antidiuretic hormone causes a decrease in the extracellular fluid volume (3).

Even though these primarily urinary mechanisms exist to help regulate extracellular fluid volume, dehydration may occur. The normally functioning kidney excretes about 400 ml of urine per day, called obligatory urinary output, under the condition of maximum conservation of water. If intake of water is less or equal to this amount, which varies in individuals, dehydration of tissues occurs.

Hydrogen ion concentration. A last consideration in the regulation of extracellular fluid balance is the acid-base balance or the hydrogen ion concentration.

The normal hydrogen ion concentration in the extracellular fluid is 4×10^{-8} Eq/liter, with a variance of an acidic 1.6×10^{-8} to an alkalytic 1.2×10^{-7} (3). The normal pH of arterial blood is 7.4. The normal intracellular fluid pH has never been measured, but is estimated to range from 7.0 to 7.2, based on CO_2 and bicarbonate concentrations. The body provides three mechanisms to control acid-base balance of the body fluids. They include buffer systems, respiratory ventilation and excretion of an acidic or basic urine.

Acid-base Buffer Systems

The buffer systems include hemoglobin in blood plasma, protein buffers, phosphate buffers and bicarbonate buffers (3). As food is consumed, it is broken down to provide many sources of acid and few sources of alkali (10). As the food is digested,

it interacts with buffer systems and thereby reduces the effects on pH of the extracellular fluids.

The bicarbonate system. The bicarbonate buffer system composes most of the extracellular fluid and is therefore, the most abundant regulator of acid-base balance. The system is composed of carbonic acid (H_2CO_3), a weak acid and sodium bicarbonate (NaHCO_3) a weak base (3, 10). Sodium bicarbonate is present in greatest amounts since most of the dietary intake is in an acidic form. First is a consideration of how the acid mechanism works (3).

A weak acid provides a slow dissociation into H ions and mostly dissociates into carbon dioxide and water. This provides only a weak concentration of acid. If a strong acid, such as hydrochloric acid were used, it would be converted to carbonic acid. The pH of the solution would then only be slightly lowered.

The major part of regulating the acid-base balance depends on the alkali bicarbonate concentration (10). It is called the alkali reserve and is an indicator of the degree of acidosis or alkalosis of the body fluids. If a strong base was used, such as sodium hydroxide, water and sodium bicarbonate would result with a net effect of a loss of carbonic acid. This would raise the hydrogen concentration slightly (3). Bicarbonate can be an effective buffer if it is in combination with potassium, calcium or magnesium (3).

Acids are ionized to a constant (K) known as the degree of dissociation. It is expressed as the hydrogen ion concentration times the bicarbonate ion concentration divided by the carbonic acid to yield a constant (K), which is in a reversible state. The same constant holds true for the dissociation of carbonic acid, except the K using carbonic acid is about 1,000 times greater than the K using carbon dioxide (3, 10). The pK plus the log of the concentration of bicarbonate ions divided by the concentration of carbon dioxide is the pH of a solution (3). The pK of the bicarbonate buffer system equals 6.1, and can be expressed by the Henderson-Hasselback equation:

$$\text{pH} = 6.1 + \log \frac{\text{HCO}_3}{\text{CO}_2}.$$

From this equation, it can be seen how an increase in bicarbonate ions causes alkalinity and an increase in carbon dioxide causes acidity in the extracellular fluids. The equation measures the degree of nonionization of an acid. The buffering system works directly proportionately to the concentration of bicarbonate and carbon dioxide in solution to limitations of 8 to 1 or 1 to 8 ratio, at which time the buffering power diminishes very rapidly.

The normal kidney has an extracellular fluid pH of about 7.4 and a pK of 6.1 (3). This means there is about twenty times more bicarbonate than carbon dioxide present in the body fluids. The buffer system has its greatest buffering effect when the pK is equal to the pH. Therefore, it can be seen the system does not work near its capacity. The system can be controlled

additionally, by the carbon dioxide from the respiratory system and the bicarbonate ions from the kidneys.

The phosphate buffer system. The phosphate buffer system is important in intracellular fluids and in the tubules of the kidneys. There is a high concentration of phosphate in the tubules of the kidney. A more acidic fluid is present in the tubules than in the extracellular fluid. Therefore, the phosphate buffering system can operate near maximum capacity. Within the cells, the phosphate concentration is greater than in the extracellular fluid and the pK and pH are more near the same. Thus, the effectiveness of the buffer system is increased within the cells. The phosphate buffer system is composed of sodium biphosphate and sodium phosphate. The pK is 6.8, whereas, the pH still remains 7.4. Therefore, the system works near its maximum capacity. However, it is only 1/6 the concentration of the bicarbonate system. Its buffering ability is not as powerful a system as the bicarbonate system (3).

The intracellular protein buffer system. Still another buffer system exists and composes 3/4 of the buffering power of the body fluids. It involves the affect of intracellular protein on the extracellular fluids. The diffusion of carbon dioxide and bicarbonate ions through cell membranes cause the pH of the intracellular fluids to change. The process works like the bicarbonate system. However, free basic amino acid radicals may dissociate into ammonium ions and hydroxyl ions. Hydroxyl ions

combine with the hydrogen ions to form water and have an effect on the pH of the solution.

Hemoglobin, in the blood, mixes with intracellular and extracellular fluids, therefore, can be considered a buffering system. The buffer systems all interact with each other. A change in one system affects all the others. This is known as the isohydric principle (3).

Acid-base respiratory ventilation. Another mechanism that maintains the acid-base balance is the respiratory system. Carbon from food is oxidized to form carbon dioxide. Carbon dioxide diffuses from the cells into the interstitial fluids and into the blood. The blood carries it to the alveoli of the lungs where it is exhaled into the atmosphere. Normally, about 1.2 millimols per liter of CO_2 is dissolved in the extracellular fluids while in transit to the lungs. Increased metabolism increases the CO_2 in the extracellular fluids and vice versa. As the rate of ventilation increases, so does the expiration of CO_2 . Under normal conditions the rate of CO_2 expiration is directly proportional to the CO_2 concentration in the plasma. The CO_2 in the plasma is dependent on the rate of metabolic formation of CO_2 . Therefore, the CO_2 concentration is controlled by a balance between the rate of metabolism and the rate of pulmonary excretion (3).

When alveolar ventilation decreases, CO_2 accumulates in the extracellular fluids and decreases the pH. If ventilation was lowered 1/4 of normal, the pH would decrease from 7.4 to 7.0.

If ventilation increased 2 times normal, the pH would increase from 7.4 to 7.63. The reverse also occurs. When the H ion concentration is high, the H ions act directly on the medulla oblongata, of the respiratory center of the brain. It increases the rate of ventilation to lower the CO_2 . If the pH were low, the ventilation would decrease to increase the CO_2 in fluid and bring the pH back to the normal 7.4. The efficiency of the system is about 50 to 75%. That is, if the pH went from 7.4 to 7.0, the respiratory system could increase ventilation and bring it back to 7.3 or 7.2 (3).

Acid-base balance within the kidney. In addition to the respiratory system, the kidneys help maintain a normal acid-base balance in the extracellular fluids. It controls the H ion concentration fundamentally, by increasing or decreasing the bicarbonate ion concentration in the body fluids.

The epithelial cells in the distal tubule of the kidney play a large role in the regulation of acid-base balance (3, 10). The epithelial cells secrete hydrogen ions into the tubular fluids (3). Carbon dioxide enters the cells from extracellular fluids. Within the cell, carbon dioxide combines with water, under the influence of the enzyme carbonic anhydrase, to give carbonic acid. The carbonic acid dissociates into carbonate and hydrogen ions. The hydrogen ions are secreted into the tubules until the pH is about 4.5. When the H ions are secreted into the tubules a sodium, potassium, calcium or magnesium ion is transported into the extracellular fluids. Therefore, when the CO_2

concentration, in the extracellular fluids, is increased the rate of H ion secretion into the tubules increases. This increases the coupled transport of cations from the distal tubules. The normal rate of H ion secretion is 3.5 millimols per minute. During the formation of hydrogen ions, in the epithelial cells of the distal tubule, bicarbonate ions are also formed. They diffuse out of the epithelial cells into the extracellular fluids, whereas, the hydrogen ions are secreted into the fluid of the distal tubules. Other bicarbonate ions enter the glomerulus and pass through the glomerular membrane into the filtrate in combination with the hydrogen to give carbonic acid, which dissociates into carbon dioxide and water. The carbon dioxide diffuses back to the extracellular fluids and the water passes on into the urine (3).

Therefore, if there is an excess of hydrogen ions, the bicarbonate ions are completely removed from the urine. The rate of bicarbonate ion entering the tubules changes in proportion to the bicarbonate ion concentration in the plasma, when the hydrogen secretion is constant and the carbon dioxide concentration in the body fluids is normal.

When the plasma bicarbonate concentration is 28 mM/l, the hydrogen ion and the filtrate bicarbonate ions are in balance thus, are neutral. This is the normal state. In acidosis, the ratio of carbon dioxide to bicarbonate ion concentration in the extracellular fluids is greater than normal. Whereas the ratio of carbon dioxide to bicarbonate ion concentration in the

extracellular fluids is less than normal alkalosis is depicted. In the case of acidosis, excess hydrogen ions are excreted into the urine. In alkalosis, excess bicarbonate ions are lost in the urine. These systems act as a regulator of hydrogen ion concentration. However, when excess hydrogen ions are excreted by the urine, they must combine with either a buffer or ammonia to prevent a rapid increase in pH (3).

The primary buffer is the phosphate buffer. The all-base reabsorption theory explains how excess H ions combine with disodium phosphate (Na_2HPO_4) to yield sodium dihydrogen phosphate (NaH_2PO_4), which is then excreted in the urine (11). The phosphate buffer system also spares sodium so it can be absorbed from the tubules and combined with bicarbonate ions formed in the secretion of H ions, to yield sodium bicarbonate (3). Sodium bicarbonate is excreted from the epithelial cells into the extracellular fluids to increase alkalinity to reduce acidic extracellular fluid and bring it back to a normal pH of 7.4.

Epithelial cells synthesize ammonia, which is secreted into the tubules. Ammonia reacts with secreted H ions to form ammonium ions. Ammonium ions then combine with primarily chloride, to form a neutral salt, ammonium chloride, and other anions to a lesser extent, which are excreted in the urine. Ammonium is important to prevent the H ions from forming hydrochloric acid (HCl). This would cause a drop in the pH to 4.5. If this occurred, the secretion of H ions would stop. Ammonia is secreted into the tubules, only if the tubular fluids are acidic.

The normal urine pH is 6.0, in contrast to the normal blood plasma pH of 7.4 (3).

From an understanding of the normal function of the kidney in relation to the formation of urine, the electrolytic balance and acid-base balance of the extracellular fluids, it is easier to understand the basic principles considered in kidney dialysis.

DISEASED STATE

There are a number of diseases of the kidneys. They include: renal insufficiencies: nephrotic syndrome, specific tubular abnormalities, acute pyelonephritis, pyelitis, renal tuberculosis, hydronephrosis, pyronephrosis, abscess of the kidney, perinephric abscess or perinephritis, neoplasms of the kidney, nephrocalcinosis, renal calcinosis, ureteral obstruction, nephrolithiasis, stag-horn calculi, and acute renal shutdown: acute and chronic glomerulonephritis and renal tubular necrosis resulting from crushing syndrome and transfusion reaction. Due to the limitations of this paper mentioned in the introduction, only the extremely terminal diseases classified under acute renal shutdown of the adult resulting with the use of kidney dialysis will be discussed. Acute and chronic glomerulonephritis, and tubular necrosis caused by nephrotoxins, crush syndrome and transfusion reaction resulting in edema will be discussed (3-6).

Kidney failure is characterized by an incapacity to maintain its excretory function (6, 12). Generally speaking,

oliguria, a urinary output of less than 400 ml/day and anuria, an absence of urinary output, may be manifested (6). The blood contains the end products of metabolism because the kidneys have an impaired function. When the blood contains over 30 mg/100 ml of urea, and/or uric acid and over 3.0 mg/100 ml of creatinine, uremia is usually present (6, 12). The creatinine clearance is less than 3.0 mg/100 ml in a shock patient with sustained trauma and sepsis (12). The electrolytes sodium and potassium are elevated, as well as the water content of the body (6). Commonly accompanying these symptoms are nausea, vomiting, bloody diarrhea and anorexia (6, 13).

Acute renal failure is characterized by a sudden decrease in the glomerular filtration rate, which leads to the cessation of renal function (4). The kidney may cease to function due to toxins, disease or trauma. Renal tubular necrosis is a dominant characteristic. The lumen of the tubules become filled with protein debris, epithelial cells and hemoglobin. If the initial phase is followed by a diuretic phase, recovery follows; otherwise, the kidney fails to function.

The first type of kidney malfunction to occur is glomerulonephritis (6). It is a form of nephritis characterized by inflammation of the glomerulus in the nephrons of the kidney. Generally, it is a result of a streptococcal infection in the blood. There are two forms of glomerulonephritis. One is the acute or initial phase and the other is the chronic or sometimes referred to nephrotic phase of the disease.

Acute Glomerulonephritis

Acute glomerulonephritis affects the glomerular capillaries (5). The earliest changes are structural. They include: swelling of the glomerular membrane, which reduces filtration, causing plasma protein, and red blood cells, to escape through severely damaged areas. Nitrogen and salt are also lost. It is readily detected by hematuria (5, 6). The interstitial fluid is also affected. Other symptoms may include oliguria, puffy eyes, edema, hypertension, anorexia, edema, nausea and vomiting (6).

If the onset is insidious, proteinurea and edema are prevalent (5). In this case, recovery is not too favorable. If the disease does not progress to the chronic phase, it lasts from 4 to 6 months and recovery is usually complete (6).

Dietary treatment. The dietary treatment, at this stage of development aims at conserving the functioning of the kidney. A very low protein diet is prescribed. The protein ranges from 0.5 gm/kg body weight or less. Sodium is restricted if edema and/or hypertension are present. However, the caloric content is adequate for the patients age, height, weight and amount of physical activity to prevent the breakdown of body tissues to be used for energy. Fluids are usually given generously, including water and ginger ale; plain or with corn syrup added. If edema and/or hypertension are present sodium is restricted. On the other hand, if oliguria is present, the fluids are limited to urinary output plus that lost by the skin.

If there is a progressive recovery, cereal with a small amount of milk, toast with butter, jelly or jam, baked potato and fresh or cooked fruit may be given to the patient. If edema and/or hypertension are still present, sodium needs to be restricted in the food, in cooking and no salt should be added at the table. Protein and salt may be restricted until there is complete recovery (6).

Chronic Glomerulonephritis

If acute glomerulonephritis persists and develops into chronic glomerulonephritis, acute renal failure may insue (5). In chronic glomerulonephritis, some or all of the capillaries become blocked, adhesions form and finally the entire glomerulus becomes scarred (14). This causes the glomerulus to stop functioning. When this occurs, the tubules are not able to receive any nutrition, resulting in atrophy (5). The body tries to correct the damage by building accessory blood vessels through the inflamed interstitial tissue. The proximal tubules respond by hypertrophy, allowing renal function to improve, but only temporarily.

Two patients with chronic glomerulonephritis revealed complete sclerosis of most of the glomeruli (15). Other glomeruli were hypercellular and contained endocapillary cells. Segments of the glomerular capillary walls were thickened by eosinophilic deposits. There was occasionally exudative or fibrin cap lesions in the glomerular capillaries, with prominent capsular

adhesions. There was pseudotubule formation within the parietal epithelium of Bowman's capsule. The arteries and arterioles from both patients showed initial thickening and hyaline deposits (15).

With the formation of fibrous tissue in the interstitial spaces, the tubules may become fragmented or amputated (5). End products of the cell and precipitated protein may block the tubules and prevent drainage, reflected by oliguria and azotemia (5, 16). Additionally, the collecting tubules may be blocked, which results in the impairment of functioning of unaffected nephrons (5).

Dietary treatment. Intrarenal nephronal blockage can lead to acute renal shutdown (17). If this happens, prompt alkalization of the urine by an intravenous diuretic solution will attempt to increase the urine flow. The alkalization increases the solubility of uric acid, preventing its precipitation. Mannitol solutions have been used with success. The use of ethacynic acid or furosemide to replace the urinary loss prevents osmotic diuresis. Osmotic diuresis may be in the form of hyperkalemia, hyponatremia or hyperosmolality. Intracellular dehydration and extracellular volume expansion are eliminated. Hyperkalemia can be controlled by 50 to 100 ml of 10% calcium gluconate solution or 200 ml of 3 to 5% sodium chloride solution given intravenously (12).

Acidosis usually accompanies renal failure. It should be corrected by an intravenous solution of 40 to 80 mEq of sodium

bicarbonate and a liter of 20% glucose with 50 units of insulin to prolong the effect. However, if there is a carbon dioxide content of 12 to 15 mM/liter or less, dialysis is required to alleviate the acidosis.

Tubular Necrosis

The second type of kidney malfunction to occur is tubular necrosis (5). Tubular necrosis is a death of tissue within the renal tubules (18). Excess fibrin degeneration products have been found (13). Scar tissue results that impairs functioning (18). It may be brought about by nephrotoxicity, the "crush syndrome" or blood transfusion reactions (4-6). The result is uremia and finally death if not treated (5, 6).

Nephrotoxicity from drugs. Nephrotoxicity may occur from drugs or from heavy metals (6). Various heavy metals may "poison" the tubules. This occurs especially with mercury. There is a normal or increased glomerular filtration rate (16). Mercury is deposited in the tubules and the kidney is unable to excrete them. The result is oliguria, a leakage of phenol sulfonphthalein from the distal tubules with the development of anuria; which ends in acute renal failure (6, 16).

Many antibiotics used to treat bacterial and fungal infections have been found to be nephrotoxic (19). Amphotericin B, used in treatment of systemic fungal disease, leads to acute renal failure (19, 20). Colistimethate and the aminoglycosides can directly cause acute renal failure accompanied with shock

and hemolysis (19). The aminoglycosides include: neomycin, kanamycin, gentamycin and streptomycin. Penicillins have caused anaphylaxis, a form of shock. It is characterized by hypotension, oliguria and azotemia, which lead to acute tubular necrosis.

Amphotericin B characteristically decreases blood flow to the kidneys. It has caused decreased renal function and an abnormal sediment in the urine of 80% of the people given the drug. It has a direct toxic effect on the tubules and is a renal vasoconstrictive agent. Its usage is suggested only when a life-threatening fungal disease is present. It is not dialysable and only 2 to 5% of the dosage is excreted in the urine. Burgess et al. (20) observed the glomerular filtration rate fell and renal blood flow decreased. There was an increase in potassium clearance in the distal tubules, renal tubular acidosis developed and the urine pH rose as the dosage increased.

Colistimethate produces toxic effects when the dose is more than 5.0 mg/kg body weight per day (19). When used in combination with other nephrotoxic drugs or when the dosage is not decreased, necrosis follows.

Neomycin causes toxic reactions when the dosage is over 40 mg/kg body weight per day. Albuminuria, hematuria, granular casts, azotemia, oliguria and uremia have been associated with the use of the drug. It may however, be hemodialysed from the blood if toxic reactions occur.

Kanamycin has replaced the use of neomycin, because of the higher incidences of toxicity caused by neomycin. It is however, a nephrotoxic agent. Decreased glomerulofiltration has been reported along with a decreased renal blood flow. Azotemia and oliguria have appeared with its use in some cases. If these toxic signs appear, kanamycin can be removed from the blood after 4 to 6 hours of hemodialysis.

Gentamycin may produce acute renal failure even when given in the recommended dosages. It does not produce oliguria as in the other drugs.

Streptomycin causes nephrotoxicity mainly in patients receiving the drug who have previously had renal damage. It is dialysable.

Acute renal failure resulting from nephrotoxins have characteristics of shock, trauma, sepsis, hemolysis and ischemia (13). These characteristics generally include oliguria with a urinary output of less than 400 ml/24 hours, severe azotemia and urinary sedimentation. There are a number of theories that explain the failure of tubules to function, which lead to oliguria. A. There may be an accumulation of debris in the tubular lumen. B. There may be compression of the tubules by interstitial edema. C. There may be a passive backflow of filtrate. Passive backflow is a theory that says a direct toxic effect or an ischemic event causes the glomerular filtrate to rapidly pass through the tubular lumen, where it is rapidly reabsorbed into circulation. The result is oliguria and

azotemia. D. There may be failure of glomerular filtration to reach the tubules (13, 16).

There is a reduction and finally absence of medullary and cortical arterial vessels creating perfusion (13). This suggests there is a reduction and finally cessation of glomerular capillary pressure by about one third of normal, resulting in sustained vasoconstriction in the glomerulus. The pressure would be about the same as plasma oncotic pressure causing a cessation of glomerular filtration. However, it is not known how a reduction in cortical perfusion is induced or sustained. The renin-angiotension axis is believed to be involved in causing vascular changes in acute renal failure. At the same time, other vasoactive mediators may be involved.

Crushing syndrome. Acute renal failure also results from a crushing injury, usually to an extremity (5). When this happens, shock, vasoconstrictive renal ischemia, oliguria, aciduria and nitrogen retention results. The kidney tubules swell and become cloudy. Damage is usually not severe. It is postulated there is hypertrophy of the juxtaglomerular apparatus (16). A vasopressor substance is released to decrease the glomerular filtration rate, which leads to acute renal failure. This mechanism is thought to be involved with the renin-angiotension system also.

After a crushing injury in rats, intravenous myoglobin was injected. Myoglobin casts accumulated and lesions appeared in the tubules. Extreme degeneration in the convoluted tubules was observed. If myoglobin was injected without injury, no response

by the kidneys was given. The cortex is usually found to be the ischemic area.

Further, renal vasoconstriction from shock may occur from trauma, hemorrhage or severe infection. These may all occur in the absence of pigment accumulations in the distal tubules (5).

Blood transfusion reaction. During a transfusion with incompatible blood, there is a sudden destruction of red blood cells involving the release of hemoglobin, causing shock, that can cause renal vasoconstriction (5).

Prevention of crush syndrome and related syndromes consist of an intravenous infusion of blood or plasma to relieve shock. A sulfonamide administration with fluid and alkali should be given to prevent an acidic urine. Sulfonamide reduces the danger of precipitation of red pigments.

A concentrated urine can lead to the precipitation of calcium salts, sulfonamides, uric acid or other substances. These can cause stones to form, which may block the tubules. When this happens acute renal failure is manifested.

Uremia. Uremia results from a decreased excretory function in acute renal failure (4). It is the presence of urinary constituents in the blood that result in a toxic condition (18). Nausea, vomiting, urinous odor of the breath and perspiration with anorexia, muscle twitching, generalized edema, pericardial pain, asterixis and convulsions are progressive symptoms (21). There is a decreased phosphate, sodium and calcium electrolytic

reabsorption in the renal tubules (22). Phosphate excretion has been shown to double during uremia, yet the phosphate balance is maintained with variations in sodium intake (23).

Dietary treatment. Low protein diets in renal management have been controversial since the early 19th century (24). The last quarter of a century a high calorie, low protein diet has routinely been used for nutritional therapy in renal failure. It limits exogenous protein and takes advantage of the nitrogen sparing effects of carbohydrates.

Animal studies. Animal studies indicate a utilization of urea in the presence of essential amino acids for the synthesis of nonessential amino acids in protein synthesis (25, 26, 27).

Karasawa (25) showed inosine, asparagine and ammonium acetate stimulated uric acid synthesis in chickens fed a 20% protein diet.

Purior (26) showed a negative growth in rats when given a diet containing only the essential amino acids. Positive growth was observed when nonessential amino acids and ammonium acetate were added to the essential amino acid diet. However, when the nonessential amino acids were replaced with ammonium acetate or urea, blood urea concentrations raised two to three times. The replacement of nonessential amino acids with ammonium acetate retarded growth by an altered amino acid supply to the tissues.

Valdemiro et al. (27) found rats fed a single meal of Maillard egg albumin decreased the supply of most amino acids

from the portal vein. Lysine, arginine and isoleucine were found to be the three least available amino acids after browning of the egg was done. Totally, most essential amino acids were unavailable for use from brown egg albumin. It can be seen the form of the egg albumins may alter their utilization within the body.

Human nitrogen requirements. Young et al. (28) did nitrogen balance studies on young adult men. He found the N requirement to range between 60 and 90 mg N/kilogram body weight when fed egg protein.

Prothro et al. (29) found the daily nitrogen retention necessary to support a weight gain of 4.6 kilograms in one year's time ranged from 26.9 to 29.7 g/day of dietary protein of high biological value 100.

Giordano diet. Giordano suggested the use of exogenous and endogenous urea to aid in protein synthesis by uremic patients (30). He used two diets, each was made of 2 g of nitrogen per day. One contained the eight essential amino acids. Otherwise, they were the same. The caloric content was 2,300 for women and 3,100 for men, with three fourths of the calories obtained from sugar and starch. The rest of the calories were in the form of margarine and vegetable oil. Vitamins, minerals and 2 g of sodium bicarbonate were given in capsules each day. The results indicated a utilization of ammonium and urea as a source of

nitrogen in the synthesis of nonessential amino acids for the body. Azotemia and hypertension decreased.

In a later study (31) five patients were studied. Four of the patients were azotemic with chronic renal failure and one was normal. Giordano showed nitrogen urea, in the presence of the eight essential amino acids could be used in the synthesis of nonessential amino acids for protein synthesis. Renal patients on a low protein diet utilized urea two to three times more than the normal patient. A diet rich in essential amino acids showed even greater utilization of urea in the synthesis of nonessential amino acids. A diet low in nitrogen, but rich in essential amino acids was recommended for treating patients with chronic uremia.

Giovannetti and Maggiore diet. Dietary treatment is essential in eliminating uremia. Giovannetti and Maggiore said the diet should lower the production of protein catabolism and prevent wastage of body proteins (21). The diet is based on the utilization of high urea levels in the blood and colon (32). Giovannetti and Maggiore used a diet composed of 1.0 to 1.59 mg nitrogen, 0.25 to 0.40 mEq of sodium, 35 to 50 mEq of potassium with 2,000 to 3,000 calories per day. The calories were in the form of unsalted butter, unsalted lard, vegetable oils, sugar, honey, maize starch and a special wheat starch. Low nitrogen fruits and vegetables were given for variety. They included: pears, apples, peaches, oranges, plums, tomatoes, pumpkin, lettuce and carrots to name a few. "Multivitamins" were used

for supplementation. Water and tea were allowed ad libitum. The essential amino acids were given in the L-form in a powder, enclosed in cachets. Egg proteins and egg albumin were also used because of their high biological value. Later it was modified to include chicken, beef steak and lamb as protein sources of high biological value (32). The results showed an improvement in uremic symptoms with a reduction in blood abnormalities (21). David and his associates (33) recommend the Giovannetti diet to alleviate uremic symptoms.

Giordano-Giovannetti modification diet. Berlyne (34) suggests an 18 g low protein diet consisting of minimal amounts of the essential amino acids except thionine, for patients with chronic renal failure. The diet contains 2,300 kilocalories; consisting of "Hycal" liquid glucose, low protein bread, spaghetti and low protein biscuits. Cream and ice cream are used whenever possible to increase the kilocalories by 1,000. Sodium varies from 10 mEq/day to a standard hospital diet of 40 mEq/day. Potassium varied from 30 mEq/day to 100 mEq/day. B vitamins and minerals were supplemented.

Most patients tolerated the diet, but a large amount of persuasion was required to stay with the diet. There was a reported reduction in nausea, vomiting, anorexia, and ambulation with an increased feeling of well-being and a return to work in 76 out of 90 patients with chronic renal failure.

Intravenous mannitol diet. Sometimes initially, mannitol is used in an intravenous solution in acute oliguric renal failure. However, it must closely be watched to prevent severe hyponatremia and hyperosmolality (35). The disadvantages of this treatment is a general lack of nutrition. It appears that even when used initially only a source of energy and water are present. Thus, the body is denied all other essential nutrients.

Sodium and fluid restriction diet. Another treatment consists of sodium and fluid restrictions (12). It consists of 25 ml per hour of 10% dextrose in water, or 600 ml/day plus urine output and any other fluid loss. An addition of 100 ml/day should be added for each degree of fever.

Amino acid, fructose and ethanol diet. In Britain, a commercial preparation of an amino acid solution, 15% fructose and 3.3% ethanol is used for patients with renal failure (36). However, the intravenous solution has been reported to increase blood- uric- acid levels causing a toxicity from acidosis. In the normal adult, an infusion rate of 0.5 g/kilogram body weight per hour gave no rise in uric acid. On the other hand, an infusion rate of 0.16 g/kilogram body weight per hour produced a rise in uric acid up to 10% in "seriously ill patient." It is thought fructose, amino acids and ethanol solution does not preserve the acid-base balance or provide for maximum usage of amino acids. It has been suggested L-amino acids and glucose solution be used in its place.

L-Amino acid, hypertonic dextrose diet. Ten renal failure patients with extreme metabolic and gastrointestinal absorption complications were studied (24). An intravenous injection of the eight essential L-amino acids, vitamins and electrolytes in a hypertonic glucose solution contained 750 to 1,000 ml total volume. The volume was determined by the degree of oliguria or anuria present. The need for dialysis was decreased in all ten patients by 50% or more. Hyperphosphatemia, hypocalcemia and acidosis were reversed spontaneously. Eight of the ten patients showed positive potassium and nitrogen balances. Blood urea nitrogen decreased and normal fluid balance was maintained. Body weight became stable. Nausea, vomiting, diarrhea, and lethargy rapidly disappeared in all patients. The indication was a utilization of the essential L-amino acids and endogenous urea nitrogen for lean tissue synthesis. The diet is recommended until oral foods can be tolerated by the patient.

In 1971 Abel et al. (37) also said food may not be utilized by way of the gastrointestinal tract in acute renal patients. He used an intravenous diet of eight essential L-amino acids with hypertonic dextrose. Recovery was noted.

L-Amino acid, glucose and vitamin supplements diet. Later, Abel and his coworkers used an intravenous solution of L-amino acids, glucose and vitamins as a dietary solution in acute renal failure (38). The results showed improved nutritional status for all patients on the diet. Wounds healed better, body weight was maintained, edema and hyponatremia disappeared.

Abel and his associates did further studies using an intravenous solution of water, glucose, the eight essential L-amino acids and vitamins as a diet for patients with acute renal failure (39). Again, the results were very favorable with recovery occurring faster than expected. The authors suggested there may be an effect of glucose and L-amino acids in combination on creatinine metabolism. The mode of action however, is unknown. They theorized the combination of glucose and L-amino acids could improve electrolytic imbalances and alleviate some of the early causes of death such as sepsis, gastrointestinal hemorrhage and pneumonia.

Blainey and his colleagues (40) pointed out an oral diet must include a patient's cultural background, socioeconomic status, personal idiosyncrasies, and habits if he is going to follow any diet. He must be convinced the diet given him is for his benefit. They suggest a low protein diet containing 0.5 g protein/kilogram body-weight/day with additional allowances to account for proteinuria.

Comparison of 20 g and 40 g protein diets. Kopple et al. (41) compared high caloric 20 g and high caloric 40 g diets of high biological value, in 15 patients with chronic renal failure. Azotemia decreased significantly only in the 20 g protein diet. However, weight decreased more frequently and bleeding time increased in the 20 g protein diet. Both diets indicated a deterioration in plasma pH, creatinine clearances, hematocrits and nerve conduction velocities. Patients adhered more to the

40 g protein diet. Only one in 17 patients adhered to the 20 g protein diet more than a few weeks at home. Dissatisfaction and overt hostility were common. When more liberal protein intake was allowed, patient acceptance, greater eating pleasure and symptomatic improvement was noted.

Blainey (40) pointed out dietary treatment alone is inferior to hemodialysis in chronic renal failure.

RENAL DIALYSIS

Dialysis is a diffusion of solute across a semipermeable membrane as a result of differing concentrations of fluid on both sides of the membranes (12). Dialysis is used when over hydration or uremia appears. The creatinine clearance is usually less than 5 ml/minute. Hypertension, congestive heart failure, hyperkalemia, infection, intracranial hemorrhage, convulsions and suicides are possible causes for death after dialysis. These causes however, only amount to a 10% death rate per year.

The advantages of dialysis include: an increased amount of safety, a more liberal administration of calories and nutrients, more comfort for the patient, the control of hypertension, congestive failure, nausea, vomiting and acidotic breathing. The major disadvantages are limited time, personnel and equipment.

There are two basic types of dialysis; one is hemodialysis and the other is peritoneal dialysis. Hemodialysis is technically more complicated (42).

Hemodialysis

Hemodialysis removes water from patients by a process known as ultrafiltration. There is a "pushing" or "pulling" of water through pores of a membrane enclosed in a container of the "artificial kidney" (12). The membranes are made of cellophane, derived from plant cell walls. It is unaffected by animal enzymes.

Hemodialysis description. The cellophane is about 25 microns thick with a pore diameter of about 5 millimicrons wide. This means substances with molecular weights up to 15,000 can pass through the pores. The surface area of the membrane is usually equal to or greater than the glomerular surface of $18,000 \text{ cm}^2$.

Hemodialysis essentially works through a principle of clearance, referred to as dialysance. Blood flows over one side of the membrane while dialysis fluid circulates on the other side. The concentration gradient and the length of time the blood is in contact with the membrane between the two solutions determines the amount of solute transfer from the blood to the dialysis fluid. Dialysance is equal to the blood flow in milliliters per minute times arterial plasma minus venous plasma, divided by the arterial plasma minus the concentration in dialysate in milligrams per milliliter. It has been written mathematically (12) as shown in figure 2. Effective transfer requires the solute to rapidly be carried away from the diffusing

$$D = \frac{F (Pa - Pv)}{(Pv - U)}$$

D = Dialysance, ml/minute

F = Blood flow, ml/minute

Pa = Arterial plasma concentration, mg/milliliter

Pv = Venous plasma concentration, mg/milliliter

U = Concentration in dialysate, mg/milliliter

Fig. 2 The mathematical formula for dialysance (12).

sites. The faster the blood flow, the greater the dialysance. However, when a point is reached, usually 200 to 300 milliliters per minute, dialysance is at its maximum rate and cannot be improved.

The dialysis fluid must be changed every three to four hours to maintain an efficient solute transfer from the blood. If not changed, the concentration gradient would not be at a needed optimum efficiency.

Increased blood flow rate increases the pressure within the blood tube that results in ultrafiltration. Ultrafiltration can also be produced by increased resistance to flow in the membrane. This can be done by constricting the outflow line from the dialysis machine. As much as 700 ml/hour of water can be removed by the use of ultrafiltration. Dialysis filters chloride, urea, potassium, sodium, creatinine, bicarbonate, uric acid, magnesium, sulfate and phosphate, in addition to removing water.

Chronic hemodialysis is feasible with use of an external prosthetic arteriovenous shunt. This allows repeated access to the patient's circulation. Superficial veins of the forearm are accessible by an arteriovenous fistula at the wrist (12, 43). The technique is known as "venipuncture" and is shown in figure 3 (43). It is demonstrated by the use of the Scribner arteriovenous shunt.

Hemodialysis fluid. Usually dialysis fluid contains sodium chloride, magnesium chloride, potassium chloride and dextrose (12). When sodium bicarbonate is used the pH will have to be adjusted to 7.4 by the addition of lactic acid. Dextrose is used to maintain the osmolality between 280 and 310 mOsm/liter. Higher concentrations rarely exceed 3.0 mEq/liter.

The preparation of the solution doesn't have to be sterile since the membrane is a barrier to infectious agents. The water should be distilled or deionized however, for the proper composition of the fluid. Solutions may be commercially prepared and used.

The dialysis fluid can be monitored constantly to be sure the solutions are prepared and delivered correctly. The temperature is continuously measured and kept at 40°C. The flow and pressure are also measured constantly. Hemoglobin can be detected in the dialysate to indicate leaks in the cellophane and bleeding into the solution (12).

The blood must constantly be anticoagulated or it will clot. Heparin is the most commonly used anticoagulant. It is

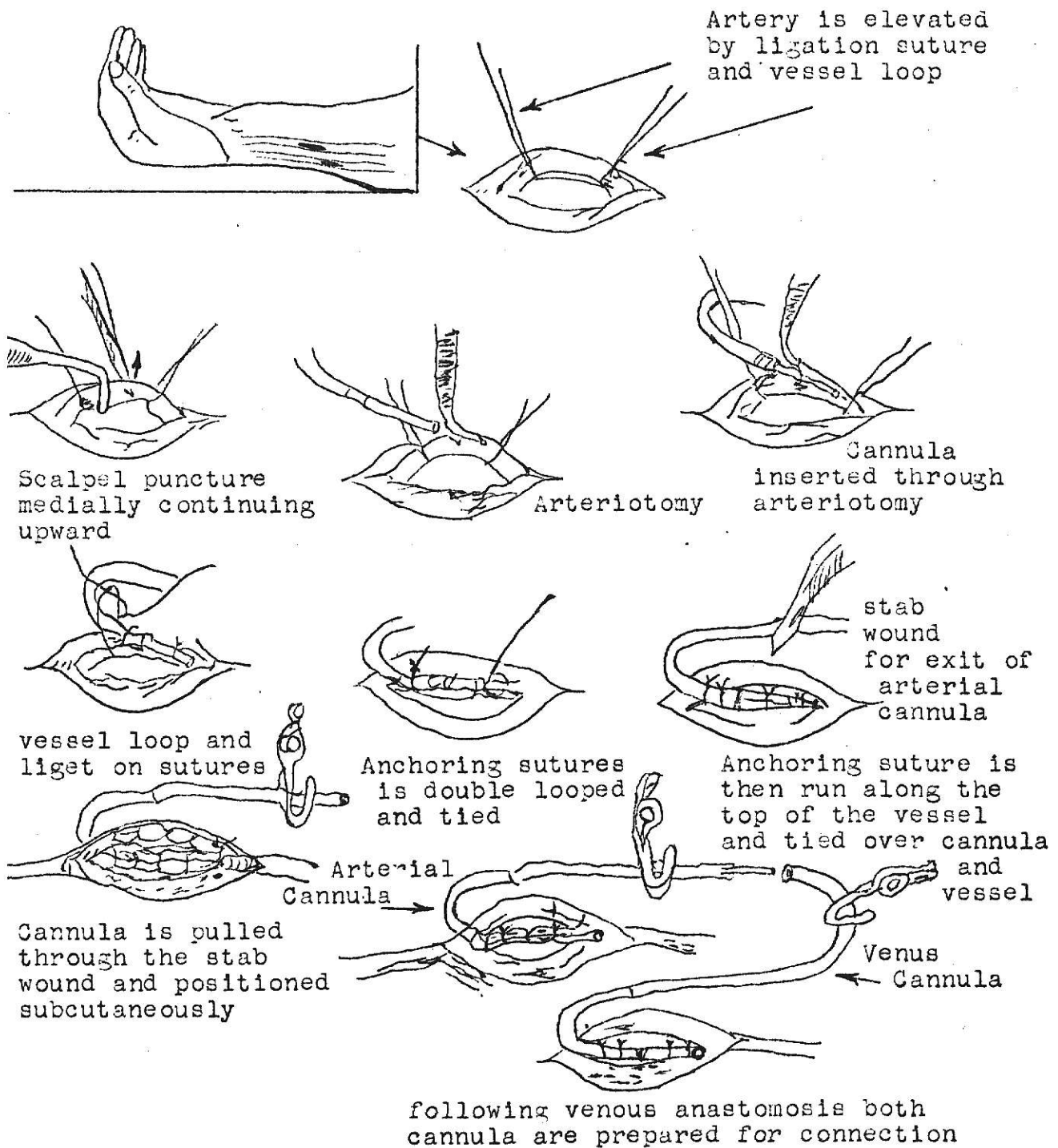


Fig. 3 Insertion of the Scribner shunt (43).

added in doses of 2,000 units per 500 milliliters of blood. It may be administered intermittently or continuously. If the intermittent method is used, 1,000 units per kilogram body weight of heparin is injected every 30 to 60 minutes.

Hemodialysis complications. Cardiovascular problems are many. High blood flow rates necessary for adequate hemodialysis may cause hypotension and shock (42). This is especially true when patients are bleeding or predisposed to bleeding. Hypotension, caused by a sudden drop in blood pressure, is caused by hypovolemia. Hypovolemia occurs during the transfer of blood to the dialysis machine (12). The Kiil hemodialysis machine has parallel membranes and requires less blood loss, because of a priming volume of 300-400 ml of dialysis fluid.

Transfusions are required when dialyzers with large priming volumes of 400-1,100 ml, as in coil dialyzers are used. Along with transfusions is a frequent occurrence of hepatitis. Sometimes, fluid is pumped into patients, while none is removed, resulting in circulatory overload.

There may be unidentified allergens or pyrogens in the blood that frequently cause transfusion reactions. Anemia may result from a decreased bone marrow production or decreased red cell survival during dialysis (12, 44). Packed red blood cells put into the dialysate may alleviate anemia (12).

Bleeding as a result of heparin may occur (12). Heparin administration during dialysis may cause lesions that lead to constructive uremic pericarditis (45). Hemorrhage causes

vasoconstriction in uremic patients, that may cause lesions if the surface of the heart is inflamed. Although this complication is not common, it does occur in acute or chronic renal failure or during short or long term dialysis. The ultimate consequence is death from congestive heart failure.

Dialysis may cause neurological complications. Dialysis disequilibrium syndrome may occur (12). It is characterized by headache, vomiting, hypertension, convulsions and coma. Cerebral edema causes the symptoms. When a patient is first dialyzed and is over hydrated, the osmotic gradient produces cellular edema; which causes encephalopathy.

In uremia, peripheral neuropathy characterized by pain and paresthesias occurs, mostly in the lower extremities. Motor defects, also occur. There is sleeplessness, fatigue and malaise.

Central nervous system disorders may cause death in chronic hemodialysis. Frequently, there is intracranial hemorrhage, thought to be caused by frequent anticoagulation treatment.

Changes in composition and temperature may result in hemolysis, hypernatremia and hyperkalemia. If sodium removal is excessive, hyponatremia occurs. Excessive amounts of glucose cause hyperglycemia and hyperosmolarity:

During uremia, there is a resistance to vitamin D by the body. This causes calcium and phosphorous abnormalities in metabolism. Large amounts of calcium bind to phosphates and are excreted in stools which causes hypocalcemia. In turn,

parathyroid hormone mobilizes the calcium store in the bone. The end results are manifest in hypocalcemia, hyperphosphatemia, vitamin D resistance and hyperparathyroidism (12). When dialysis is used daily there is a high amino acid loss through the dialyser membrane, deterring recovery (46).

Peritoneal Dialysis

Peritoneal dialysis is utilized in uremic patients. Its use was not very great until about 1940, even though the first study was done in 1877 on rabbits (12). In 1951, a single catheter was first used to insert and drain the dialysis solutions.

In chronic uremia, peritoneal dialysis has been used for as long as a year to sustain patients. It is more flexible for active people (47). Patients are allowed to eat as they wish as soon after peritoneal dialysis treatment has started than can the hemodialysis patients (47).

Peritoneal dialysis description. The peritoneal membrane is used in peritoneal dialysis for filtration of the blood. It is located between the parietal and visceral peritoneum. The membrane is $22,000 \text{ cm}^2$, whereas, the glomerular surface is $18,000 \text{ cm}^2$.

Dialysis fluid, dialysate, is run into the peritoneal cavity and interchanges or equilibrates with the body fluids. Then it is removed. This process is referred to as a single dialysis cycle or exchange.

The effectiveness of dialysis depends on the clearance. Clearance can be written mathematically (12) as shown in figure 4. The shorter the time allowed for dialysis fluid to remain in

$$C = \frac{UV}{P}$$

C = Peritoneal clearance ml/min of substance

U = Concentration of material in the dialysate that has come in contact with the peritoneium in a given amount of time

V = Volume of dialysate

P = Plasma concentration of the material

Fig. 4 The mathematical formula for clearance (12).

the abdomen, the higher the rate of dialysis and clearance. The most economical conditions are dialysis employed over 2.5 l/hour.

A 1.5% glucose isotonic solution doesn't exchange significantly with body fluids if left in for several hours. A 4 to 5% isotonic solution is needed to raise the osmotic pressure high enough to pull out solute and water from body compartments. It can remove as much as a liter or more of water per hour. Thus, urea clearance is raised (12).

If the dialysis fluid is at body temperature, there is 35% more urea clearance than if the fluid is at 20°C. Heat increases vasodilation and blood flow, improving clearance. Larger clearances are obtained from higher concentrations of urea in the blood.

A plastic or Silastic catheter is inserted through the peritonium. The area around the catheter must be covered with sterile dressings as well as all equipment used must be sterile and handled aseptically. The tubing and containers must be sterile. The inflow and outflow systems should be closed to the atmosphere.

The abdomen is shaved, washed and disinfected. About 1 to 2 liters of dialysis fluid is administered by an 18 gauge needle inserted into the peritoneal cavity to distend the abdomen to prevent injury to the intraabdominal organs before the cannula is inserted. The catheter should lie posteriorly to the paravertebral area and extend into the pelvis. Dialysis fluid is allowed to run through the catheter in positioning it. The catheter should be rotated and inserted until there is a free flow in and out of the cannula. The catheter is fixed tightly to the skin by a wire suture. Then a sterile dressing is applied.

An alternate method is the use of commercially prepared kits. The catheters can be used for two weeks to three months. A "device" may be inserted into the anterior abdominal wall so a catheter could be inserted through it. Another method of catheter insertion is to place the catheter directly into the abdominal cavity.

Peritoneal dialysis fluid. The composition of the fluid must be sterile (12). Glucose is 1.5%, 4%, or 7% concentrations in the fluid. The higher concentrations remove more fluid by osmotic force.

Sodium bicarbonate is added to dialysis fluid if the patient is acidotic. However, this also increases the sodium in the abdominal cavity. This is especially important if congestive heart failure is a complication.

Potassium should conform with the patients serum level. However, no potassium is used in acute renal failure. Heparin is added to prevent fibrin formation in the cannula, in an amount of 300 units per liter of fluid. However, it is not required (42).

Commercial preparations are available so that sodium, calcium, magnesium, potassium or chloride cations may be removed (12, 47). Usually, 2 liters of fluid is put into the abdominal cavity and left for 30 minutes to equilibrate (12). The fluid requires 20 minutes to drain out by the use of gravity. However, the fluid may be put into the abdominal cavity through one catheter; the dialysis machine can recirculate the dialysate through the peritoneum, then out another catheter, then in again after being reconstituted.

Peritoneal dialysis makes the removal of excess water easier sometimes. However, as much as 0.5 g of protein and amino acids are lost in each liter of dialysate (12, 42).

Peritoneal dialysis complications. The same problems that are present in hemodialysis are present in peritoneal dialysis. However, infections in and around the cannula may develop in peritoneal dialysis. They are treated by the addition of antibiotics to the dialysis fluid. Adhesions or destruction of

parts of the peritoneal space make further peritoneal dialysis impossible. Leakage of dialysate may be a problem that upsets fluid balance. There is a loss of protein; with an average loss of 10_g to 40_g/dialysis (48). There may be bleeding around the cannula, that goes to the peritoneal cavity. Peritoneal dialysis may be a preferred method of dialysis when bleeding or hypotension is present (48).

HOME DIALYSIS

Lowrie and his colleagues (49) found renal failure patients put on home hemodialysis had a lower survival rate than patients receiving a living sibling donor kidney after one years time. Home hemodialysis patients survived longer than those receiving a cadaver kidney transplant. Recipients of kidneys from parents had an 84.2% survival rate after one year; transplants from siblings survival rate was even higher with an 89.5%. Cadaver transplanted kidney patients only survived 68.7% of the time. One year's survival rates for patients on home dialyzers was 88.5%. After two years, the rate dropped to 77.8%.

On the other hand, patients on hemodialysis within a research center showed a survival rate of 92.9% after one year and 86.1% after two years duration (49).

In another study done by Neff (50), outpatient home dialysis has been successful in prolonging life. Dependencies on the families have been created. Financial and social burdens have been placed on patients. Successful home hemodialysis requires

a stable home with a cooperative stable assistant to help motivate the patient, who fully understands the treatment in general.

SUMMARY

The incidence of acute renal failure is high. It is not known how high, because of its difficult detection and association with cardiovascular diseases.

The normal kidney functions primarily through individual nephrons within the two kidneys. The kidneys have three main functions. They include: the formation and excretion of urine, the maintenance of electrolytic balance and the regulation of acid-base balance. End products of metabolism are collected in the blood. The blood is filtered through the glomerulus and renal tubules. Some constituents are returned to the blood, while others are kept and passed on as urine. In this way, electrolytes and fluids are balanced, because of the concentrating power of the kidney.

The kidneys cease to function correctly in the diseased state. The glomerulus becomes increasingly perforated and deteriorates in glomerulonephritis. End products of metabolism are released into the extracellular fluid, or may become blocked as debris in the tubules.

Dietary treatment used in acute glomerulonephritis includes: a low protein diet with a sodium restriction. Chronic glomerulonephritis requires an intravenous alkaline diuretic solution.

Mannitol intravenous solutions may be used, as well as glucose and inulin intravenous injections.

Tubular necrosis involves the death of tissue in the renal tubules. It may happen because of nephrotoxicities, the crushing syndrome or blood transfusion reactions. Uremia results. Many diets have been given for uremic patients. They include: the Giovannetti Maggiore diet; an intravenous mannitol diet; sodium and fluid restriction diet; amino acid, fructose and ethanol diet; L-amino acid, glucose, and vitamin supplemented diet, and L-amino acid and hypertonic dextrose diet. Most recently, an intravenous L-amino acid, glucose and vitamin supplemented diet is used with dialysis to improve kidney functioning.

Dialysis has restored many patients to normal kidney functioning after acute renal failure. Hemodialysis employs the use of an artificial kidney; whereas, peritoneal dialysis is an infusion of solutes into the peritoneal cavity. Home hemodialysis machines are now used as an outpatient treatment for acute renal failure. The patients require constant supervision, usually from a family member; but are being used successfully.

In conclusion, the influence of a proper diet, the use of hemodialysis and peritoneal dialysis has increased the life expectancy for acute renal failure patients significantly within the last 10 to 20 years.

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LITERATURE CITED

1. Weir, E. C. (1971) An evaluation of research in the United States on human nutrition--Division of Agricultural Research Service. Benefits from Nutrition Research, Human Nutrition Research, Report #2, United States Department of Agriculture, pp. 9, 106.
2. Lordon, R. E. & Burton, J. R. (1972) Post-traumatic renal failure in military personnel in Southeast Asia. Amer. J. Med. 53, 137.
3. Guyton, A. C. (1971) Formation of urine by the kidney: II. Regulation of urine concentration; autoregulation of renal blood flow; and the concept of clearance. In: Textbook of Medical Physiology, ed., W. B. Saunders Company, Philadelphia, Penn., p. 406.
4. Frenay, A. C. (1973) Urogenital disorders. In: Understanding Medical Terminology, ed. 2, The Catholic Hospital Association, St. Louis, Mo., p. 167.
5. Corcoran, A. C., Hines, D. C. & Page, I. H. (1961) In: Kidney Function in Health, Kidney Function in Disease, ed., Lilly Laboratory for Clinical Research, Indianapolis, Ind., p. 2.
6. Mitchel, H. S., Rynbergen, H. J., Anderson, L. & Dibble, M. V. (1968) Diseases of the kidney; kidney stones. In: Cooper's Nutrition in Health and Disease, ed., J. B. Lippincott Company, Philadelphia, Penn., p. 258.
7. Memmler, R. L. & Rada, R. B. (1970) In: The Human Body in Health and Disease, ed., J. B. Lippincott Company, Philadelphia, Penn., p. 258.
8. Garrett, T. A. (1969) In: The Fundamentals of Body Water and Electrolytes, ed., Baxter Laboratories, Morton Grove, Ill., p. 1.
9. Guyton, A. C. (1969) Formation of urine by the kidney, and micturition; regulation of body fluid constituents and volumes. In: Function of the Human Body, ed., W. B. Saunders Company, St. Louis, Mo., p. 195.
10. Best, C. H. & Taylor, N. B. (1961) The excretion of urine. In: The Physiological Basis of Medical Practice, ed., The Williams and Wilkins Company, Baltimore, Mass., p. 532.

11. Orten, J. M. & Neuhaus, O. W. (1970) Hormones; biochemistry of specialized tissues and body fluids. In: Biochemistry, ed., The C. V. Mosby Company, St. Louis, Mo., pp. 470, 658.
12. Rosenberg, J. C. (1972) Dialysis as a substitute for renal function in surgical patients. In: Current Problems in Surgery, ed., Year Book Medical Publishing Company, p. 3.
13. Hollenberg, N. H., Adams, D. F., Oken, D. E., Abrams, H. L. & Merrill, J. P. (1970) Acute renal failure due to nephrotoxins. N. Engl. J. Med. 282, 77.
14. Davson, H. (1964) In: A Textbook of General Physiology, ed., Little, Brown, and Company, Boston, Mass., p. 559.
15. McCoy, R. C. & Tisher, C. C. (1972) Glomerulonephritis associated with sarcoidosis. Am. J. Pathol. 68, 339.
16. Flamenbaum, W. (1973) Pathophysiology of acute renal failure. Arch. Intern. Med. 131, 911.
17. Martinez-Maldonado, M., Eknoyan, G. & Suki, W. N. (1973) Diuretic in nonedematous states. Arch. Intern. Med. 131, 797.
18. Lloyd, W. D. (1965) In: Dorland's Illustrated Medical Dictionary, ed. 24, W. B. Saunders Company, Philadelphia, Penn., p. 1.
19. Kavnat, P. E., Lavovita, E. & Levison, S. P. (1973) Antibiotics and the kidney. Med. Clin. North Am. 57, 1045.
20. Burgess, J. L. & Birchall, R. (1972) Nephrotoxicity of amphotericin B, with emphasis in tubular function. Am. J. Med. 53, 77.
21. Giovannetti, S. & Maggiore, Q. (1964) A low-nitrogen diet with proteins of high biological value for severe chronic uremia. Lancet 1, 1,000.
22. Massry, S. G., Friedler, R. M. & Coburn, J. W. (1973) Excretion of phosphate and calcium. Arch. Intern. Med. 131, 839.
23. Schultze, R. (1973) Recent advances in the physiology of potassium excretion. Arch. Intern. Med. 151, 893.
24. Dudrick, S. J., Steiger, E. & Long, J. M. (1970) Renal failure in surgical patients. Treatment with intravenous essential amino acids and hypertonic glucose. Surgery 68, 180.

25. Karasaura, Y., Tasaki, I., Yokota, H. & Shibata, F. (1973) Comparative effect of intravenously administered nitrogenous compounds on uric acid synthesis in chickens fed a 20% protein diet. *J. Nutr.* 103, 1208.
26. Prior, R. L., Clifford, A. L. & Visek, W. J. (1973) Growth and metabolism of rats fed non-protein nitrogen diets supplemented with nicotinimide, succinate and pyridoxine. *J. Nutr.* 103, 1099.
27. Scarbieri, V. C., Amaya, J., Tanaka, M. & Chichebter, C. O. (1973) Response of rats to amino acid supplementation of brown egg albumin. *J. Nutr.* 103, 1731.
28. Young, V. R., Taylor, Y. S. M., Rand, W. M. & Scrimshaw, N. S. (1973) Protein requirements of man: efficiency of egg protein utilization at maintenance and submaintenance levels in young men. *J. Nutr.* 103, 1164.
29. Prothro, J., Mackellar, I., Reyes, N., Linz, M. & Chow, C. (1973) Utilization of nitrogen, energy, and sulfur by adolescent boys fed three levels of protein. *J. Nutr.* 103, 786.
30. Giordano, C. (1963) Use of exogenous and endogenous urea for protein synthesis in normal and uremic subjects. *J. Lab. Clin. Med.* 62, 231.
31. Giordano, C., DePascale, C., Balestrieri, C., Cittadini, D. & Crescenzi, A. (1968) Incorporation of urea N in amino acids of patients with chronic renal failure on low nitrogen diet. *Am. J. Clin. Nutr.* 21, 394.
32. David, D. S. (1973) Dietary treatment of renal failure. *Am. Heart J.* 86, 1.
33. David, A. S., Hochgelerent, E., Rubin, A. L. & Stenzel, K. H. (1972) Dietary management in renal failure. *Surgery* 2, 34.
34. Berlyne, G. M., Gaan, D. & Ginks, W. R. (1968) Dietary treatment of chronic renal failure. *Am. J. Clin. Nutr.* 21, 547.
35. Harrington, J. T. & Cohen, J. J. (1973) Clinical disorders of urine concentration and dilution. *Arch. Intern. Med.* 131, 822.
36. Peaston, M. J. T. (1973) Dangers of intravenous fructose. *Lancet* 1, 266.

37. Abel, R. M., Abbott, W. M. & Fischer, J. E. (1971) Acute renal failure. *Arch. Surg.* 103, 513.
38. Abel, R. M., Abbott, W. M. & Fischer, J. E. (1972) Intravenous essential L-amino acids and hypertonic dextrose in patients with acute renal failure. *Am. J. Surg.* 103, 632.
39. Abel, R. M., Beck, C. H., Jr., Abbott, W. M., Ryan, J. A., Jr., Barnett, G. O. & Fischer, J. E. (1973) Acute renal failure: treatment with intravenous amino acids and glucose. *N. Engl. J. Med.* 288, 695.
40. Blainey, J. D. & Chamberlain, M. J. (1971) Dietary treatment of chronic renal failure. *Br. Med. Bull.* 27, 160.
41. Kopple, J. D., Sorensen, M. K., Coburn, J. W., Gordon, S. & Rubini, M. E. (1968) Controlled comparison of 20-g and 40-g protein diets in the treatment of chronic uremia. *Am. J. Clin. Nutr.* 21, 553.
42. Tzamaloukas, A. H., Garella, S. & Chazan, J. A. (1973) Peritoneal dialysis for acute renal failure after major abdominal surgery. *Arch. Surg.* 106, 639.
43. Najarian, J. S. & Simmons, R. L. (1972) In: *Transplantation*, ed., Lea and Fibiger, Philadelphia, Penn., p. 423.
44. Penington, D. G. & Kincaid-Smith, P. (1971) Anaemia in renal failure. *Br. Med. Bull.* 27, 136.
45. Weiss, S. W., Taw, R. L. & Hutchins, G. M. (1973) Constructive uremic pericarditis following hemodialysis for acute renal failure. *Hopkins Med. J.* 132, 301.
46. Anonymous (1973) Prognosis of acute renal failure. *Br. Med. J.* 2, 435.
47. Rae, A. & Pendray, M. (1973) Advantages of peritoneal dialysis in chronic renal failure. *JAMA* 225, 937.
48. Berlyne, G. M., Jones, H. J., Hewitt, V. & Nitwarangur (1964) Protein loss in peritoneal dialysis. *Lancet* 1, 738.
49. Lowrie, E. G., Lazarus, J. M., Mocelin, A. J., Bailey, G. L., Hampers, C. L., Wilson, R. E. & Merrill, J. P. (1973) Survival of patients undergoing chronic hemodialysis and renal transplantation. *N. Engl. J. Med.* 288, 863.
50. Neff, M. S. (1973) Outpatient dialysis. *Arch. Intern. Med.* 131, 717.

DIET AND DIALYSIS IN ACUTE RENAL SHUTDOWN

by

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Acute renal shutdown suggests a serious malfunction in the normal filtration of blood by the kidneys. Renal dialyses and dietary regimen are used in treatment. The malfunction centers around individual nephrons with deterioration of glomeruli and/or the renal tubules. The unfiltered end products of metabolism re-enter the body fluids with a resulting uremia.

Dietary treatment for acute renal shutdown, includes the use of several diets, depending on the nature and severity of the damaged nephrons. Generally speaking, intravenous injections of electrolytes and a simple sugar is utilized. Fluids are restricted. Sodium is restricted, if edema is present. A low protein diet is administered as soon as the patient can physically tolerate it.

Hemodialysis or peritoneal dialysis may be used in addition to the diet, or may be used in place of it. Nutrients, in the form of a simple sugar, as glucose, and electrolytes can be added to the dialysate to function as a temporary diet plan.

However, dialysis serves its major function as an artificial kidney. It filters waste products from the blood and restores the electrolytic and acid-base balance in the blood to as close to normal as possible.

The use of a proper diet and dialysis has been shown to lengthen the life expectancy of patients with acute renal shutdown.