Renal Failure and the Nurse's Role

An Honors Thesis (ID 499)

By

Nancy Cortright

Thesis Director

Mr. Richard Hakes

Ball State University

Muncie, Indiana

May 1979
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>ANATOMY AND PHYSIOLOGY</td>
<td>2</td>
</tr>
<tr>
<td>External Structures</td>
<td></td>
</tr>
<tr>
<td>Internal Structures</td>
<td></td>
</tr>
<tr>
<td>Blood Supply</td>
<td></td>
</tr>
<tr>
<td>Main Functions</td>
<td></td>
</tr>
<tr>
<td>ACUTE AND CHRONIC RENAL FAILURE</td>
<td>9</td>
</tr>
<tr>
<td>CAUSES OF RENAL FAILURE</td>
<td>10</td>
</tr>
<tr>
<td>Pre-renal</td>
<td></td>
</tr>
<tr>
<td>Renal</td>
<td></td>
</tr>
<tr>
<td>Post-renal</td>
<td></td>
</tr>
<tr>
<td>STAGES OF RENAL FAILURE</td>
<td>16</td>
</tr>
<tr>
<td>CLINICAL MANIFESTATIONS OF RENAL FAILURE</td>
<td>17</td>
</tr>
<tr>
<td>COMPLICATIONS OF RENAL FAILURE</td>
<td>20</td>
</tr>
<tr>
<td>Metabolic Alterations</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular Alterations</td>
<td></td>
</tr>
<tr>
<td>Hematological Alterations</td>
<td></td>
</tr>
<tr>
<td>Respiratory Alterations</td>
<td></td>
</tr>
<tr>
<td>Bone Alterations</td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal Alterations</td>
<td></td>
</tr>
<tr>
<td>Dermatologic Alterations</td>
<td></td>
</tr>
<tr>
<td>Neurologic Alterations</td>
<td></td>
</tr>
<tr>
<td>MEDICAL TREATMENTS AND NURSING INTERVENTION</td>
<td>28</td>
</tr>
<tr>
<td>Fluid and Diet</td>
<td></td>
</tr>
<tr>
<td>Dialysis</td>
<td></td>
</tr>
<tr>
<td>Transplants</td>
<td></td>
</tr>
<tr>
<td>CONCLUSION</td>
<td>41</td>
</tr>
</tbody>
</table>
INTRODUCTION

The nurse's role in the management of patients in renal failure, is becoming an increasingly complex and crucial one. Due to many scientific advancements in recent years, the possibilities in caring for patients in renal failure have greatly increased. For example, successful kidney transplants have come into use just within the past fifteen years.

The challenge to nursing in considering a multitude of variables and interrelationships in assessing and planning nursing interventions is enormous. Knowledgeable and skilled nurses must assist the patient through the stages of renal failure and assist the patient to achieve his highest level of rehabilitation.

In the first part of this paper, the anatomy and physiology of the renal system will be reviewed. Explanations will be given of chronic and acute renal failure in the second section. In the third part the causes of renal failure will be reviewed. Then the stages of renal failure will be discussed. Complications of renal failure and their associated nursing implications consist of the fourth section. Lastly, the medical treatments and associated nursing interventions will be explained.
External Structures

The kidneys lie opposite the bodies of the first and second lumbar vertebrae. They begin just under the eleventh rib. The kidneys are shaped like lima beans, the convex border on the lateral side and the concave border is turned toward the vertebral column. The right kidney may lie slightly lower than the left.

The kidney measures about 11.25 centimeters lengthwise, 5 to 7.5 centimeters wide, 2.5 centimeters thick, and weighs from 125 to 170 grams in the adult male and 115 to 155 grams in the female.¹

A medially directed concavity is termed the hilus; it leads to the renal sinus. The blood vessels, nerves, and ureters enter and exit the organ at this region.

The kidney is embedded in a mixture of perirenal fat and fibrous tissue called the renal fascia. The renal capsule is though, fibrous tissue that envelops the surface of the kidney. The kidneys are retroperitoneal.²

Internal Structures

In longitudinal section the exterior third of the kidney has a typical brownish red color. This is the cortex. A number of more or less separate red triangular masses, the renal pyramids, with apexes pointed away from the cortex, penetrate the interior two-thirds of the kidney, which is the medulla. Cortical substance dips deeply between adjacent pyramids.³
Each kidney contains about a million nephrons. These drain into a treelike arrangement of collecting tubules that convey the urine to the renal pelvis and thence by way of the ureters to the bladder. Urine is ejected from the bladder through the urethra by the process called micturition or urination.

The renal pelvis is funnel shaped, with two or three main divisions, the major calyces, which drain the upper and lower halves of the kidney. These in turn divide into several smaller conduits, the minor calyces, each of which terminates around the base of one or more urinary papillae in the form of a calyx, or cup, into which the urine is delivered. The major and minor calyces contain circular smooth muscle fibers whose contractions propel the urine into the ureter whence further contractions force it into the bladder.4

Nephrons are the microscopic and mainly tubular structural and functional unit of the kidneys. It is estimated that there are one to one and a half million nephrons in each kidney. A nephron begins with a double walled cup termed the glomerular or Bowman's capsule. The inner wall of this capsule is known as the visceral layer, and it follows the twists and turns of the glomerular capillary network. It is formed of highly modified cells known as podocytes.5 The outer or parietal layer of the capsule is a simple squamous epithelium and lies a short distance from the visceral layer so that an actual space between the two layers is created. The capsule and the contained glomerulus form a unit designated as the
renal corpuscle. Leading from the capsule is a proximal convoluted tubule in which the cells are cuboidal, have central nuclei, and have a brush border on the lumen side. The brush border consists of minute cytoplasmic extensions of the cell called microvilli. These serve to increase the surface area for reabsorption and or secretion of materials by this part of the nephron.

The proximal tubule then becomes straight as it nears the medullary region. The cells become flat, and the tube narrows and dips toward or into a pyramid as the descending loop of Henle. Then the tube bends back upon itself, and enlarges. Its cells become rectangular and the ascending loop of Henle returns to or toward the cortical region. In the cortex, the tube again becomes straight and then convoluted and is known as the distal convoluted tubule. Its cells are cuboidal, have central nuclei, and carry no brush border. This portion joins a collecting tubule. The collecting tubules receive the distal terminations of many nephrons. They open into the calyces of the pelvis through the papillary ducts.

**Blood Supply**

Blood is supplied to the kidney by the abdominal aorta through one renal artery to each kidney. Interlobar arteries, which are from the renal artery, lie between the renal pyramids. These form a series of incomplete arches, the arciform, or arcuate arteries, crossing the base of the pyramids. Interlobular arteries radiate laterally toward the surface of the kidney from the arciform vessels. The interlobular
arteries have branches which are the afferent vessels to the glomeruli. Efferent vessels from the glomeruli break up into a second set of capillaries about the area of the convoluted tubules. From these blood passes through the venous system and out the renal veins into the inferior vena cava.

**Main Functions**

The kidney's primary function is that of regulation and concentration of solutes in the extracellular fluid of the body. This function is performed by the removal of metabolic waste and excess concentrations and by conserving those substances which are present in normal or low quantities.

The production of urine begins with filtration through the glomerular capillaries of a fluid that resembles plasma. This filtrate then passes down the tubules, and its components and volume are altered by the tubules' ability of secretion and absorption.

The glomerular membrane is composed of all the membranes of a group of capillaries. This capillary bed, in some respects, is like others throughout the body, and in other respects differs. Like other capillary membranes, it is basically impermeable to plasma proteins. They differ in that the glomerulus has an increased permeability to water and small molecular solutes.

The glomerular membrane is well adapted for effective filtration by three distinguishing characteristics. First, the tufts of capillaries from in the glomeruli are very numerous; in consequence, the surface for filtration in the kidneys
is very large. Secondly, the vas efferens taking the blood from the glomerulus is smaller than the vas afferens and breaks up into a second set of capillaries around the convoluted tubules. A great amount of resistance to the outflow of the blood from the glomerulus is produced. Therefore, a high blood pressure in the glomerulus is produced. This blood pressure is estimated at 60 to 70 mm Hg in comparison to other capillaries which run from 10 to 25 mm Hg. Thirdly, the membrane that separated the blood from the cavity of the capsule is extremely thin, around 0.1 micron thick. As the result of these three factors, the flow of fluid through the capillary wall of the glomerulus is estimated to be more than a hundred times that through the walls of other capillaries throughout the body.\textsuperscript{11}

The glomerular filtration rate is the rate at which fluid flows from the glomerulus into Bowman's capsule. This rate is directly proportional to the net filtration pressure. On the average this rate is about 125 cc of filtrate being formed per minute. This results in about 180 liters per day. Nearly all but one liter of this is reabsorbed into the blood.\textsuperscript{12}

The excretory function of the kidney begins with the glomerular filtrate entering the proximal convoluted tubule with a solute concentration of 300 milliosmols. From 60 to 70\% of the sodium ions are actively transported into the interstitial space. An equivalent of chloride and water diffuse out of the tubule in response to the electrical and osmotic gradients established by the active transport of sodium ions. All the
glucose and amino acids that enter the tubules are completely reabsorbed and do not appear in the urine. Some of the urea, phosphate, and bicarbonate are reabsorbed into the interstitium, and from there goes back into the blood. All of the potassium ions are actively reabsorbed in the proximal tubules.

The filtrate then travels down the descending limb of the loop of Henle and through the loop of Henle to the ascending limb. While at the ascending limb 20 to 30% of sodium in the filtrate is transported out into the interstitial space. The cells of the ascending limb are impermeable to water, therefore, the filtrate goes from isotonic in the proximal tubule to hypertonic in Henle's loop and reaches the distal convoluted tubule as hypotonic.

When the filtrate reaches the distal tubule, 8 to 10% of the sodium is reabsorbed under the influence of aldosterone. This action takes place for the exchange of a potassium or hydrogen ion. If the antidiuretic hormone is present, the sodium is followed by an equivalent amount of water. Also, while in the distal tubule, bicarbonate is reabsorbed out of the tubule and back into the blood.

As the filtrate leaves the distal tubule and enters the collecting duct, it is now urine. If antidiuretic hormone is now present, water will be reabsorbed here and the urine will become more concentrated. The urine now contains foreign proteins that have entered the blood and certain excess catabolic products, such as urea, uric acid (as urates), creatinine, hippuric acid and various salts such as nitrates, sulfates,
and phosphates.\textsuperscript{16}

The kidneys also aid in the maintenance of osmotic pressure and fluid volume. This is accomplished through conserving or eliminating water or salt. When the osmolarity of the extracellular fluid and plasma exceeds the normal value, water is conserved. When the concentration is less, water is eliminated. Antidiuretic hormone output is stimulated by increases in the extracellular osmolarity and is inhibited by decreases.\textsuperscript{17} Antidiuretic hormone is released from the posterior pituitary when the osmoreceptors in the hypothalamus are stimulated.

Maintenance of acid-base balance is an important function of the kidneys. Non-volatile acids such as phosphoric and sulfuric acids are excreted. Organic acids, such as ketone bodies, are also excreted. Hydrogen ions are actively secreted into the tubular fluid by the cells of the proximal and distal tubules and the collecting ducts. These ions are exchanged for sodium ions in the tubular fluid. If the buffer in the filtrate is limited, then excess hydrogen ions can be excreted after combination with ammonia ions.\textsuperscript{18} If the pH of the extracellular fluids is too high a greater quantity of bicarbonate buffer is filtered. The reverse occurs when the pH is too low.

The manufacture of red blood cells takes place in the bone marrow, especially the marrow of the vertebrae and flat bones in the adult human. The kidney produces and secretes an enzyme in response to low partial pressure of oxygen in
renal arterial blood. This substance stimulates the bone marrow to produce more erythrocytes.  

Renin is a hormone produced by a group of cells next to the glomeruli called the juxtaglomerular apparatus. Renin has its main function in the control of blood pressure. It is converted by the substance angiotensin I to angiotensin II which causes arteriolar vasoconstriction. This process results in increased blood pressure. Renin is secreted under the following conditions: hypotension, hypovolemia, hyponatremia, decreased glomerular filtration rate, renal ischemia, renal artery stenosis, and hemorrhage.

Regulating calcium metabolism is another function of the kidney. It transforms vitamin D from a less active form to a more active metabolite. This active form of vitamin D is necessary to absorb calcium from the gastrointestinal tract. Without this, hypocalcemia would develop.

ACUTE AND CHRONIC RENAL FAILURE

Renal failure is the term used when a patient's renal function has become decreased to such an extent that the patient begins to develop symptoms. The kidneys are no longer able to maintain the volume or composition of body fluids under normal dietary conditions. Renal failure implies disease of both kidneys, as one normal or nearly normal kidney has sufficient function to maintain normal fluid balance.

Acute renal failure is differentiated from chronic renal failure. Acute renal failure has an relatively rapid onset
developing over a period of days or weeks. It is often a reversible process, but in certain cases it may not be corrected. Chronic renal failure damage is basically permanent and irreversible. Chronic failure may be a stable state of decreased function lasting for months or years, often progressing slowly to terminal renal failure.\(^\text{24}\)

**CAUSES OF RENAL FAILURE**

The causes of both acute and chronic renal failure, may be classified according to the location of injury. The pre-renal causes will be considered first. Secondly, the renal factors leading to failure will be discussed. Lastly, the post-renal causes of failure will be examined.

**Pre-renal**

When the blood supply to the kidney is reduced due to hypovolemia, hemorrhage, myocardial infarction, shock, burns, or other trauma, the renal perfusion is greatly reduced. When the renal plasma flow has fallen to \(5\%\) of its normal value, renal ischemia and oliguria result.

Another pre-renal cause of failure is vascular obstructions. Renal arterial obstruction, although rare, may result from atheromatous disease of the aorta, renal arterial embolism, renal artery thrombosis, or avulsion of the renal pedicle. The decreased blood supply causes the extensive infarction of the kidney.\(^\text{25}\)
Renal

The second group of renal failure causes is due to renal parenchymal diseases. The basic site of damage is to the functioning nephron units of the kidney. One of these diseases is glomerulonephritis. Glomerulonephritis has various types, but all of these have two common factors. Firstly, the glomeruli are in some way abnormal so that protein leaks into the tubules. Therefore, proteinuria is one main sign. Secondly, the basic trouble in all cases is an antigen-antibody reaction of the glomeruli. The exact mechanism of this allergic or autoimmune reaction is not clearly understood. One antigen which has been linked to glomerulonephritis, is the hemolytic streptococcus organism. Other microorganisms, certain drugs and poisons have also been identified with the initial allergic insult. This disease is usually contained, with full recovery occurring. However, if oliguria occurs and continues for more than three days, irreversible glomerular damage may occur. The prognosis improves if no hematuria is present.26

The symptoms of acute glomerulonephritis often include proteinuria, edema, hematuria, oliguria, hypertension, headache, and increased blood urea nitrogen levels. If no recovery occurs, the disease develops into subacute or chronic glomerulonephritis. Hemoptysis, retinopathy, and death from uremia or lung hemorrhage occurs in the subacute form. The chronic form of glomerulonephritis may develop without an acute episode and develop insidiously. Proteinuria and progressive
hypertension may be present for many years, or death from uremia may develop rapidly.\textsuperscript{27}

Histologically, glomerulonephritis is divided into three main types. These are minimal change, proliferative, and membranous.

Minimal change is the term used when damage is so slight, that the glomeruli appear normal with only minor abnormalities. Proliferative means that there is a proliferation of various cells in the region of the glomeruli. Proliferative appears in all grades of severity with glomerulonephritis. The glomeruli look abnormally cellular, and the proliferation is either focal or diffuse. When the glomerular capillary membrane walls look thick and the capillary lumen narrows, it is termed membranous. Histologically, there exist a fourth type called membrano-proliferative. This is when membranous and proliferative changes are combined in the same glomeruli.\textsuperscript{28}

Renal involvement in the form of glomerulonephritis is a frequent accompaniment of the collagen diseases, such as systemic lupus erythematosus, polyarteritis nodosa, Wegener's granulomatosis and scleroderma. In the absence of treatment, once renal involvement occurs with collagen diseases, it nearly always leads to renal failure.\textsuperscript{29}

Pyelonephritis is an inflammation of the kidney caused by a bacterial infection of the kidney substance and renal pelvis. Occasionally the infection is carried to the kidneys in the blood, however, it is usually an ascending infection due to organisms that have reached the bladder and refluxed into the
ureter. Usually the infection affects the interstitial tissues surrounding the tubules in the medulla.\textsuperscript{30}

*Acute pyelonephritis may be asymptomatic or may cause minimal symptoms.* Usually though, there are symptoms of this form of pyelonephritis. The patient will need to void frequently, have a burning sensation in the urethra, severe flank pain, chills, fever and weakness. The urine contains many bacteria, pus cells, and red blood cells. The urine may also be cloudy and have a foul smell.\textsuperscript{31}

In *chronic pyelonephritis* bacterial inflammation of the kidney has left scarring, fibrosis, and tubular dilation. The intervening tissue may be quite healthy. At this stage the urine may not contain bacteria, instead diagnosis is made by intravenous pyelogram and renal biopsy. The patient is usually hypertensive and has signs of renal failure.\textsuperscript{32}

Hypertension may also lead to renal failure. In the kidneys progressive thickening of arterial walls, narrowing of the lumen, and progressive ischemia of the renal tissue occurs. The ischemia tends to lead to increased renin secretion which further aggravates the hypertension. Symptoms of nephrosclerosis range from mild to severe proteinuria, hematuria, malignant hypertension, or retinal hemorrhage.\textsuperscript{33}

Nephrotoxic agents may lead to episodes of renal papillary necrosis. This in turn leads to scarring and fibrosis in the medulla, which is called interstitial nephritis. Heavy metals such as mercury and uranium, and exogenous poisons such as carbon tetrachloride and the phenols are nephrotoxic.
agents. Analgesics, such as phenacetin mixtures and antibiotics such as tetracyclines, neomycin, ampicillin, sulfonamides, kanamycin, cephaloridine, and colistin may cause damage to the renal tubules. If the toxin is not withdrawn in time irreversible kidney damage may result.  

Patients with diabetes mellitus may develop a renal lesion called Kimmelstiel-Wilson lesion or diabetic glomerulosclerosis. Renal biopsy reveals extensive glomerular and arteriolar involvement. The glomeruli gradually lose their delicate structure and become replaced by shapeless masses of tissue which can no longer act as filters. Taking years to develop, it is characterized by proteinuria which becomes persistent and heavy. Renal failure eventually occurs. 

Renal failure due to polycystic kidney occurs as each kidney slowly becomes a mass of large numbers of fluid filled cysts which compress and destroy the healthy renal tissue. Frequently there are episodes of hematuria due to bleeding from the cysts, and there may be pain from time to time caused by bleeding into the cysts or due to clot obstruction of the ureters. Polycystic kidney is considered an hereditary disease. 

Another disease that causes renal failure due to direct parenchymal damage, is gout. Gout is an abnormality due to abnormally high levels of uric acid in the blood. Beside damaging the joints, it also may have a progressive destructive effect on renal tissue. Basically the tubular and interstitial tissue is damaged and replaced by scar tissue.
Also, urate crystals may aggregate to form calculi which cause obstructions in the ureter.

Tuberculosis and neoplasms are both rare, but still may occur. Tuberculosis causes progressive renal destruction. In tuberculosis the symptoms do not appear until late, at which time burning and frequency of urination occur. Symptoms also do not occur until late in neoplasms, at which time gross hematuria without pain occurs.

Post-renal

Post-renal causes of renal failure deal with the urinary system below the renal pelvis. Urinary tract infections fall under this group. Lower urinary tract infections are a danger because they ascend up the ureters to the kidneys where pyelonephritis may result. This infection is associated with frequency, and dysuria.

Renal or bladder calculi are often composed of a calcium salt. They are caused either by an excess excretion of calcium in the urine or by patients with an alkaline urine which allows the calcium to settle out of the urine and crystallize. Some stones are composed of uric acid which were previously discussed with gout. Some of these stones are small and easily voided. When the stone is very large it may block the ureter causing hydronephrosis due to the obstruction to the flow of urine. The renal pelvis dilates and the increased pressure may cause extreme damage to the kidney. Due to stagnant and refluxed urine, the risk of developing pyelonephritis is greatly increased. There may be no symptoms of calculi unless
a large stone enters the ureter. When this occurs severe flank pain, sweating, pallor, nausea and vomiting may accompany it.\textsuperscript{41}

Urinary obstructions also occur due to prostatic hyper trophy, tumors of the bladder, prostate, or pelvis, and strictures of the urethra and ureters. All of these may lead to the retension of urine, hydronephrosis, and kidney destruction.

**STAGES OF RENAL FAILURE**

The course of renal failure is divided into four phases. First is the onset phase. This is when the precipitating event responsible for the damage occurs. If the insult continues and is not stopped, the oliguric or anuric phase begins. Total anuria is rare and may indicate obstruction of the urinary tract. Oliguria is an output below 500 cc per 24 hours.\textsuperscript{42} This phase averages around two weeks. Symptoms include protein, red blood cells, and casts in the urine. The urine's specific gravity and serum sodium are low. If this phase last beyond two weeks uremia and hypertension develop. This phase may progress to permanent loss of renal function.

The early diuretic phase begins as urine output rapidly increases over a number of days. The glomerular filtration rate usually remains very low because the kidneys are not yet fully healed. As a result, the blood urea nitrogen usually does not decrease until the urine output reaches two liters per day. In fact, the blood urea nitrogen usually rises
In the late diuretic phase, the excretory function of the kidneys returns. The glomerular filtration rate begins to increase rapidly. The average patient recovers about 80% of his normal glomerular filtration rate. The blood urea nitrogen starts to fall until it is back to normal. The urinary volume increases and may exceed 3,000 ml daily. This massive diuresis causes a great loss of sodium and potassium. So daily electrolytes need to be monitored. Although proteinuria decreases during diuresis, tubular functions take longer to recover.

If no complications arise, the patient will enter the convalescent phase within six weeks. The blood urea nitrogen level is stable and normal kidney function resumes. In chronic renal failure, the damage is permanent and the patient will not reach these final phases. However, with correct management, acute failure is reversible and will reach these final phases.

**CLINICAL MANIFESTATIONS OF RENAL FAILURE**

The clinical manifestations of renal failure depend on several factors. These factors include the durations of the illness, the patient's age, the effectiveness of treatment and the presence or absence of other complications. Alterations occur as the kidney attempts to maintain normal function. Due to the hypertrophy and hyperphasia of the remaining normal nephrons, the nephrons will increase their workload ability.
This allows the kidney to maintain function at a normal limit. Only after destruction of 80% of the nephrons, does it become diagnostic.\(^4\)\\

Chronic renal failure can develop unnoticed over a period of years, or as result of a bout of acute renal failure from which the patient has recovered. Nocturia, polyuria and inability to concentrate the urine are early signs of failing kidneys. Extreme thirst develops during the day. Anemia makes the patient feel tired and lethargic. Headaches associated with hypertension, is a common early sign.\(^4\)\(^6\) The patient develops a bleeding tendency caused by uremia which leads to repeated epistaxis. Bruising from minor trauma, some degree of fluid retention, earthy pigmentation of the skin, and generalized itching of the skin may also appear.\\

Diagnostic test may reveal proteinuria and a fixed urine specific gravity in the region of 1.010. Elevations in serum creatinine and blood urea nitrogen, increasing anemia, a high serum phosphate and a reciprocally low serum calcium point toward chronic renal failure.\(^4\)\(^7\) Diagnosis is confirmed when X-rays show that the kidneys are small and contracted. Intravenous urogram and renal biopsy help to provide clues to the underlying cause.\\

Treatment of chronic renal failure is done by treating the cause and preventing further deterioration. For example, pyelonephritis is treated with antibiotics and its resulting hypertension is controlled by medication. Lower urinary tract infections are treated with antibiotics. There is no
treatment that will heal the glomerular lesions in glomerulonephritis. Treatment is directed toward controlling infections, hypertension and uremia. The alleviation of the symptoms of uremia is attempted with a low protein diet, drugs, and dialysis. Obstructions are treated by removal of the factor causing the obstruction. Other treatments include treating complications as they arise. A kidney transplant or dialysis will eventually be necessary.

In acute renal failure there is an abrupt loss of kidney function caused by various problems previously alluded to. The first symptoms of acute renal failure are a rapidly decreasing urinary output. The hourly volume of urine may fall to zero. Other symptoms include sudden oliguria, nausea, vomiting, and lethargy. There will be progressive increases in blood urea nitrogen, creatinine, potassium, and serum sulfate levels. Most patients complain of thirst and have a dry, crusted, foul mouth. Anorexia, nausea and vomiting are common symptoms. Diarrhea or ileus may also occur. Hyperkalemia and acidosis are also diagnostic tests that show signs of acute renal failure. There will also be a recent history of a cause of renal failure.

Treatment of acute renal failure is centered around treating complications and preventing deterioration of renal tissue. Dietary restriction of potassium intake, cationic exchange resins, and control of fluid intake may be initiated. Most patients require either peritoneal or hemodialysis sometime during the course of acute renal failure.
Metabolic Alterations

When a patient experiences renal failure, either chronic or acute, he may develop many complications. Some of these are caused due to metabolic alterations. Because of the decrease in urinary output the end products of nitrogen metabolism accumulate and the blood urea nitrogen and serum creatine rises. Hypoprotein uremia may occur because of proteinuria, decreased synthesis of protein due to the lack of essential amino acids, and decreased intake due to anorexia and nausea. Hypoprotein uremia makes the patient prone to edema due to decreased colloidal osmotic pressure. The patient may also experience malnutrition.52

Carbohydrate intolerance develops because the kidney's role in insulin degradation is impaired. Glucose utilization and insulin secretion, utilization, and degradation are all impaired. This glucose intolerance is distinct from that in conditions of diabetes mellitus. Marked hyperglycemia and ketosis do not occur.53

Lipid metabolism is markedly altered in the nephrotic syndrome. Hypercholesterolemia is the term used to describe this condition. In uremia the fatty acids and cholesterol are within normal limits; but the triglycerides are increased. Increased synthesis and impaired removal of the triglycerides cause the Type IV hyperlipoproteinemia that is often associated with vascular disease.54
An metabolic acidosis occurs due to the decreased ability of the tubules to excrete non-volatile and organic acids like ketone bodies. Also, there is a decreased ability to reabsorb bicarbonate added to the decreased formation of dihydrogen phosphate and ammonia. All of these lead to create metabolic acidosis. To replace lost kidney function while in uremia, the respiratory system becomes the major route of acid excretion. Bone demineralizes to help buffer the excess hydrogen ions; this in turn has a role in osteodystrophy.55

The nursing implications for metabolic alterations include the following interventions. Asses for metabolic alterations, such as protein levels, blood glucose levels, and blood gases. The patient may be placed on a low protein diet because protein may increase blood urea nitrogen levels and bring on uremia. Sodium bicarbonate may have to be administered due to metabolic acidosis.

If the renal failure is a result of burns, trauma, or surgery, amino acids, glucose, and insulin may be given by subclavian. This is done in an attempt to prevent severe nutritional deficiencies and the catabolism of tissue, which would increase the blood urea nitrogen and bring on uremia. In any case, providing the patient with calories in the form of fats and carbohydrates decreases the body's need to break down protein for energy and therefore, decreases the production of urea. The nurse should also observe for edema due to fluid retention and hypoprotein uremia.
Cardiovascular Alterations

Complications due to cardiovascular alterations may become very serious. Hypertension is the most frequent cardiovascular alteration. Renal ischemia and the decreased glomerular filtration rate lead to increased renin secretion; renin consequently increases the blood pressure further due to the renin angiotension mechanism. Aldosterone and antidiuretic hormone are released in greater quantities due to the decreased renal blood flow. The extracellular fluid volume increases until the body becomes overloaded with fluid. The acidotic state and the increased peripheral resistance causes left ventricular hypertrophy and congestive heart failure. Hypertensive encephalopathy may occur, accompanied by increased intracranial pressure, headaches, retinal changes, seizures, coma and cerebro-vascular accidents.56

Pericarditis is an sterile hemorrhagic and fibrinous inflammation of the pericardial sac. The reason why this occurs is unknown, but it usually occurs in patients with severe long standing uremia levels. The actual blood urea level at which it occurs is variable from patient to patient.57 A continuous pain in the front of the chest and a scratching sound called a pericardial friction rub can be heard with the use of a stethoscope. Hypotension and a low grade fever may also be present. Correction of the uremia usually is a cure for this condition. However, a patient occasionally will develop a large pericardial effusion. The heart sounds
will become distant and muffled and the blood pressure will fall due to the compressive effect of the fluid on the heart. A pericardial tap is usually necessary to allow the heart to function efficiently. An occasional late complication of uremic pericarditis is a thickening and fibrosis of the pericardium which restrains the heart and prevents normal contraction. This condition, called constrictive pericarditis, may be treated by surgical removal of the thickened membranes or it will lead to congestive heart failure.58

Vascular abnormalities occur as a result of the secondary hypertension. The arteries and arterioles become less elastic. Due to the increased amounts of triglycerides in the blood, increased platelet adhesiveness, and the fact that a patients in renal failure is usually immobilized, the patient is prone to develop phlebitis and thrombosis formation.

Cardiac arrhythmias also occur. These are the result of transient myocardial ischemia, electrolyte imbalances, decreased coronary perfusion, and cardiac damage.59

The nursing implications for the patient with cardiovascular alterations due to renal failure areas follows. Assess the patient for signs of congestive heart failure, pericarditis, hypertension, and cardiac arrhythmias. Take the patients blood pressure lying down and sitting up, especially if the patient is on antihypertensive medications. When taking the pulse note quality, regularity, and pulse deficits. Look for peripheal edema and jugular distension, both signs of congestive heart failure. Due to increased platlet
adhesiveness, the patient needs to be assessed for peripheral pulses and Homan's sign. Monitor and assess blood gases and electrolytes as they affect myocardial performance.

**Hematological Alterations**

Hematological alterations will occur in renal failure. Due to the decreased secretion of erythropoietin, a normochromic and normocytic anemia occurs. Red blood cell production, breakdown and survival time are decreased in renal failure. Bone marrow functioning is depressed. The mass of the red blood cells decreases. It is theorized that this decrease in mass functions as an adaptation, since an increase in solids within the vascular system increases the plasma oncotic pressure against the glomerular filtration. Bruises occur easily due to the decreased platelet adhesiveness, increased bleeding time, and increased capillary permeability. Bleeding may occur from any orifice.

White blood cells stay within normal limits, but the lymphocytes are less responsive than normally to infections. For reasons that are unknown, the lymphoreticular system is depressed but the immunoglobulin levels stay normal. Cell mediated responses are altered, but the formation of antibodies to red and white cell antigens is not inhibited.

The nurse should assess the patient's hemoglobin, hematocrit, bleeding and clotting times for increases. Hematest stools and vomit if there is any suspicion of it containing blood. Provide a safe environment since the patient bruises easily due to increased bleeding time. Due to increased chance
of infection, the nursing staff should take the initiative in obtaining cultures of all infected or potentially infected sites. Good oral hygiene and good skin hygiene are necessary. Bladder catheters should never be left in unless there is justification. Early recognition of infection with prompt and adequate antibiotic therapy will aid in eradication of the majority of infections. The most common sites of infection will be those of the skin, mouth, salivary glands, urinary tract and lungs.

**Respiratory Alterations**

Respiratory alterations will also occur. Due to advanced fluid overload, the patient in renal failure is prone to develop pleural effusion and pulmonary edema. These problems are the result of pulmonary congestion which is severe enough that the distended capillaries leak fluid into the interstitial and alveolar spaces of the lungs. Another complication of renal failure is pneumonia. Before dialysis and renal transplants, pneumonia was frequently the terminal event in uremia.

The nurse must monitor arterial blood gases and blood electrolytes to detect respiratory complications early. Listening to breath sounds, having the patient turn, cough, and deep breath will aid in the prevention of serious respiratory complications.

**Bone Alterations**

Renal osteodystrophy is when the bone demineralizes due
to renal failure. The exact mechanism by which this happens is not fully understood. Impaired calcium absorption in uremia is one factor that contributes to this disease. Metabolic acidosis is also a factor since calcium tends to dissolve out of bone in an acid medium. The main symptoms are bone pain, mainly in the spine and the long bones. This appears in children as rickets and in adults as osteomalacia. Secondary hyperparathyroidism is present in both age groups. Osteosclerosis and pathological fractures will become apparent. Patients will respond to oral vitamin D supplements. Monitoring serum calcium levels is very important to early detection. The nurse should protect the patient from fractures which will occur easily.65

Gastrointestinal Alterations

Due to potassium imbalances, there is an weakness of smooth muscles in the gastrointestinal tract. Decreased intestinal motility, abdominal distention and paralytic ileus may happen when the smooth muscle is not functioning properly. Hemorrhage, due to hemostatic defects, pre-existing ulcer disease, and erosions of the mucosal lining at all levels of the gastrointestinal tract, occurs in about 15% of patients experiencing renal failure. A serious complication of massive bleeding may occur. Potassium levels become markedly increased due to absorption of blood by the gastrointestinal tract.66

Gingivitis and moniliasis will occur due to alterations in the normal mouth flora. The urease of normal mouth flora hydrolyzes salivary urea, forming ammonia which irritates.
Also, hiccups, caused by increased nitrogenous waste products irritating the phrenic nerve, may be prolonged and highly irritating.

Esophagitis with reflux heartburn and dysphagia are common. Anorexia and vomiting lead to weight loss and compound fluid and electrolyte balance problems.

The nurse assesses for signs of gastrointestinal bleeding, such as bloody vomit or stools. Good mouth care is important. Also, electrolytes must be monitored for possible potassium imbalances.

**Dermatologic Alterations**

Dermatologic alterations consist of the skin being very dry, pale, and will have a number of bruises. The color is yellow tan from retained urochrome pigments. Pruritus and tickling or crawling sensations cause a great deal of discomfort. Relief can be attained by applying lotion to the skin. Safety measures to protect skin integrity should be taken.

**Neurologic Alterations**

The central, peripheral, and autonomic nervous systems all are affected in renal failure. Alterations in the central nervous system includes metabolic encephalopathy which causes behavioral changes such as loss of memory, inability to concentrate, and personality changes.

Peripheral neuropathy manifests itself in gait changes, burning feet, restless legs and foot drop. These changes are asymmetrical and begin distally; they may move up to cause
paraplegia or to involve the upper extremities. Cranial
nerve changes may occur causing changes in vision and smell.

The autonomic nervous system changes appear as de­
creased responsiveness of the baroreceptors. Therefore, the
heart loses the ability to compensate for changes in stroke
volume. 68

The nursing implications for neurologic alterations are
to check the patient's sensorium, look for gate abnormalities,
and to assess for twitching or tremors. The onset of poly­
neuritis in a patient with renal failure is a sign to start
dialysis as soon as possible.

MEDICAL TREATMENTS AND NURSING INTERVENTIONS

The main objective for medical and nursing care in renal
failure is to identify the cause of the damage and remove or
treat it. For example, if the patient is hypovolemic, the
main objective of treatment is to restore circulating blood
volume. When the renal failure is due to nephrotoxic drugs,
the dosage is altered or discontinued.

Fluid and Diet

In renal failure the fluid intake is limited to prevent
fluid overload. Twenty millimeters per hour plus the total of
the previous hours urine output is usually what is given.
Daily weights are also taken to assess fluid retention.

Maintaining an anabolic state is vital to the patient's
condition. By preventing infection is one way to assist in
this. Another way is to restrict protein intake. The mere finding of a raised blood urea is not an indication for diet, however when the patient develops symptoms it will be necessary. When symptoms such as nausea and vomiting begin to develop, with a blood urea of 150 miligrams a 40 gram protein diet should be tried. The consequent reduction in blood urea may bring symptomatic relief. As renal failure advances the protein may have to be cut to 30 grams per day. In very severe renal damage it is sometimes possible to keep the patient ambulant on only 18 to 20 grams of dietary protein.

The Giovannetti diet is usually followed. It contains almost equal amounts of all essential amino acids in the form of the first class animal proteins with the exception of methionine which is given as a supplement in tablet form. The patient is unable to eat ordinary bread because of the protein content of the flour. The diet has a high calorie content in the form of carbohydrates and fat which also aids in maintenance of an anabolic state. When this diet is followed, the blood urea can be kept at an acceptable level and there is evidence that urea is actually utilized in the synthesis of proteins.69

Dialysis

When the kidneys have failed it will be necessary to use either peritoneal or hemodialysis. Each may be used to relieve the symptoms produced by renal failure temporarily until the patient regains his function or to sustain life in the person with irreversible kidney disease. When the damage
is permanent dialysis must be continued intermittently for the rest of the patient's life. Dialysis is also used to overcome uremia and physically prepare the patient to receive a kidney transplant. Frequently dialysis is necessary to keep the patient alive until a suitable donor kidney is found; even then, dialysis is used when the kidney does not function adequately immediately.

Dialysis consists of providing a carefully composed physiological solution in contact with a large area of a semi-permeable membrane on the other side of which the patient's blood is allowed to flow. The semi-permeable membrane must have the property of allowing the passage of water and small molecules such as urea and potassium, but its pores must be sufficiently small to prevent the escape of large molecules such as protein. In peritoneal dialysis the membrane is the peritoneal membrane and in hemodialysis it is an artificial membrane. Substances such as urea and creatinine will diffuse across the membrane from the area of high concentration (the blood) to the area of low concentration (the dialysis fluid). The dialysis fluid is constantly exchanged and replaced with fresh fluid so that the urea is carried away.70

There are four basic goals to dialysis therapy. The first is the removal of the end products of protein metabolism from the blood. Second is the maintenance of a safe concentration of the serum electrolytes. Thirdly, dialysis corrects acidosis and replenishes the blood's bicarbonate buffer system. Then the last goal is the removal of excess
fluid from the blood. Peritoneal dialysis is used frequently with patients in acute renal failure. It requires very little equipment and can be performed in most hospital settings. Peritoneal dialysis is also used once in a while as a lifesustaining treatment for chronic renal failure. Patients may be trained to perform this treatment in their own homes.

In peritoneal dialysis, the physician places a catheter through the abdomen into the peritoneal space. Two liters of dialysis fluid suspended in bottles on a drip stand is run by gravity via a coil through a warming bath at body temperature, into the peritoneal cavity through the catheter. The dialysate is a sterile commercially prepared solution which is isotonic or hypertonic. The isotonic solution is used when there is no necessity to remove large amounts of water from the patient because his hydration state is near normal. The hypertonic fluid is used when the patient is overloaded with fluid. The hypertonic fluid because of its high dextrose content and high osmotic pressure is able to draw water from the patient's circulation.

After the solution remains in the abdomen for about twenty minutes in contact with the semipermeable peritoneal membrane. The fluid is then allowed to run into a collecting bag at the side of the patient's bed. When the dialysate solution flows out it has picked up urea and creatinine. The whole cycle of flowing into the abdomen and draining out takes about one hour. An average dialysis might consist of hourly
cycles for about forty-eight hours. Uremia is never com-
pletely corrected by the dialysis.74

Peritoneal dialysis is entirely a nursing procedure, except for the insertion and removal of the peritoneal catheter. Infection is the most common complication of peritoneal dialysis. Meticulous sterile procedure is used during the insertion of the special peritoneal catheter and induction of fluid into the peritoneal cavity. As the result of contamination during this procedure, diffuse peritonitis can occur.75

The nurse must remember that is a warming bath is not available the dialysate solution is warmed to body temperature in a warm water bath before it is introduced into the peritoneal cavity. The patient will feel great discomfort and may chill if warming is not done.76

The patient should never experience pain during the procedure. If he does, the solution is either too hot or too cold or the catheter is not in the proper place.

The nurse must keep a meticulous record of the amount of solution introduced and the amount returned. If the record shows three liters or more retained, the physician should be notified. Fluid overload could occur rapidly. The nurse must take the patient's vital signs often during the procedure. She can expect a lowering of the blood pressure as the patient's blood volume is reduced. If excess salt and water are removed he may become severely hypotensive. The nurse should be alert to developing symptoms of depleted volume so
that replacement of fluid can begin before there is a significant depletion. Allow the patient to eat and drink within the restrictions imposed by his other problems, during peritoneal dialysis. 77

Although peritoneal dialysis is an excellent treatment method, it has many limitations. These limitations cause the artificial kidney to be the approved method in many cases requiring long term dialysis. During hemodialysis blood is taken from the patient's artery, passed between layers of cellophane, which are sterile on the blood side, and returned to the circulation. The outside of the membrane is bathed by a fast moving stream of dialysis fluid. 78 The dialysate solution is carefully compounded to contain physiological concentrations of sodium, chloride, calcium and magnesium. The end products of protein metabolism diffuse from the blood into the dialysis fluid. The toxic solutes do not accumulate in the dialysis fluid. The dialysis fluid is discarded continuously after one passage through the dialyser or is recirculated and changed at different intervals through a larger tank. 79

To prevent blood from clotting in the tubes and cellophane a continuous infusion or or intermittent injections of heparin are given.

In order to have access to the patients circulation, cannulas are inserted into the blood vessels of an extremity. The exact placement of the cannula will depend on which arteries and veins are available for use. The cannula is made of
Teflon tips and silastic tubing. The Teflon tips are inserted in a superficial artery and vein. These are attached to the silastic tubing which is brought out through the patient's skin. When dialysis is not being performed, the two cannula ends are connected by the use of a Teflon joint. This creates an external shunt between the patient's artery and vein. For hemodialysis the joint is opened and each is connected to special fittings on the artificial kidney. When dialysis is stopped, the joint is reconnected for resumption of flow through the shunt. The indwelling cannulas have eliminated the use of repeated cutdowns and permits ready access to the patient's bloodstream when hemodialysis is indicated. The nurse must be aware the daily cleansing of the cannula is necessary. The nurse checks to see whether fibrin or clots have developed.80

Creation of an internal arteriovenous fistula is also a surgical procedure, which allows easier access to the patient's bloodstream. An artery in the arm is anastomosed to a vein in a sideways fashion. This creates an opening or fistula between a large artery and a large vein. The leaking of arterial blood into the venous system results in the veins becoming engorged. A needle is placed in one vein and another needle is placed in a different vein or in an opposite direction before dialysis is to begin. Blood is pumped on the tubing leading to the hemodialyzer, arterial blood is pulled out of the vein by way of the fistula. Blood will return to the patient by a tube connected to the other needle.81
Nursing care of the patient undergoing hemodialysis is very complex. No nurse should care for patients undergoing hemodialysis unless she has received special instructions. To prevent coagulation of the blood, the nurse will give heparin throughout the procedure. Bacteremia may result from contamination of the circuit. The entire circuit through which the blood flows must be sterile. Therefore the nurse must take precautions using aseptic technique. The entire blood circuit must also be a completely closed system. Any break in the tubing or a small hole in the membrane compartment will allow the blood to escape. The nurse must be alert to this because if undetected the patient can exsanguinate.

During dialysis, the dialysate concentration must be maintained within a certain safe limit. If these concentrations become too high or too low, the results may be fatal to the patient. Since these problems could be fatal, monitoring devices with audible alarms to signal pressure changes, blood leaks, and dialysate concentration changes are an essential part of safe hemodialysis equipment.

During dialysis the nurse must be aware that the patient's weight must be monitored. Also, during dialysis the patient's blood pressure needs to be taken frequently.82

Patients may need to be on dialysis for the rest of their lives. The nurse needs to be aware that once the patient realizes permanence of the treatment in his life, a period of depression is expected. The patient who handles stress poorly or who has little support from others, will require psycho-
logical support from the nurse.

Home dialysis is the ultimate rehabilitation for the patient who requires hemodialysis. Once the patient or his assistant is trained adequately, his feelings of competence and independence are greatly enhanced. Also, the flexibility in his schedule is beneficial. The cost of home dialysis runs about three thousand dollars per year as opposed to twenty-one thousand dollars per year for hospital based dialysis treatment.83

Transplants

Although dialysis has proved itself to be an acceptable means to continue an individual's life, unfortunately the degree of rehabilitation which is achieved is variable and sometimes quite poor. Seldom are strength, vitality and stamina returned to normal. Dependence on the machine is expensive and a continuing source of frustration and anxiety. For these patients a successful kidney transplant offers a chance for an almost normal life.

The ideal recipient for a kidney should be one who is free of generalized irreversible systemic diseases. Those patients with brain damage, severe liver disease, advanced vascular or collagen disease, cancer, or any other disease in which the prognosis is fatal. Transplantation should only be attempted when the patient's general condition is good.84

Before the operation the patient must be well dialysed. The patient's metabolic state should be brought as close to
normal as possible. Dialysis may be by peritoneal or hemo-
dialysis methods. He should be well nourished. He should be
active and rehabilitated. He should also be free of any
infection, especially in the urinary tract. Cystoscopy should
be carried out to ensure that the patient has an anatomically
normal lower urinary tract. If the bladder is grossly ab-
normal then transplantation may still be possible by fashion-
ing an ileal conduit or loop. Then the ureter of a trans-
planted kidney can be placed at a later surgery.

The patient's blood pressure should be well controlled.
In patients who are on regular oral anticoagulant therapy for
shunt clotting, this medication must be stopped before the
operation. Appropriate doses of vitamin K may need to be
given.

Information regarding potential family donors is obtained
during the initial evaluation of the recipient. Transplants
between siblings and from parent to child are the most com-
monly performed. The most consistently successful transplants
have been between siblings where there is an identical match
demonstrated by tissue typing techniques. Living donors must
be evaluated by the nurse to ensure that the donor is a true
volunteer and is not under pressure from other members of the
family. If there is any doubt about willingness the operation
should not be done. 85

All willing family members are given histocompatibility
testing and those proved immunologically suitable are
scheduled for medical evaluation. A complete medical
evaluation with history, physical, and investigation designed to demonstrate that he has an entirely normal urinary tract.

If a living related donor is not available, a cadaver donor may be used. Unfortunately, even with optimal tissue typing, results of cadaver donor transplantation is not as successful. The usual source of cadaver kidneys are accident victims, or persons who have died from massive cerebral trauma or hemorrhage or following cardiovascular surgery. When death appears imminent, the physician approaches the family for consent to remove the patients kidneys at death. The donor is evaluated as completely as possible without risking further deterioration of his condition.

In some situations where ventilation is continuing life, the donor's physician determines when irreversible damage has occurred in the brain. The patient can be said to have brain or neurological death. It has been proposed that having ascertained repeatedly that there is no spontaneous cerebral activity and no spontaneous respiration it is justified to switch off the ventilator in the operating room and remove the kidneys while circulation is still maintained. In this case the doctor must be completely sure of the diagnosis and preferably have signed a death certificate before the operation. Kidneys should be removed within one hour after effective circulation in the donor has ceased. After removal, the kidneys are perfused and cooled to prevent ischemia. The kidney is then packed in iced saline and transported to the nearest organ perfusion machine. Centers where transplants
are performed work together to find the best possible match for the cadaver kidney. Kidneys may be transported by car or plane to the recipient.

The nurse, as the professional person in closest contact with the recipient and his family, is responsible for reinforcing information and determining the patient's level of understanding and acceptance. An understanding and awareness of the burdens that this situation places upon the family psychologically will help the staff in developing tolerance to hostilities, lack of cooperation, unreasonable demands, and emotional outburst of the family to the therapy. Prior to surgery, the nurse may arrange for the recipient and living donors to meet and talk with patients who have successfully recovered from their surgery.

The operative procedure consists of removal of the recipient's own kidneys, if they have not been removed previously. The donor kidney is placed in the iliac fossa and the renal vessels are anastomosed to the recipient's iliac vessels. This surgical procedure is done swiftly to decrease the time the donor kidney is without a blood supply. Periods of ischemia longer than thirty minutes can damage the function of the newly transplanted kidney.

The kidney usually begins functioning immediately. There is usually a period of diuresis for the first few days. Sometimes adequate functioning is delayed a few days. Dialysis may be performed intermittently until adequate function is established.
The main problem with transplantation is the homograft rejection reaction. Genetically dissimilar tissue in the body of the patient causes a reaction to set in that will ultimately destroy the foreign tissue. The only exception to this is in the case of identical twins. Immunosuppressive therapy is necessary to prevent and to control the rejection reaction. This therapy is designed to block the patient's normal immune responses. Azathioprine (Imuran), Adrenal corticosteroids (usually prednisone), and Antilymphocyte globulin are the three medications most commonly used to suppress the rejection reaction. Serious side effects accompany the use of these drugs. The nurse observes for the development of any side effects, reports them to the doctor, and aids in their treatment. She helps the patient obtain relief of side effects and reassures him that cosmetic changes are only temporary in nature.88

The patient must be forever watchful for signs of rejection. Acute rejection can occur within the first week to two years afterwards. These episodes of acute rejection are handled by temporarily increasing the drug therapy. Chronic rejection usually starts after six months and is insidious, leading to destruction of the kidney. Symptoms of both types of rejection are as follows: increased blood urea nitrogen levels and serum creatinine levels, decrease in creatinine clearance, decrease in urine volume, elevated temperature and blood pressure, weight gain, restlessness, irritability, and a tender, palpable kidney.89
The nurse must stress to patients that they will continue to need close medical supervision for the rest of their life. These patients must be prepared emotionally with a realistic attitude. As in all situations when continued medical supervision is necessary, independence needs to be encouraged.

CONCLUSION

As a basis for providing care to patients with renal failure, there is a large body of knowledge and technical expertise that the nurse must master. As this field of knowledge grows, so will nephrology nursing grow along with it in order to strengthen the quality of services provided for these patients. The challenge to the nursing profession in assessing and planning interventions is enormous. It is up to the nurse to obtain the knowledge that she will use to assist the patient to achieve his highest level of rehabilitation.
FOOTNOTES


4 Crouch, Human Anatomy and Physiology, p. 668.

5 Ibid., p. 670.

6 Ibid.

7 Ibid., p. 671.

8 Frances, Introduction to Human Anatomy, p. 408.

9 Ibid., p. 409.


12 Hudak, Critical Care Nursing, p. 214.

13 Schottelius, Textbook of Physiology, p. 516.

14 Ibid., p. 517.

15 Crouch, Human Anatomy and Physiology, p. 681.


17 Crouch, Human Anatomy and Physiology, p. 680.

18 Schottelius, Textbook of Physiology, p. 517.


21 Ibid., p. 324.

24 Ibid., p. 87.
26 Uldall, Renal Nursing, p. 64.
28 Uldall, Renal Nursing, p. 65.
30 Ibid., p. 56.
32 Luckmann, Medical-Surgical Nursing, p. 725.
33 Brener, Patient Care, p. 58.
34 Luckmann, Medical-Surgical Nursing, p. 729.
35 Uldall, Renal Nursing, p. 83.
36 Ibid., p. 82.
37 Brener, Patient Care, p. 63.
38 Luckmann, Medical-Surgical Nursing, p. 726.
39 Ibid., p. 727.
40 Uldall, Renal Nursing, p. 56.
41 Luckmann, Medical-Surgical Nursing, p. 727.
42 Meltzer, Concepts, p. 308.
44 Gallo, Critical Care, p. 326.
45 Brundage, Nursing Management, p. 43.


48. Luckmann, Medical-Surgical Nursing, p. 728.


50. Ibid., p. 319.

51. Gallo, Critical Care, p. 326.

52. Brundage, Nursing Management, p. 44.

53. Ibid., p. 44.

54. Ibid., p. 44.

55. Ibid.

56. Ibid.

57. Ibid.

58. Giblin, Nursing Care, p. 672.


60. Ibid., p. 47.

61. Ibid., p. 47.


63. Ibid.

64. Brundage, Nursing Management, p. 48.

65. Uldall, Renal Nursing, p. 106.


68. Ibid., p. 47.

69. Uldall, Renal Nursing, p. 103.

Luckmann, Medical-Surgical Nursing, p. 733.


Uldall, Renal Nursing, p. 142.

Giblin, Nursing Care, p. 684.

Ibid., p. 684.

Brener, Patient Care, p. 98.

Luckmann, Medical-Surgical Nursing, p. 734.

Ibid., p. 734.

Ibid.

Gallo, Critical Care, p. 355.

Luckmann, Medical-Surgical Nursing, p. 734.

Ibid., p. 735.

Ibid.


Ibid., p. 507.

Uldall, Renal Nursing, p. 231.

Luckmann, Medical-Surgical Nursing, p. 735.


Ibid.
BIBLIOGRAPHY


Beck, Edna; McCluskey, Audrey; Sawyer, Janet; Shaffer, Kathleen; and Phipps, Wilma. Medical-Surgical Nursing. Saint Louis: C.V. Mosby Comp., 1975.


