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Renal Failure and Critical Care Nursing

I. Introduction and Assessment

The kidneys, which are generally smaller than a person’s hand, are required to filter approximately 1700 liters of blood every day and remove the waste products of this blood into about one liter of urine every day. This affects the composition of our blood tremendously. The kidneys filtered as top particles from the blood and selectively reabsorbed those that are needed to maintain life. By regulating the volume and composition of body fluids, the kidneys perform both a waste removal as well as a metabolic or endocrine.

A. The History, Presence, and Nature of Renal Failure

Some other things which my predispose patients to the development of renal failure are; chronic renal disease, previous radiation therapy, and urinary tract infections. Circulatory disease which is generally associated with the poor renal perfusion may also be a factor in the development of renal failure. Some of these problems may include; aorta aneurysm, peripheral vascular disease, cardiac disease, and of course hypertension. Diabetes is another major factor in the United States today. The use of nephrotoxic agents such as antibiotics, and chemotherapy may also predispose one to the development of renal failure. By obtaining a good patient database the nurse can ascertain the risk of patients developing chronic or acute renal failure.

B. Signs and Symptoms

The general appearance of the patient in acute renal failure may be; anemia, pallor, itching, dryness of the skin, dry mucous membranes, costoalvertebral angle angled tenderness, and lumbosacral, periorbital
or extremity edema. the kidneys may affect the heart are cardiovascular system by causing pericarditis, hypertension, or retinopathy.

Pulmonary wise the patient in real failure may develop respiratory distress due to acidoses. The breath sounds may be diminished and there may be a uremic odor to the breath.

Most of the time, there is a change in the urinary patterns of the patient. There may be a change in frequency, color, quality or odor of the urine. However, urinary output is not always an indicator of acute renal failure. A patient may be making more than adequate urine, but his system may not be removing the waste products from his body.

The patient may complain of ammonia taste. He may have slight gastrointestinal disturbance such as; hiccups, anorexia, nausea, vomiting, coated tongue, or he may have none of the symptoms at all.

As the patient becomes more and more uremic he may develop central nervous system disturbances. These may include headache, confusion, disorientation, drowsiness, insomnia, muscle twitch and, or weakness.

The patient's vital signs may be affected as he becomes more uremic. He may develop orthostatic changes in his blood pressure and pulse. The skin may become cooler. His urinary elimination pattern, volume, frequency, specific gravity, and state of hydration may change. His weight may increase.

All of these are things the nurse muthest assess.

II. Anatomy – Structure and Function

The kidneys are two main shape structures which lie in the retroperitoneal space between the 12 thoracic and third lumbar vertebra. The left kidney sets slightly behind the spleen, while the right kidney sets behind the liver and is slightly lower than the left. Each kidney is enclosed in a tough fibrous capsule and is supported and protected by fat tissue. There is a fissure in the central content portion of the kidney where the blood vessels enter and leave. This is called the hilus. Also coming from the hilus, are to the ureters which connect the kidney to the bladder. The cortex is a brownish–tissue which covers the outer third of the kidney. The medulla are light–colored and cone–shaped, these are the renal pyramids. The papilla are formed by the free ends of the pyramids which opens into the renal pelvis. the renal pelvis is made up of calyces, which drained up or
lower hands of the kidney. Days unite with a renal pelvis at the upper end of the ureter. The functional unit of the kidney are the nephrons, each kidney contains approximately 1.2 million of these. Within each nephron there is a glomerulus and a tubule.

Within the glomerulus, there is a structure called Bowman's Capsule which contains a network of capillaries. Fluid in particles from the blood and are filtered through this membrane. Water, nutrients and electrolytes, as well as other substances, are reabsorbed as they pass through these tubulars. There is collecting duct which collects fluid from several nephrons and passes this fluid into the renal pelvis.

Two capillary beds, a glomerulus, and a peritubular network supply the nephron. The glomerulus is a unique, high pressure capillary filtration system that is located between two arterioles, the afferent and efferent arterioles. The low-pressure reabsorptive system of the peritubular capillary network arises from the efferent arteriole.

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III. Physiology

A. Excretory functions

1. Filtration – the process of removing fluids and small particles from the blood.

   a. the glomerulus, which lies between two arterioles, allows for high-pressure filtration system. Capillary filtration pressure in the glomerulus is 2–3 times as high as that of other capillary beds in the body. The filtration pressure and the glomerular filtration rate (GFR) are regulated by the constriction or relaxation of the afferent and efferent arterioles. During strong sympathetic stimulation, which causes marked constriction of the afferent arteriole, the filtration pressure is reduced to the point where GFR drops to almost 0.

   b. Capillary membrane of the glomerulus is composed of 3 layers:

      1. endothelial layer of the
capillary
2. basement membrane
3. a layer of epithelial cells
   that line Bowman's capsule

c. Glomerular capillary permeability is 100–1000 times as great as capillaries elsewhere in the body. All 3 layers allow water and dissolved particles, such as electrolytes, to leave the blood and pass rapidly into Bowman's capsule. Blood cells and plasma proteins are too large to pass through the glomerular membrane of a healthy kidney.

d. Glomerular filtration rate (GFR) is normally about 125 ml per minute. GFR can provide a measure to assess renal function, and can be measured clinically by collecting timed samples of blood and urine.

2. Creatinine – product of creatine metabolism by the muscle. Is filtered by the kidney, but not absorbed in the renal tubule.

Formula for creatinine clearance: \[ C = UV P \]

\[ C = \text{clearance rate} \]
\[ U = \text{urine concentration} \]
\[ V = \text{urine volume} \]
\[ P = \text{plasma concentration} \]

Normal creatinine clearance is 115–125 ml/min (corrected for body surface area) Usually 24 hour collection with blood drawn when urine collection is completed.

3. Tubular reabsorption and secretion

The filtrate from the glomerulus passes through:

1. proximal tubule
2. loop of Henle
3. distal tubule
4. collecting duct

Then it reaches the pelvis and kidney
Reabsorption: water, sodium, and other substances leave the lumen of the tubule and enter the blood.

Secretion: substances from the blood enter lumen of the tubule.

Glucose and amino acids – completely reabsorbed

Filtered water – 99% reabsorbed
Urea – about 50% reabsorbed
Creatinine – none
Electrolytes – determined by need

3. Urine concentrating ability of the kidney: 2 mechanisms (SLIDE)

1. Increased solute concentration in the medullary area surrounding the collecting tubules. Loop on Henle and peritubular capillary (vasa recta) descending into the renal medulla. Here a countercurrent mechanism controls water and solute flow. As a result, water is kept out of the peritubular area surrounding the tubules, and sodium and urea are retained.

2. Selective permeability of collecting tubules (controlled by ADH) During dehydration, the kidney plays a major role in maintaining water balance. Osmoreceptors in the hypothalamus sense the increase in extracellular osmolality and stimulate release of ADH from posterior pituitary. Collecting tubules (under influence of ADH) become permeable to water. In the absence of ADH, the renal tubules remain impermeable to water and a dilute urine is formed. Specific gravity (osmolality) of urine varies with its concentration of solutes. Specific gravity provides index of hydration status and functional ability of the kidneys. Concentrated urine: 1.030 – 1.040 (SLIDE) Marked hydration or dilute: 1.000

4. Sodium and potassium regulation
Sodium and potassium regulation (SLIDE) glomerular filtrate reabsorbed in proximal tubule. Na and KC1 pumped (requires energy) into intercellular spaces, and absorbed into peritubular capillaries. Water movement accompanies the movement of these particles. Na reabsorption in distal tubule is variable and dependent on aldosterone. In the presence of aldosterone, almost all of sodium is reabsorbed and urine becomes almost sodium free.

5. Potassium regulation

Potassium regulation – aldosterone mediated secretion of K into tubular fluid. (Can be reabsorbed in distal and collecting tubules, but since dietary intake far exceeds need, secretion usually exceeds reabsorption.)

6. Endocrine fuctions

1. **Renin** – released by special cells located near the glomerulus (juxtaglomerular cells) in response to:

   Reduction in GFR

   Sympathetic stimulation – Combines with angiotensinogen, a plasma protein that circulates in the blood to form angiotensin I, then converted to angiotensin II (potent vasoconstrictor and stimulator of aldosterone release).

2. **Erythropoietin** – released in response to hypoxia. Acts on bone marrow to stimulate production and release of RBCs. Persons with chronic hypoxia often have increased RBCs (polycythemia) due to increased erythropoietin levels. Examples: congestive heart failure, chronic lung disease, living at high altitude.

3. **Vitamin D** – activated and converted in
V. Pathophysiologies

80,000 – 110,000 die per year due to renal problems. 1.2 million conditions requiring hospitalization are related to renal stones, UTIs and other conditions.

Medicare covers 95% of dialysis and transplants.

A. Congenital defects

10% of persons born have potentially significant malformations of the urinary system.

1. Unilateral agenesis – relatively common; people often unaware.
2. Agenesis – Incompatible with life; infants usually stillborn.
3. Hypoplasia – Kidneys are not normal size; often only affects 1 kidney. If both, it progresses to renal failure, dialysis, and/or death.
5. Dysplasia – Most common; i.e.: multicystic kidney, atresia or obstruction of the ureter.
6. Polycystic disease – Inherited; requires interventions such as surgery, drug therapy, transplant, and dialysis.

B. Urinary Tract Infections (UTIs)

Second most common type of infection (Respiratory is first). 20% of all women will have one in their lifetime.

1. Bacteriuria. Presence of 100,000 or more organisms per ml of urine. Common complication associated with the use of foley catheters. CDC recommends that patients with a foley catheter not share a room.
2. Cystitis. Infection of the bladder. Characterized by frequency, urgency, lower abdominal discomfort, and dysuria.
3. Pyelonephritis. Inflamed areas of the kidney and renal pelvis. Can develop scar tissue. Patient is usually very ill: symptoms include pain and chills, decrease in renal function.
Chronic Pyelonephritis. Characterized by scarring and deformation. May lead to loss of tubular function. May have severe hypertension which contributes to a significant cause of renal failure.

a. Treatments
   a. Sulfonamides
   2. Fluids

C. Obstructive Disorders

2. Urolithiasis (stones). One-third of people with recurrent stones will lose a kidney.
   a. Types
   b. Staghorn – fills renal pelvis
   c. Calcium (oxalate or Phosphate) make up 80–90% of stones
   d. Magnesium ammonium phosphate bacteria causes splitting of urea then stone forms.
   e. Uric acid – found in people with gout
   f. Cystine – rare and genetic in cause.

D. Glomerulonephritis

Most common following infections by strains of group A, beta-hemolytic streptococci. In this situation, there is an abnormal immune reaction, causing immune complexes to become entrapped in the glomerular membrane, inciting an inflammatory response. The capillary membrane swells and is then permeable to plasma proteins and blood cells. Usually follows a strep infection by 10 days to 2 weeks (the time needed for formation of antibodies). Oliguria is an early symptom, Na and H2O retention causes edema, particularly of the face and hands, along with hypertension. Proteinuria and hematuria follow from the increased capillary permeability. This may give a smoky hue to the urine ("cola" colored).
E. Diabetic glomerulosclerosis


F. Acute Renal Failure (ARF)

Is a potentially reversible condition that results in acute suppression of renal function. Acute renal failure may rapidly present a life threatening situation which is amenable to appropriate medical management provided that the situation is recognized. Evidence of renal involvement may be masked by the primary medical, surgical, or obstetric condition.

1. Pathogenesis

The pathogenesis remains controversial and inconclusive but the theories most often encountered include backleak, tubular, obstruction, vascular obstruction, and reninangiotensin. Regardless of the pathogenesis, pathologic studies have described two characteristic histologic insults:

**Iscemia**–This tends to produce pathcy lesions affecting the proximal and distal tubular segments. The basement cell membrane is disrupted and tubular necrosis present

**Nephrotic**–This usually leaves the basement membrane intact while tubular destruction can range from simple cellular swelling to frank necrosis. Damage is seen primarily in the proximal tubular segments. The influences that may bear on kidney function are usually considered in terms of three separate but frequently interrelated categories.

2. Categories of ARF

   a. Prerenal
Prerenal causes of renal failure act by reducing glomerular perfusion either by vasoconstriction, or a reduction of mean arterial pressure. This may be due to local or general causes:

i. Local: - embolism or thrombosis, - surgical operation, - hepatorenal syndrome

ii. General: - hypovolemia due to hemorrhage, burns, cardiac insufficiency, - GI losses, - peripheral vasodilators associated with excessive anihypertensivetherapy, - bacteremic shock

iii. Many of these conditions can occur in the presence of a normal blood pressure and go undetected until renal symptoms present.

iv. Frequent consideration of the clinical situation will help assist in identifying prerenal causes for ARF.

b. Renal (or intrarenal)

Renal causes of ARF are due to parenchymal changes resulting from disease or nephrotoxic agents that induce renal disease.

Diseases of the renal parenchyma other than ischemia account for 25% of all cases of ARF. These include glomerular lesions such as acute poststreptococcal glomerulonephritis, SLE, papillary necrosis, and vasculitides (polyarteritis nodosa), Goodpasture's, Wegener's, and malignant hypertension.
Ischemia – occurs when perfusion to the kidney is obliterated or reduced below a mean systemic blood pressure of 60–70 mmHg in the afferent arteriole.

Can be grouped into 5 categories:

i. Injury to the glomerular membrane (acute glomerulonephritis)

ii. Acute tubular necrosis (ATN) characterized by destructive changes in the tubular epithelium due to ischemia or exposure to nephrotoxic agents. Shock and heart failure, for example, cause prerenal failure, tend to cause renal ischemia, and if allowed to progress, can produce tubular necrosis. As a rule, the blood supply to a normal kidney can be interrupted for about 30 minutes without inflicting damage to the kidney. Trauma, sepsis, and heart failure may interrupt blood flow for a longer duration. Nephrotoxic drugs may be ingested, inhaled therapeutically, accidentally, or with suicidal intent. Widespread use of nephrotoxic antibiotics has contributed to a high frequency of ATN. The sulfonamides, methicillin and cephalosporins, along with aminoglycosides such as gentamicin, tobramycin, vancomycin, and amikacin are some of the most common nephrotoxic antibiotics. The
aminoglycosides bind avidly to proximal tubular epithelial cells with a half life of 109 hours (5 1/2 days). Radiologic contrast media can also produce tubular damage. Radiographic dye usually promotes an osmotic diuresis and urine losses of up to 7 ml for every 1 ml of dye used.

iii. Intratubular obstructions – due to accumulation of casts and cellular debris, secondary to severe hemolytic reactions or myoglobinuria. Skeletal and cardiac muscle contains myoglobin, which accounts for their rubiginous color. Myoglobin corresponds to hemoglobin in function, serving as an oxygen reservoir within the muscle fibers. Myoglobin is not normally found in the serum or urine. It has a low molecular weight, so should it escape into the circulation, it is rapidly filtered in the glomerulus. Myoglobinuria is most commonly due to muscle trauma, but may result from extreme exertion, hyperthermia, sepsis, prolonged seizures, potassium or phosphate depletion, alcoholism or drug abuse. Can be traumatic or non-traumatic. Hemoglobin may also escape into the glomerular filtrate due to severe hemolytic reaction. Both myoglobin and hemoglobin cause discoloration of the urine, ranging from the
color of tea to red, brown, or black.

iv. Acute pyelonephritis or necrotizing papillitis – bacterial infection of the kidney and renal pelvis.

c. Postrenal

Postrenal causes of ARF are due to urinary tract obstruction, secondary either to structural or functional lesions of the urinary passages. The obstruction may occur anywhere from the tubules to the kidneys to the external urethral orifice. The cause may vary from stricture through stone to tumors of the urinary passages or of adjacent pelvic or abdominal organs.

Functional obstruction may follow the use of drugs which interfere with the autonomic supply to bladder or urinary passages such as ganglion blockage or antihistamines.

With diabetes mellitus the advent of an imbalance in the neurogenic supply may be sufficient to disturb the equilibrium and precipitate the obstruction.

Anuria associated with ARF is usually indicative of a post renal cause.

G. Chronic Renal Failure (CRF)

A slow, progressive renal disorder culminating in end stage renal disease (ESRD). The decline in kidney function correlated with the degree of nephron loss.

1. Pathophysiology – Systemic changes occur when overall renal function is less than 20–25% of normal.
2. **Pathogenesis** – Bricker's "intact nephron" hypothesis provides an explanation for the kidney's ability to compensate and preserve homeostasis despite a significant loss of 80% of nephron function.

3. During CRF, regardless of etiology, injury occurs to the nephrons in a progressive manner. Significant damage to groups of nephrons will eliminate them from their contributing role in maintaining renal function. The remaining intact nephrons will compensate by experiencing cellular hypertrophy. This growth process will enable them to accept larger blood volumes for clearances resulting in the exertion of greater solute levels, thus compensation results.

4. **Stages of CRF**
   a. Diminished renal reserve
   b. 50% nephron loss
   c. Kidney function is mildly reduced while the excretory and regulatory function are sufficiently maintained to preserve a normal internal environment.
   d. The patient is usually problem free.
   e. The patient's normal serum creatinine will double.

5. **Renal insufficiency**
   a. 75% nephron loss
   b. --Evidence of impaired renal capacity that appears in the form of mild azotemia, slightly impaired urinary concentrating ability, and anemia.
   c. --Factors that can exacerbate the disease at this stage by increasing nephron damage are: infection, dehydration, drugs and cardiac failure.

6. **End Stage Renal Disease (ESRD)**
   - 90% of the nephrons are damaged
   - Renal function has so deteriorated that chronic and persistent abnormalities exist in the internal environment.
   - Patient requires artificial support to sustain life, i.e. dialysis, transplant
   - Uremic Syndrome
--The body's systemic responses to the buildup of uremic waste products and the results of the failed organ system.

--Usually described as the constellation of signs and symptoms demonstrated by the RF patient.

--Symptoms may be avoided or diminished by initiation of early dialysis treatment.

**H. LUPUS NEPHRITIS**

Is the cause of approximately 3% of cases of ESRF (end-stage renal failure) requiring maintenance dialysis or transplantation. It is characterized by deposits of immune reactants in different sites along the nephron. There are many different forms of lupus nephritis each with its own characteristics. Treatment is based on the severity and progression of the disease. Many tests are used to follow the course of acute episodes and are used as a guideline to therapy. No one test is specific enough to be used individually.

Treatment includes: corticosteroids, cytotoxic drugs, plasma exchange therapy, Cyclosporine A, and pulse methylprednisone. Presenting symptoms include: proteinuria and hematuria.

**J. GOODPASTURE'S**

Is a syndrome consisting of pulmonary hemorrhages and glomerulonephritis of primary crescentic type. Frequently the term Goodpasture's is used in a purely clinical sense without reference to pathology or immunopathology.

The disease is most common in young adult males but occurs at any age.

The onset is sometimes preceded by "flu-like" symptoms.

**VI. Interventions**

**A. Pharmacotherapy**

1. Bumetanide (Bumex) – acts on the ascending limb of the loop of Henle to inhibit reabsorption of water and electrolytes.
Nursing Interventions – Monitor for signs of electrolyte imbalance. One or 2 daily doses appear to be more effective than small doses administered frequently. May need potassium supplements or potassium-sparing diuretics. Watch for hypokalemia. Concurrent use with aminoglycoside antibiotics may increase potential for ototoxicity. Avoid use of indomethacin or probenecid.

2. Ethacrynic acid (Edecrin) promotes the excretion of water, sodium, chloride and other electrolytes by inhibiting tubular reabsorption, especially in the medullary and cortical portions of the ascending limb of the loop of Henle.

Nursing Interventions – Administer with meals to reduce gastric upset. Avoid using with neurotoxic drugs such as amikacin, gentamicin, vancomycin. Watch for hypokalemia. Concurrent use with corticosteroids can produce severe hypokalemia.

3. Furosemide (Lasix) – promotes excretion of water, sodium, chloride and other electrolytes by inhibiting tubular reabsorption, especially in the medullary and cortical portions of the ascending loop of the loop of Henle.

Nursing Interventions – Watch potassium levels. Watch for hearing loss. Give with caution in patients receiving neurotoxic drugs (see Edecrin) When giving IV push – give 10 mg/min.

4. Hydrochlorothiazide (Esidrix, Hydro-diuril, Oretic) – promotes excretion of water, sodium and chloride by inhibiting the reabsorption of sodium ions in the ascending limb of the loop of Henle and in the early distal tubule of the nephron. Thiazide diuretic.

Nursing Interventions – Watch for potassium depletion, Electrolyte imbalance may increase when used with steroids, corticotropin, or Ampho B.
5. Mannitol (Osmitrol) elevates blood plasma osmolality resulting in flow of water from tissues, including brain and CSF. Mannitol is not reabsorbed in the renal tubule, which increases osmolality of the glomerular filtrate, facilitates the excretion of water and inhibits reabsorption of Na, Cl, and solutes.

**Nursing Interventions** – Use with in-line IV filter.

### B. Hemodialysis

1. most widely used treatment for renal failure.
2. Invented during WWII but not widely used until approximately 1960.
3. Hemodialysis replaces excretory functions of the kidney but not hormonal functions. Eliminates wastes, electrolytes and water by:

   a. Passive diffusion – solutes pass through a semipermeable membrane from an area of high concentration to low.

      i. Factors which influence clearance capacity

         - Pore size of the membrane
         - Concentration gradient of the dialysate,
         - Surface area of the dialyzer

   b. Convection (ultrafiltration) – water movement encouraged by establishment of a hydrostatic pressure force across a membrane (some solute passes also)

      - Factors which influence:
        - transmembrane hydrostatic pressure
        - surface area
        - selective permeability of the membrane

b. Indications

   i. volume overload
   ii. electrolyte imbalance
   iii. contraindications to peritoneal dialysis
   iv. uremic symptoms

- c. Contraindications/disadvantages
- i. Hemodynamic instability
- ii. vascular access problems
- iii. adherence to rigid diet
- iv. disequilibrium syndrome
- v. hepatitis
- vi. muscle cramping
- vii. bleeding tendencies due to anticoagulant used

- d. Types of vascular access:
  - i. Quinton or temporary double or single lumen catheters
  - ii. AV fistulas
  - iii. grafts (Gortex)
  - iv. shunts (Scribner)

D. Continuous Arteriovenous Hemodialysis (CAVHD) and Slow Continuous Ultra-filtration (SCUF)

Uses patient's arterial blood pressure to deliver blood to a low-resistance hemodialyzer primarily for water removal. Alternative for patients who are oliguric and require large quantities of parenteral fluids, such as hyperalimentation, antibiotics, or vasopressors. Also when other forms of dialysis are contraindicated.

- 1. Contraindications to SCUF or CAVHD
  - a. inability to tolerate anticoagulation
  - b. hematocrit greater than 45

2. Difference between SCUF and CAVHD:

  - a. smaller volumes with SCUF, so control of uremia and electrolytes is impossible.

3. Advantages

  - a. Better for cardiovascular stability since the process of volume removal is slower
  - b. Can be managed by critical care nurse rather than hemodialysis staff

4. Disadvantage:

  - a. limited ability to remove wastes and excess solutes
  - b. need arterial and venous access

VI. Labs and Tests

A. Urea – an end-product of protein metabolism. Urea rises with high-protein diet, excessive tissue breakdown, or in presence of GI bleeding (blood protein broken down in the intestine and urea absorbed into the blood). Kidneys
regulate BUN levels, filter urea in the glomeruli and reabsorb it in the tubules.

*BUN increases during dehydration

*Excretion is markedly decreased when GFR drops. (Longer the tubular fluid remains in the kidney, the greater the reabsorption of urea into the blood.)

**B. Creatinine** – product of creatine metabolism in muscle. is filtered in the glomeruli, but not reabsorbed in the tubules. Therefore, blood values depend closely on GFR.

*Normal creatinine level is proportional to muscle mass. ex: small woman – 0.5 mg/100 ml blood, man – 1.0 mg/100ml, muscular man – 11.4 mg/100ml

*If value doubles, GFR – and renal function – probably have fallen to half of normal state.

*If value triples – suggests 75% loss of renal function.

*Values of 10 mg/100 ml – 90% loss of function

**C. Urinalysis** – Normal urine contains metabolic wastes and little, if any, plasma proteins, blood cells, or glucose.

Casts – molds of distal nephron lumen. Tamm and Horsfall mucoprotein (gel-like substance) forms the matrix of casts. Hyaline casts – contain Tamm and Horsfall mucoprotein, without cells. Develop when protein content of urine is high (such as nephrotic syndrome) urine osmo high, urine pH low.

**D. Other Serum lab tests**

1. Potassium, phosphate – tend to increase in renal failure
2. Calcium, pH, bicarbonate – tend to decrease in renal failure.

**E. Cystoscopy** – visualize the urethra, bladder, and ureteral orifices. Biopsy specimens, small stones, lesions, small tumor, and foreign bodies can be removed from urethra, bladder, or ureters by this means.

**F. Radiologic exams**

1. Computerized axial tomography (CAT) delineate tissue at any level. May be used to outline kidney and detect tumors therein.
2. Radiopaque iodine contrast medium – allows for visualization of urinary structures. Dye can be introduced into urinary system or into a vein.

3. Intravenous pyelogram (IVP) – allows for x-ray visualization of renal calyces, renal pelvis, and ureters as dye is excreted by the kidneys.

4. Retrograde pyelography – cystoscope is used to introduce dye into the ureters.

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