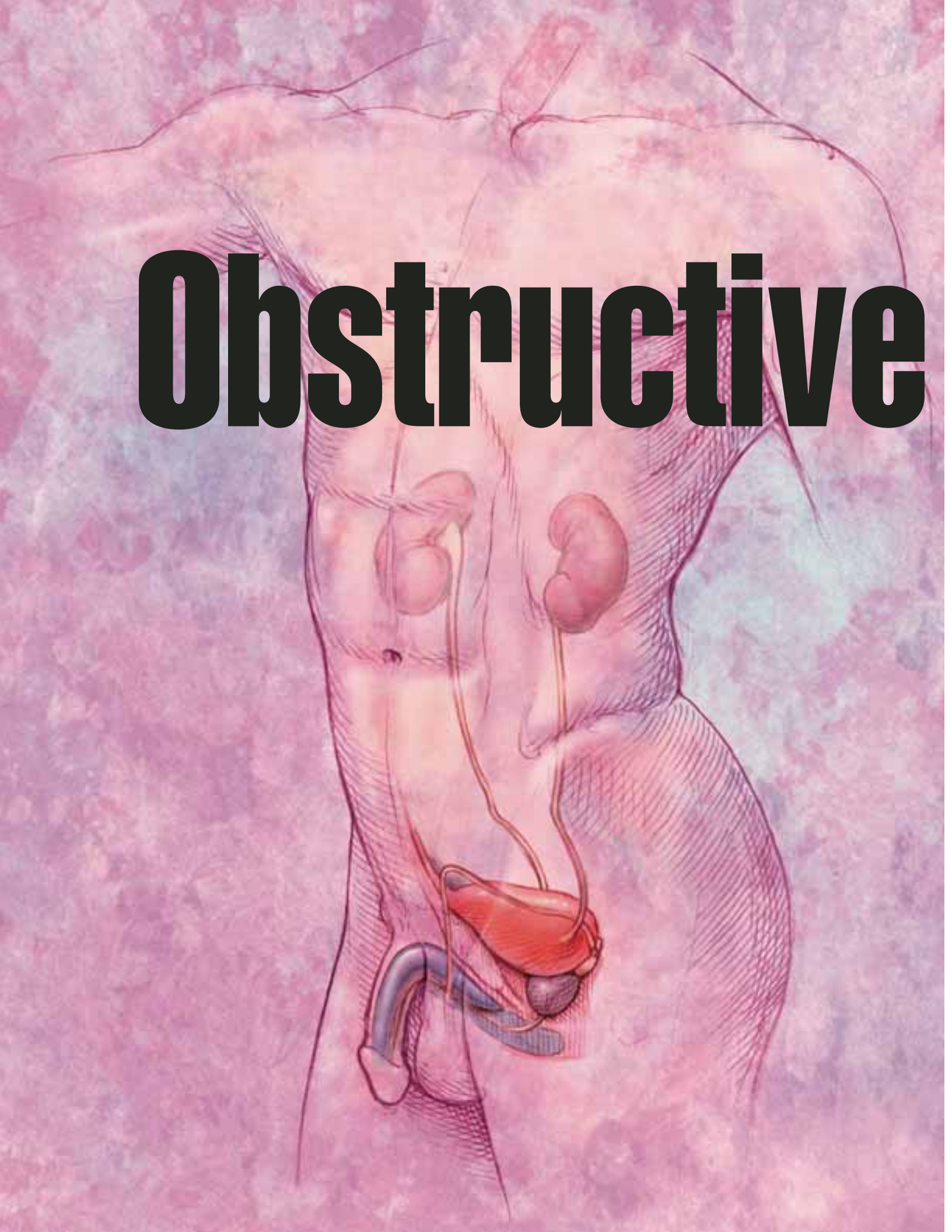
Obstructive



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Uropathies

Urinary flow can be obstructed at any location between the glomerulus and the end of the urinary meatus.[2] The clinical pathology can cause increased intraluminal pressure, urinary tract infection (UTI), urinary stasis, formation of stones, and the possible loss of total renal function.[2,3] The CE article in September's Journal, "Renal Transitional Cancer," discussed obstructive tumors and reviewed the anatomy of kidneys. This article discusses the remaining obstructive uropathies.

Kidney stones are one of the most common of these disorders. The number of cases has increased steadily over the last 20 years. As many as 10% of all Americans will have a kidney stone in their lifetime. The disorder affects men more often than women; however, the number of cases in women has also risen over the past 10 years.[20]

Hydronephrosis

There are two types of hydronephrosis, primary and secondary. With primary, there is no ureteral dilation and the obstruction occurs at the ureteropelvic junction. The causes of obstruction are due to: intrinsic stricture; a defect in the function of the smooth muscles at the ureteropelvic; a high insertion of the ureters into the renal pelvis; a kinking or compression of the ureters; and finally a renal stone or a renal tumor.[2,3]

The secondary type is due to a reflux from the vesicoureteral area, and a possible distal obstruction.[2,3] Other causes of secondary obstruction include: stones and tumors; compression by nearby primary or metastatic tumors; diseases that cause myoneurogenic pathology of the ureters and the bladder; secondary fibrosis due to surgery and radiation therapy; fibrosis in the retroperitoneal area; malignancies of the renal system; ureterocele; congenital or acquired vesicoureteral reflux; malignant or benign prostate disease; meatal stenosis; and congenital deformities.[2,3,14] (Table 1)

In female patients, hydronephrosis is a concerning problem during pregnancy. As the uterus enlarges it puts pressure on the ureters and may lead to a mechanical obstruction causing hydronephrosis. The effect of progestational hormones can also be problematic. Antenatal hydronephrosis can lead to chronic UTI.[3,4]

Clinical pathology

The most common symptom is pain.[14] Pain in the urinary tract is caused by distention of the collection system and renal capsule due to obstruction. The severity of pain is due to the rate of distention. In the case of acute supravesical obstruction by a stone in the ureter, this type of pain is continuous and often radiates to the lower abdomen including the testes or labia.[2,3,14,16] When the patient has a more insidious obstruction, as in the narrowing of the ureteropelvic junction, the patient may have little or no pain.[2,3,14]

Azotemia occurs when the overall excretory function is impaired.[2,3,14,15] If anuria is present, acute renal failure is suspected.[14,15] Patients with acute renal failure and a history of nephrolithiasis, hematuria, diabetes mellitus, prostatic enlargement (either benign or malignant), trauma, surgery, should be worked up for possible urinary tract obstruction.[2,3,4] The patient with bilateral obstruction will show prerenal azotemia. As the obstruction becomes larger, the patient will show polyuria and nocturia with impaired ability to concentrate urine.[2,3,4,14,15] This concentration defect is due to disturbances in the sodium-chloride pump within the ascending loop of Henle, which causes the nephron to decrease medullary hypertonicity, leading to the concentration defect.[9,12,14,18,19]

In patients with partial urinary obstruction, the amount of urine output is greatly increased, and if the patient has poor fluid intake, there is a greater possibility for severe dehydration and hypernatremia.[14,16,18,19] In patients with partial bilateral urinary obstruction, severe derangements can occur, including acquired distal tubular acidosis, renal salt wasting and hyperkalemia.[2,3,14,16] These can lead to renal tubulointerstitial damage.

Urinary stasis promotes bacterial growth leading to urinary tract infections and the formation of crystals, especially magnesium ammonium phosphate (struvite stones).[3,6,7,9] In patients with acute and subacute, unilateral obstruction, patients will show hypertension due to the release of renin by the involved kidney.[3,6,14,16]

Urinary Calculi Nephrolithiasis Epidemiology

Stone formation is as high as 12%. Males are three times more likely to form a renal stone generally during the third through fifth decades.[2,14] Hereditary diseases and some genetic predisposition may contribute to formation.[8] The patient's lifestyle is a main factor in the specific type of stone formation.

Patients that live in a particular area (mountains, tropical and desert) and those that have a sedentary lifestyle or sedentary job, have a higher incidence of stone formation. There is a direct correlation with warmer weather and stone formation in patients residing in the southeastern region of the United States.[8] Patients on certain medications, such as diuretics, are predisposed to stone formation.[9,10] Studies have shown that

Table 1 **Causes of mechanical urinary tract obstruction**

Acquired intrinsic factors

Ureter	Bladder	Urethra
blood clots	benign prostatic hyperplasia (BPH)	trauma
tumors	calculi	tumor
trauma	diabetic neuropathy	phimosis
inflammation	spinal cord disease and trauma	stricture
calculi	cancer	
	anti cholinergic drugs	

Acquired extrinsic factors

Ureter	Bladder	Urethra
pregnancy	trauma	trauma
surgical ligation of the ureter	CA of cervix and colon	
CA prostate, uterus, colon, bladder, rectum		
fibrosis of the retroperitoneal area		
pelvic inflammatory disease (PID)		

Congenital

Ureter	Bladder	Urethra
ureterocele	ureterocele obstruction	abnormal anterior and
ureterovesical obstruction	of the bladder neck	posterior urethral valves
ureteropelvic obstruction		

patients with increased fluid intake have a lower incidence of stone formation.[9]

Cases of renal stone formation in children are generally metabolic, urologic anomalies, infection and finally immobilization. Patients with a history of renal stone formation will have a recurrence rate within the first year by one-third of all patients. This is probably due to an anatomical abnormality with the urinary tract system.[10]

Physiology of stone formation

Urinary stone formation occurs when the urine becomes supersaturated with particulate matter. Proteins normally found in the urine inhibit the formation of crystals from the particles. Stones form when an imbalance occurs. Another factor that contributes to urinary stone formation is urinary stasis due to structural abnormalities of the urinary system, neurogenic disorders, or presence of foreign material.

Types of stones

Calcium stones

Calcium stones are the most common type affecting men more often within the third decade of life. Patients that form their first calcium stone will generally reform another stone within approximately the next 10 years. This type of formation is generally familial.[3,9,14] (Table 2)

Calcium excretion by the kidneys is increased in certain conditions as 1) a high dietary intake of calcium, 2) immobilization, and 3) hyperparathyroidism.[9,16,18] Patients with hyperparathyroidism will show hypercalcemia with hypercalciuria.[2,3,6] Renal damage that occurs with

Table 2 **Causes of renal stone formation**

Type	Etiology	Treatment
Calcium Stone Hypercalcuria	Hereditary	Thiazide diuretic
Hyperuricosuria	Diet	Diet and or Allopurinol
Primary hyperparathyroidism	Cancer	Surgery
Distal renal tubular acidosis	Hereditary	Alkali replacement
Intestinal hyperoxaluria	Bowel surgery	Cholestyramine or oral calcium
Hereditary hyperoxaluria	Hereditary	Pyridoxine and fluid
Idiopathic stone disease	Unknown	Oral phosphate
Uric Acidstone (gout)	Hereditary	Alkali and Allapurinol
Idiopathic	Hereditary	Alkali and Allapurinol
Dehydration	Habit, Intestinal	Alkali, fluids
Lesch-Nyhan syndrome	Hereditary (males only)	Allopurinol
Malignant tumors	Cancer	Allopurinol
Cystine stones	Hereditary	Fluids, Alka, D-penicillamine
Struvite stones	Infection	Anti microbial agents

this disease is due to hypercalcemia and an increased secretion of parathyroid hormone (PTH). The hypersecretion of parathyroid hormone causes an increased secretion of phosphate in the urine, thus causing an increase in urinary alkalosis leading to calcium precipitation.[2,3,6]

Renal damage occurs as a consequence of hypercalcemia, and depends on the degree of the disease. Total renal damage is worse in patients that show nephrocalcinosis than in patients with renal calculi alone.[2,3,6]

Uric acid stones

Uric acid stones are more common in males.[2,3,6,14] Half of these patients will present with gout.[2,3,6,16] This is recurrent acute or chronic arthritis found in the peripheral joints that result in deposition of monosodium urate crystals in the tendons and joints due to over saturation of uric acid in body fluids. Other causes for gout are high ingestion of purines (found in meat, fish and poultry), alcohol abuse, emotional stress, and fatigue.[2,3,6,14]

Uric acid buildup can cause an acute monoarticular joint involvement (typically in the great toe). Generally, the first symptom is nocturnal pain, followed by joint pain with increasing intensity, inflammation, and the overlying skin appearing tense and shiny.[2,3,4,14] The patient may also exhibit pain in other joints (wrist, knee, ankle) and as well as fever, tachycardia, chills and increased white blood count.[2,3,14]

Renal uric acid lithiasis is more familial, even if gout is not present.[2,3,14] The crystals appear as red-orange in color.[16]

Struvite stones

Struvite stones are very common and can be very dangerous.[2,3,14] These stones are more common in patients who experience chronic urinary tract infections caused by the urease-producing bacteria, proteus. Ten percent of these stones are magnesium-ammonium-phosphate.[2,3,14,16]

When urea-splitting bacteria cause an infection in the renal pelvis, a struvite or staghorn calculi is formed. Generally these are larger in size because of formation within the renal pelvis. In patients with staghorn calculi, antibiotics are not useful, due to poor penetration into the calculi.[2,3,14,16]

Along with struvite stones, cysteine and uric acid stones often grow larger than the ureter, and at that point they fill-up the renal pelvis and extend outward through the infundibula into the renal calyces.[2,3,14,16]

Diagnosis

Patients with a history of dysuria, pain, infection and any changes in urinary output and evidence of a distended abdomen with a palpable

kidney or bladder should be examined further.[2,3,14] A rectal exam should be done to rule out an enlarged prostate, abnormal rectal sphincter tone, or a pelvic or rectal mass.[4,14]

In the male patient, inspection of the meatus for stenosis or the presence of phimosis.[7,11,12] In the female patient, palpation and inspection of the vagina, rectum and uterus should rule out secondary causes of urinary tract obstruction.[4,14] Complete urinalysis is performed to rule out hematuria, pyuria and bacteriuria; however, in the early stage of UTI, the urine sample may not be abnormal.[16] A scout film of the abdomen may be obtained to see if it's possible that nephrocalcinosis or a radiopaque stone is present.[3,4,14]

A Foley catheter may be inserted if urinary tract obstruction is suspected. If diuresis does not occur, an abdominal ultrasonography should be performed to assess renal, ureteral and bladder size. On ultrasound, the detection of hydronephrosis is approximately 90% accurate. Ultrasound may not show hydronephrosis in the presence of staghorn calculi, infiltrative renal disease or retroperitoneal fibrosis.[3,14]

In most cases, intravenous urogram is used to define the site of obstruction.[3,4] The nephrogram may be more beneficial than a urogram because the contrast medium is more concentrated. The best visualization of a lesion within the ureter or kidney pelvis may occur during retrograde or antegrade urogram. Intravenous urography can cause a contrast-induced acute renal failure in patients with proteinuria, renal insufficiency or diabetes mellitus.[3,4,14]

Retrograde urogram is performed by placing a catheter within the ureter via cystoscope. Antegrade urography is performed by placing a catheter percutaneously within the renal pelvis with the assistance of ultrasound or fluoroscopy.[11,12,13] Patients with intermittent ureteropelvic obstruction must have a radiologic evaluation while they are in pain, to locate the obstruction.[14] Also, overhydration can provoke a symptomatic attack.[2,3] Voiding cystourethrography is best for the diagnosis of vesicoureteral reflux and bladder neck and urethral obstruction.[7,11,12,13,14] If radiographic films fail to show the obstruction, endoscopic visualization in surgery permits precise identification of lesions of the urethra, bladder and ureteral orifices.[3,14]

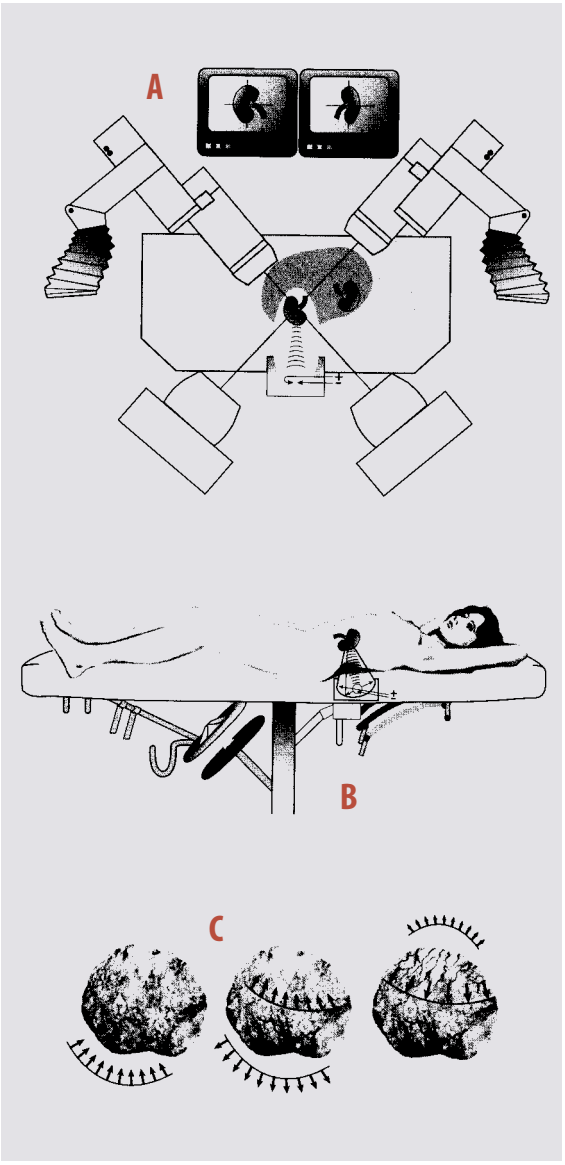

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FIGURE 1

ESWL.

- Two x-ray beams crossing at second focal point for proper positioning of stone.
- Focal point.
- Reflection of shock waves at first and second acoustical interfaces of stone and surrounding fluid with fracture of stone.

Treatment

Treatment of a stone in the kidney and/or urinary tract requires a combination of medical and surgical approaches. The medical treatment depends on the location of the stone. The extent of obstruction directly affects the kidney, and presence of urinary tract infection.[3,4,14]

Historically, stones were removed by an open surgical procedure (pyelolithotomy and ureterolithotomy) or cystoscopy. Now medical advances provide alternative, less-invasive pro-

cedures. Electrohydraulic Shock Wave Lithotripsy (ESWL) is performed by giving the patient shock waves to fragment the stone in the kidney, renal pelvis or ureter (Figure 1). Percutaneous ultrasonic lithotripsy is performed by passing a rigid nephroscope into the renal pelvis through a small incision in the flank. Laser lithotripsy, performed endoscopically, is also an option.[3,4,14]

Prognosis

The main factor in prognosis is the removal of the obstruction, which will determine whether any renal damage is reversible. Other factors affecting outcomes are whether the obstruction was complete or incomplete, bilateral or unilateral, and if a urinary tract infection was present.[3,4] Patients with complete renal obstruction have a worse prognosis, due to renal damage that may not be reversible.[3,4,16] Renal damage may be noted on a radionuclide scan.[14]

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References

1. Robbins SH. *Pathologic Basis of Disease,* 2nd ed. Philadelphia, Pa: Saunders;1979: 1180-1181.
2. Berkow R, et al, eds. *The Merck Manual,* 16th ed. Rahway, NJ: Merck Research Laboratories; 1992: 1803-1920.
3. Braunwald E, et al, eds. *Harrison's Principles of Internal Medicine.* 15th ed. New York, NY: McGraw Hill Medical Publishing Division; 2001: 1456-1635, 492-493.
4. Schwartz SI, et al, eds. *Principles of Surgery.* 4th ed. New York, NY: McGraw-Hill;1984: 1706-1707, 325-327, 335.
5. Ballinger WF, et al, eds. *Alexander's Care of the Patient in Surgery.* 5th ed. St Louis, Mo: Mosby;1972.
6. Sefel A and Resnick MI. *Metabolic Evaluation of Urolithiasis.* Urol Clin North Am. 1990 Feb;17(1):159-69.
7. Walsh PC, et al, eds. *Campbell's Urology.* 6th ed. vol 3. Philadelphia, Pa: Saunders; 1992.
8. Soucie JM, Coates RJ, McClellan W, Austin H, Thun M. Relation between geographic variability in Kidney Stone prevalence and risk factors for stones. *Am J Epidemiol.* 1996 Mar 1;143(5):487-95.
9. Borghi L, Meschi T, Amato F, Briganti A, Novarini A, Giannini A. Urinary volume, water and recurrences in idiopathic calcium nephrolithiasis: a 5-year randomized prospective study. *J Urol.* 1996 Mar;155(3):839-43.
10. Kroovand RL. Pediatric Urolithiasis. *Urol Clin North Am.* 1997 Feb;24(1):173-84.
11. Brenner BM, ed. Brenner and Rector's *The Kidney.* 6th ed. Philadelphia, Pa: Saunders; 2000: 1820-1843.
12. Walsh PC et al, eds. *Campbell's Urology.* 7th ed. Philadelphia, Pa: Saunders; 1998: 342-385.
13. Schrier RW and Gettschalk CW, eds. *Diseases of The Kidney.* 6th ed. Boston: Little Brown; 1997: 709-738.
14. Tintinalli JE, et al, eds. *Emergency Medicine: a comprehensive study guide.* 5th ed. New York: McGraw Hill, Health Professions Division; 2000: 611-661, 1967-1987.
15. Sweeney LJ. Basic *Concepts in Embryology: a student's survival guide.* New York: McGraw Hill, Health Professions Division; 311-324.
16. Bakerman S. *A,B,C's of Interpretive Laboratory Data.* 2nd ed. Greenville, NC: Interpretive Laboratory Data; 1984: 360-364.
17. Snell RS. *Clinical Anatomy for Medical Students.* 4th ed. Boston, Mass: Little, Brown; 1992:256-301.
18. McCance KL and Huether SE. *Pathophysiology.* 2nd ed. St Louis, Mo: Mosby; 1994: 1212-1276.
19. Guyton AC. *Physiology of the human body.* 6th ed. Philadelphia, Pa: Saunders; 1984: 273-353.
20. Kidney Stones in Adults. National Kidney and Urologic Diseases Information Clearing-

house. ***www.niddk.nih.gov/health/urolog/pugs/sonadul/sonadul.htm*** Accessed 9/12/02

Sources

1. Berkow R, Fletcher AJ, eds. *The Merck Manual.* Vol. I. 15th ed. Rahway, NJ: Merck, Sharp, and Dohme Research Laboratories; 1987.
2. Andreoili TE. *Cecil Essentials of Medicine.* 2nd ed. Philadelphia, Pa: Saunders; 1990.
3. Shoemaker WC, et al, eds. *Textbook of Critical Care.* 2nd ed. Philadelphia, Pa: Saunders; 1989.
4. Conn HF, Clohecy RJ, and Conn RB, eds. *Current Diagnosis.* 6th ed. Philadelphia, Pa: Saunders; 1980.
5. Sodeman Jr WA and Sodeman TM. *Sodeman's Pathologic Physiology: Mechanisms of Disease.* 6th ed. Philadelphia, Pa: Saunders; 1979.
6. Walter JB, Israel MS. *General Pathology.* 6th ed. New York, NY: Churchill Livingstone; 1987.
7. Sidransky H. Nutritional Pathology: *Pathobiology of Dietary Imbalances.* Biochemistry of Disease Series. Vol 10. New York, NY: Dekker; 1985.
8. Thomas SJ, ed. *Manual of Cardiac Anesthesia.* New York, NY: Churchill Livingstone; 1984.
9. Fuller JR. *Surgical Technology.* 2nd ed. Philadelphia, Pa: Saunders; 1986.
10. Schwartz SI, Shires GT. *Principles of Surgery.* 3rd ed. New York: McGraw-Hill; 1979.
11. Spence AP. *Basic Human Anatomy.* 2nd ed. Menlo Park, Calif: Benjamin/Cummings; 1986.
12. Hollinshead WH and Rosse C. *Textbook of Anatomy.* 4th ed. Philadelphia, Pa: Harper and Row; 1985.
13. Weiss L. *Cell and Tissue Biology: a Textbook of Histology.* 6th ed. Baltimore: Urban and Schwarzenberg; 1988.
14. Guyton AC. *Physiology of the Human Body.* 6th ed. Philadelphia, Pa: Saunders College Publishing; 1984.
15. Hole Jr JW. *Human Anatomy and Physiology.* Dubuque, Iowa: William C Brown Company; 1978.
16. Milnor WR. *Cardiovascular Physiology.* New York, NY: Oxford University Press; 1990.
17. Little RC. *Physiology of the Heart and Circulation.* Chicago: Year Book Medical Publishers; 1971.
18. Harper HA, Martin DW. *Harpers Review of Biochemistry.* 20th ed. Martin DW, et al, eds. Los Altos, Calif: Lange Medical Publications; 1985.
19. Ballinger WF, Treybal JC, Vose AB. *Alexander's Care of the Patient in Surgery.* 5th ed. St Louis, Mo: Mosby; 1972.
20. White A, Handler P, Smith EL. *Principles of Biochemistry.* 5th ed. New York, NY: McGraw-Hill; 1973.
22. Tortora GJ, Evans RL. *Principles of Human Physiology.* 2nd ed. New York, NY: Harper and Row; 1986.
23. Hall-Braggs ECB. *Anatomy as a Basis for Clinical Medicine.* 2nd ed. Baltimore, Md: Urban and Schwarzenberg; 1990.
24. Armstrong FB. *Biochemistry.* 3rd ed. New York, NY: Oxford University Press; 1989.
25. Porter R, O'Connor M, Whelan J. *Mobility and Function in Proteins and Nucleic Acids.* Ciba Foundation Symposium 93. Summit, NJ: CIBA Pharmaceutical Co; 1983.
26. Adams RLP, Knowler JT. *The Biochemistry of the Nucleic Acids.* 10th ed. New York, NY: Chapman and Hall; 1986.
27. Archakov AI, Bachmanova, GI. *Cytochrome P450 and Active Oxygen.* English ed. New York, NY: Taylor and Francis; 1990.
28. Wills ED. *Biochemical Basis of Medicine.* Bristol: Wright; 1985.
29. Martin BR. *Metabolic Regulation: a Molecular Approach.* Boston, Mass: Blackwell Scientific; 1987.
30. Frisell WR. *Human Biochemistry.* New York, NY: Macmillan; 1982.
31. Martinez J. *Peptide Hormones as Prohormones: Processing, Biological Activity, Pharmacology.* Ellis Horwood Series in Pharmacological Sciences. New York, NY: Halsted Press; 1989.
32. Norman AW, Litwack G. *Hormones.* Orlando, Fla: Academic Press; 1987.
33. Dixon M, Webb EC. *Enzymes.* 3rd ed. New York, NY: Academic Press; 1979.