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Editorial Review

Obstructive Nephropathy

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INTRODUCTION

Obstructive uropathy is not only a common cause of loss of renal function but is also potentially reversible.

Different terms are used to discuss the urinary tract obstruction. Obstructive nephropathy is referred to the functional and pathologic changes in the kidney due to obstruction. The terms obstructive uropathy is used as a general term to indicate partial or complete obstruction to flow of urine, any where from the renal calyx to external urethral meatus. The pressure proximal to obstruction must rise so that urine may pass through the point of narrowing at its usual rate. Hydronephrosis refers to abnormal dilatation of renal pelvis and calyces with various degrees of renal parenchymal atrophy. Hydroureter is used to describe dilatation of ureter. It is important to remember that presence of hydronephrosis or hydroureter does not necessarily mean that obstruction exists and such changes may or may not be associated with renal parenchymal damage. Vesicoureteral reflux, primary megureter, urethral dilatation associated with pyelonephritis, high urine flow states (e.g. a mannitol induced diuresis) and residual ureteral dilatation following surgical correction of obstruction are examples of non-obstructive causes of ureteral dilatation.

Incidence and causes

The age related incidence of obstructive uropathy seems to have a bimodal curve. Campbell[1] have indicated that approximately 2% of the pediatric population coming to autopsy had some form of obstructing lesion in the urinary tract. Out of these patients 81% were below the age of 1 year. The overall incidence at autopsy in adults is between 3.5 and 3.8%[2]. Between the ages of 20 and 60 years, hydronephrosis is considerably more frequent in women than in men, principally because of pregnancy and pelvic cancer. Over the age of 60, the majority of patients are men, this preponderance being related to the high frequency of prostatic hypertrophy and carcinoma in this group.

It has been calculated that 166 patients per 100,000 population were hospitalized in United States in 1985 with a presumptive diagnosis of obstructive uropathy. In 1985, slightly in excess of 397,000 hospital discharge diagnoses were recorded as obstructive uropathy in United States[2].

Classification

Ureteral obstruction may be classified in a variety of ways. It may be classified according to duration of obstruction. Acute obstruction is said to exist when the cause of obstruction is of short duration (hours or days). Chronic obstruction is used to describe those lesions present for months or years, as in ureteropelvic and ureterovesical abnormalities. Obviously subacute obstruction is an intermediate classification and is used to describe obstruction with a duration of days to weeks. Ureteral obstruction may also be classified according to the site of lesion. Intrarenal obstruction is said to exist when intratubular obstruction is present. The lesion is said to be upper urinary tract obstruction if it exists above the uretero-vesical junction. In this case it is usually unilateral in nature. It may be defined as lower urinary tract obstruction when the lesion is located at some site below the uretero-vesical junction (i.e. within the bladder trigone or at some point along the urethral. In this setting the ureteral obstruction will be bilateral in nature. Obstructive uropathy may be defined according to the degree of occlusion. Thus complete high grade or total obstruction is said to be present when the lumen of the affected segment of the urinary tract is completely obstructed, where as incomplete or low grade obstruction is said to be present when the offending lesion produces only partial occlusion. Finally, ureteral obstruction can be defined according to cause as shown in Table 1. In this classification the causes are divided initially into intrinsic and extrinsic categories. The intrinsic causes can be again divided into intraluminal and intramural. The extrinsic causes are best divided into groups based on the system (i.e. vascular, gastrointestinal, reproductive) from which the obstruction originates. It is important to remember that the most common cause of intrarenal

Table 1. Causes of obstructive Nephropathy.

I. Intrinsic causes

A. Intraluminal
1. Intrarenal
   Uric acid,
   sulphonamide
   mutiple myeloma?
2. Extrarenal
   Calculi,
   papillary tissue,
   blood clots,
   fungus ball

B. Intramural
1. Functional
   a. Ureteral
      Ureteropelvic or ureterovesical dysfunction
   b. Bladder (neurogenic)
      (1) Congenital
         Myelodysplasia
         Spinal cord defects
      (2) Acquired
         Tabes dorsalis
         Diabetes mellitus
         Multiple sclerosis
         Spinal cord trauma
         Parkinson's disease
         Cerebrovasclar disease
   c. Bladder neck dysfunction
2. Anatomic
   a. Tumors (polyps, carcinoma)
   b. Granulomatous infection (tuberculosis, ureteritis cystica)
   c. Strictures (radiation therapy: postinstrumentation)
   d. Posterior and anterior urethral valves
   e. Ureteroceles
   f. Trauma

II. Extrinsic causes

A. Reproductive system
1. Females
   a. Uterine
      (1) Pregnancy
      (2) Tumor
         (a) Fibroadenoma
         (b) Carcinoma of the cervix
      (3) Uterine prolapse
      (4) Endometriosis
   b. Ovarian
      (1) Abscess
      (2) Tumor
      (3) Cysts
2. Males
   a. Benign prostatic hypertrophy
   b. Adenocarcinoma of the prostate

B. Gastrointestinal system
1. Crohn's disease
2. Pancreatitis (pseudocyst)
3. Appendicitis
4. Malignancy

C. Vascular system
1. Aberrant vessels at the ureteropelvic junction
2. Aneruysmal dilatation
   a. aorta
   b. Iliac vessels
3. Venous
   a. Ovarian
   b. Vena cava ("retrocaval ureter")

D. Retroperitoneal
1. Inflammatory
   a. Idiopathic fibrosis
   b. Secondary fibrosis
2. Iatrogenic (surgical complications)
3. Tumor (lymphoma, metastatic or primary carcinoma)
4. Infection (abscess)
5. Hemorrhage (hematoma)
6. Lymphocele
7. Urinoma (post-trauma)

obstruction is hyperuricemia secondary to treatment of malignant disease[3] and that urolithiasis is the most common cause of intrinsic extra renal obstruction[3].

The intramural causes of obstruction are divided into functional and anatomic categories. Functional causes are related to dynamic abnormalities in the urinary tract. In the ureter these are usually due to a failure or poor transmission of peristaltic waves through a segment[4]. This defect may be related to a disproportionate decrease or absence of smooth muscle. The most common sites of functional obstruction in the ureter are at the pelvic[5] and urinary bladder junctions. The urinary bladder is another site where functional obstruction can be an important cause of obstructive nephropathy. In this case neurogenic bladder dysfunction can be a consequence of upper neuron damage and produce involuntary micturation (Spastic bladder dysfunction) or lower spinal tract injury and give rise to a flaccid atonic bladder. In both cases the ureterovesical reflux and dilatation can occur, which will result in a significant increase in back pressure

and produce renal parenchymal damage. The extrinsic causes of obstructive uropathy should be suspected because of the clinical diagnosis of disease states frequently associated with urologic obstruction. For instance the diagnosis of regional ileitis involving the cecum and colon should make one to suspect right ureteral obstruction.

Mechanisms of renal impairment in obstructive nephropathy

The effects of obstruction result from a variety of factors with complex interactions. Changes in renal blood flow, glomerular filtration and tubular function occur.

Renal hemodynamics in obstructive nephropathy

Glomerular function

A) Unilateral complete ureteral obstruction (UUO)

Most studies have describe a triphasic renal vascular response[6].

- The initial phase, occurring in the fist 1-2 hour after obstruction is characterized by a transient rise in blood flow, due to afferent arteriolar vasodilatation, trying to maintain GFR, against raised intra tubular pressure[2].

  This arteriolar dilatation is mediated by intrarenal mechanisms as indicated by its occurrence in denervated renal transplant[7] and in isolated perfused kidney. Most of the evidence indicate that local production of prostaglandins, mainly prostacyclin and PGE2, may account for this change[8,10,3].

- The second phase begins about 2 hours after obstruction and lasts approximately 3 hours. This phase is characterized by elevated postglomerular (efferent) renal vascular resistance, in addition to afferent arteriolar dilatation. By increasing intraglomerular hydrostatic pressure, these changes tend to maintain GFR.

  The third phase, beginning after 5 hours of obstruction, is characterized by marked rise of renal vascular resistance, especially preglomerular (afferent arteriole). At this time GFR will decrease.

  Increase in renal vascular resistance is due predominantly to an increase in the resistance of afferent arterioles[11]. The increase in resistance is mediated by several vasoconstrictors[12]. Three major vasoconstrictors of the renal circulation, angiotensin II, thromboxane A_2, and ADH, play a role in decreasing the renal plasma flow per nephron and decrease in SNGFR[13-15]. Besides being potent vasoconstrictors, both angiotensin II and thromboxane A_2 have been shown to contract mesangial cells in culture and, therefore, can potentially reduce the glomerular capillary area available for filtration. Leukotrines, potent mediators of inflammation, are synthesized by cells through 5-lipoxygenase pathway. Increased synthesis of leukotrine B_2 has been found in isolated glomeruli from rats with (BUO)[2].

B) Bilateral complete ureteral obstruction (BUO)

The phases are similar to those described above but changes in afferent arteriolar resistance is not as marked as in UUO, thus maintaining GFR at a relatively higher level[11].

C) Partial ureteral obstruction (PUO)

Only few studies are available[16]. Unlike significant obstruction, mild partial obstruction is characterized by afferent arteriolar dilatation, increasing intraglomerular pressure to maintain GFR[17].

D) Pathophysiology of glomerular function changes

Reyes et al.[18] have recently examined the potential contribution of EDRF to the changes in glomerular filtration and effective renal plasma flow observed after unilateral release of BUO. The result of these studies suggest a decreased availability of the substrate for EDRP synthesis during BUO. The results can also be interpreted to indicate that decreased EDRF activity during obstruction plays a role in the hemodynamic changes observed after release of BUO of 24 hours duration. Recent data also suggest that platelet activating factor (PAF) has a vasodilatory role in obstructive nephropathy[2].

Ureteral obstruction increases renin secretion. Increased renin release may result from stimulation of intra renal mechanisms due to reduced delivery of sodium and chloride to the macula densa or to a reduction in transmural pressure at the baro-receptor as a consequence of the dilatation of afferent arteriole described above. The vaso dilatory

eicosanoids, such as prostacyclin or PGE_2, play a role in renin release from Juxta glomerular cells[19]. The increased renin secretion leads to increased intra renal production of angiotensin II.

At least two sources appear to account for the increased synthesis of thromboxane in the obstructed kidney, infiltrating leukocytes[20] and intrinsic glomerular cells. Infiltrating leukocytes consist mainly of macrophages and T-cells.

E) Tubuloglomerular feedback in obstructive uropathy

In various studies, it has been observed that urinary tract obstruction may modulate the activity of tubuloglomerular feedback[21,22,23].

F) Role of atrial natriuretic peptide

It is a potent antagonist of various vasoconstrictors. Levels of atrial peptide in the circulation are markedly greater in animals with BUO than in those with UUO[2]. This may be responsible for relatively less afferent arteriolar constriction in BUO as compared to UUO.

Medullary circulation

Renal vasoconstrictors (angiotensin II, thromboxane and ADH) constrict medullary vessel. Atrial peptide enhance the action of vasoconstriction, and may exchange medullary blood flow in BUO, which by washing medullary tonicity, impair the concentrating ability of the kidney[11]. PGE_2 could also dilate medullary blood vessels[23].

Tubular abnormalities in obstructive nephropathy

Several abnormalities in tubular function have been described in obstructive nephropathy. These abnormalities include decreased reabsorption of solutes and water, impaired excretion of hydrogen and potassium and inability to concentrate the urine.

a) Reabsorption of solutes and water

The mechanisms underlying the decreased reabsorption of sodium are thought to be due at least in part to changes in the activity of Na-K-ATPase activity in the nephron[24].

Impaired ability to concentrate the urine is evident after relief of obstruction in rats with either UUO or BUO[25]. Vasopressin (ADH) administration does not reverse the defect[26]. Thus, the concentrating defect in obstruction is presumably due to:

1. A decreased absorption of solutes from the thick ascending limb of Henle's loop.
2. Washout of solute from the medulla due to increased papillary plasma flow (PPF).
3. Decreased hydroosmotic response of cortical collecting duct to ADH
4. Decrease in number of juxtamedullary nephrons.

B) Potassium and hydrogen excretion in obstruction

The fractional excretion of potassium is decreased in patients with obstructive nephropathy than in patients with comparable degree of renal insufficiency due to a variety of renal diseases[2]. Hyperkalemic/hyperchloremic acidosis is observed in patients with chronic obstructive nephropathy[27]. Three major hypothesies may explain how it develops:

1. A defect in renal H+ secretion, so that pH of the urine cannot be lowered maximally in the presence of systemic acidosis and the urinary excretion of both ammonium and titrable acid is decreased.
2. A defect in aldosterone secretion probably secondary to diminished production of renin by the kidney (hyporeninemic hypoaldosteronsim).
3. A combination of these two defects.

An inability to acidify the urine is seen after release of BUO or UUO in both human and experimental animals[26]. Moreover a distal renal tubular acidosis with inability to lower the urine pH to normal minimum values in responses to acidemia is common in patients with obstructive nephropathy.

Genesis of parenchymal damage during obstruction

Several factors contribute to the renal parenchymal damage (evidenced by appearance in urine of tubular enzymes e.g. N-acetyl glucosaminidase (NAG)[28] caused by ureteral obstruction: (1) There is definite evidence of necrosis due to increased pressure on medullary tissue. Pressure may cause local destruction of tissues in other areas also (2) Ischemia due to decreased blood

flow, appears to be an important factor early in the pathogenesis of the disease. Pressure from outside or kinking or distortion of vessels due to distended pelvis and calyces may decrease the blood flow. (3) Prolonged ureteral obstruction result in a proliferation of interstitial cells and the formation of dense fibrous tissue. When this process is extensive the microcirculation will be impaired, leading to ischemia. The increase in the intratubular pressure could result in back movement of fluid from renal tubule[29]. Secondarily ruptures or tears at the site of the renal fornices, due to high pelvic pressure, causes reentry of ureteral urine into interstium. Monocytic infiltrate by releasing growth factors may also contribute to fibroblast proliferation. The reduced nephron number caused by other mechanisms results in a remnant nephron population having increased glomerular filtration per nephron. This hyperfiltration of the remnant nephrons during a long period is believed to result in the characteristics histological lesion of focal segmental glomerulosclerosis[30-32].

Effects of contralateral kidney (CLK) in cases of unilateral obstruction

Unilateral obstruction is usually accompanied by contralateral natri-diuresis[33]. It may be due to following mechanisms:

1. Stimulation of renal pelvic receptors, leading to increase r-MSH activity, which by acting directly or through increased release of atrial natriuretic peptide, produces natri-diuresis[33].
2. Stimulation of renal receptor, by inhibitory renorenal reflex, decreases sympathetic activity in CLK.
3. Increased angiotensin II production and release into circulation by obstructed kidney, leads to enhanced prostaglandin · production by contralateral kidney, and these prostaglandins may be responsible for natri-diuresis[34,10].

INTERACTIVE OBSTRUCTIVE UROPATHY IN MAN

The entire urinary tract from glomerulus to external urethral meatus can be regraded as a single functional unit[35]. The purpose of this unit is the formation and unidirectional excretion of urine at low pressure in order to maintain normal body homeostasis. Sometimes changes in one part of the system can be overcome by alterations elsewhere, but on other occasions this may result in inefficient or poor quality urinary excretion. Such a state of affairs, if allowed to persist for any length of time, can result in nephron failure and a uremic death.

It has been proposed on the basis of theoretical models, that upper tract flow characteristics and pressures will vary according to the rate of urine production and the degree of bladder filling. These suggestions are supported by observations in pigs suggesting that detrusor instability in these animals influences upper tract pressures and that high pressure in porcine bladders can affect vesicoureteric junction transport[36].

Studies in man also suggested a relationship between bladder and renal function. There had been studies describing changes in the urinary volume excretion related to that bladder filling and proposing this may be due to a vesico-renal reflex, although subsequent attempts to clarify the nature of such a reflex have suggested that it may be a purely hydrodynamic phenomenon. This later view is supported by studies of intrvenous pyelography films in patients with full bladders or acute (painful) urinary retention which show, rather "full" looking ureters suggesting some delay in upper tract drainage.

Non-neuropathic bladder dysfunction

As long ago as 1955, it was suggested that there might be different types of chronic retention with differing effects on upper tract function. Studies had been performed by slowly filling, the bladder on top of the residual urine attempting to imitate more closely the natural cycle of events and subsequently in 1983 George et al.[37] Coined the term "high pressure chronic retention" (HPCR) to describe a group of patients with abnormally high intravesical pressures throughout the micturation cycle, thick-walled, trabeculated bladders and associated hydroureteronephrosis.

The upper tract dilatation appears to be related to the duration and magnitude of bladder pressure elevation during filling.

Relationship between upper and lower tract dynamics in HPCR

Standard radioisotope renography in patients with HPCR prior to any intervention shows poor excretion of isotope while the bladder is full or with patient supine. Assuming the erect posture or withdrawing a volume of urine equivalent to a

normal void from the bladder results in rapid wash out of tracer from the kidney[38]. This suggests that the hydrostatic pressure (about 25 cm of water in the erect posture) between kidney and bladder and regular voiding are important factors which contribute to upper tract drainage into bladder in HPCR. Indeed, prolonged periods of recumbency, can result in deteriorating renal function[38].

Neuropathic bladder dysfunction

Neuropathic bladder disorders are a well recognized cause of upper tract dysfunction, although this may take the form of either reflux or obstruction and certain urodynamic features are unique to these disorders.

A number of studies suggest a relationship between poor bladder compliance and upper tract dilatation[39,40]. Bladder outflow obstruction, usually detrusor sphincter dyssynergia or isolated distal sphincter obstruction, has also been associated with upper tract dilatation[41,42]. Bladder pressures between 20 and 40 cm of water have been shown to be deleterious to upper tract function and at a recent symposium on interactive obstruction the consensus was that detrusor pressures > 25 cm of water for any length of time during an individual's micturation cycle are likely, when repeated over a period of time, to produce significant upper tract obstruction, and therapeutic efforts should be directed towards maintaining lower pressures whenever possible[35]

CLINICAL SYNDROMES AND COMPLICATIONS OF OBSTRUCTIVE NEPHROPATHY

Although obstructive uropathy is a common cause of renal failure, the early presenting signs and symptoms of this disease are quite non-specific. In the incipient stages, it may be manifested only by the extra renal signs and symptoms of the underlying pathological process (e.g. local and distant metastasis of tumors that have occluded at some point in the urinary tract). In the end-stages the clinical course is dominated by the effects of renal functional impairment. There are, however, certain symptoms and signs that suggest the proper diagnosis, as listed in Table 2. The clinical manifestations of urinary obstruction will be conditioned by duration, location, and degree of obstruction.

Table 2: Clinical manifestation and laboratory findings suggestive of urinary tract obstruction.

I. Signs and symptoms
- A. Pain
- B. Renal enlargement - abdominal mass
- C. Recurrent or refractory urinary tract infections
- D. Gross hematuria
- E. Changes in urine out put (Anuria, Polyuria, Decreased stream, hesitancy, etc.)
- F. Hypertension

II. Laboratory changes
- A. Plasma electrolyte derangements
  - 1. Hyperchloremic, Hyperkalemic metabolic acidosis
  - 2. Hypernatermic Dehydration
- B. Alterations in the urinary sediment
  - 1. Crystalluria
  - 2. Bacteriuria
  - 3. Pyuria
  - C. Unexplained impairment of renal function.
- D. Polycythemia

III. History of factors that can cause obstruction
- A. Malignancy
- B. Previous abdominal, pelvic or genito-urinary surgery.
- C. Renal calculi
- D. Regional enteritis
- E. Methysergide
- F. Disorders associated with papillary necrosis e.g. diabetes mellitus, analgesic abuse, sickle cell disease.

Renal (flank) pain

The relationship of pain to urinary tract obstruction depends on the acuteness of the obstructing event. The pain is due to stretching of the collecting system or renal capsule[43] and is relieved when distension is eliminated. Its severity correlates with the rate of distension rather than degree of dilatation. Thus, urinary obstruction as seen with renal calculi is often associated with severe pain, whereas marked hydronephrosis induced by a more chronic lesion may be associated with only mild or absent flank pain. In the latter instance vertebral angle pain may be noted only after the ingestion of a large volume of fluid or a diuretic agent[44]. Acute obstruction usually produces a steady crescendo pain in the flank overlying the obstructed kidney, which radiates into groin and testicle or labia. The pain is constant and fluctuates very little. thus the term "renal colic" is a misnomer.

Renal enlargement

With longstanding obstruction the kidneys may enlarge and become readily palpable. Sometimes

marked hydronephrosis may present as a flank mass on physical examination. This is especially true in the pediatric population.

Recurrent or refractory urinary tract infection

Urinary tract infection is a common and potentially serious accompaniment of obstruction. Acute pyelonephritis with severe pyrexia, costovertebral angle pain and tenderness, or bacteremia may be presenting clinical features[15]. Recurrent bacteriuria and/or urinary tract infection may be the first clue to the presence of obstructive uropathy. Renal and perirenal abscesses[16] and xanthogranulomatous pyelonephritis[45] are more frequent in patients with obstructive uropathy. Candidal infection is also more prevalent[47].

Hematuria

Gross hematuria in a patient with acute or chronic renal failure should always alert one to the possibility of urinary tract obstruction from tumors, blood clots, or stones, and is not usually a feature of other causes of acute or chronic renal insufficiency.

Alterations in urine output

If the obstruction is bilateral and complete, total anuria result. Thus the presenting symptoms of such a patient may be those of acute renal failure. Similarly complete obstruction of a solitary functional kidney will lead to anuria.

When the obstruction is partial and long standing, the patent may note increased urine output or nocturia or both. Urine output may fluctuate. This is characteristic of intermittent obstruction and should be considered in individuals known to have only one functioning kidney and a history of renal stones.

Alterations in micturation are associated frequently with lower tract obstruction. So called "overflow incontinence" is a frequent symptom in the older aged population.

Hypertension

Acute and chronic hydronephrosis either unilateral or bilateral, may be accompanied by a significant elevation in blood pressure. The mechanisms responsible for the elevation in blood pressure appears to vary with the duration and type of obstruction. In patients with acute unilateral obstruction, renin secretion is usually enhanced and lateralizing renal vein renin studies similar to those in unilateral renal artery stenosis are often found.

In contrast, renin secretion is usually normal in patients with bilateral obstruction (including obstruction of a solitary kidney). In this condition, renal failure leading to volume expansion is commonly present. As a result, the elevation in blood pressure may be volume mediated, since relief of the obstruction usually leads to the loss of excess fluid and a fall in blood pressure[18].

Polycythemia

The occasional patient with obstructive nephropathy has an abnormally high erythrocyte mass. This polycythemia is presumably a consequence of increased synthesis and release of etythroprotein.

Electrolyte abnormalities

Obstruction may result in hyperchloremic hyperkalemic metabolic acidosis. In patients with partial obstruction, acquired nephrogenic diabetes insipidus may develop and lead to the development of hypernatremic dehydration. If salt wasting develops, patient may present with postural hypotension.

Changes in urinary sediment

Analysis of urine sediment and finding of hematuria, crystaluria or bacteriuria may provide very important information as to the etiology of obstruction.

Deterioration of renal function without apparent cause

The urinary tract obstruction can lead to further deterioration of renal function in patients with uremia or renal insufficiency.

Stone formation and papillary necrosis

Stone formation and papillary necrosis may result from obstruction. The former is most likely to occur in patients who become infected with a urease producing organism, such as proteus mirabilis. The ensuing alkaline urine favors the formation of magnesium-ammonium-phosphate (Struvite) stones, typically in the renal pelvis. Bladder stone may also form. Obstruction can also cause papillary necrosis[19].

Urinary ascites

Spontaneous intraperitoneal extravasation of urine is rare, but has been reported in children and rarely in adults.

Diagnostic imaging in obstructive nephropathy

In order to diagnose or exclude obstruction, most procedures rely almost exclusively on their ability to detect "Dilatation" of the collecting system. Therefore it is important to remember that obstruction can occur without dilatation[50-53]. This finding is seen in the following situations:

1. Partial (mild) and intermittent obstruction. These cases usually develop hydronephrosis when flow is high (about 10 ml/min).
2. When there is encasement of the collecting system in the retroperitonium by local tumors or fibrosis.
3. Straghorn calculi causing obstruction.
4. Acute obstruction in a person with volume depletion.

There can also be patients in whom hydronephrosis can be found in the absence of obstruction (non-obstructive dilatation). This occurs in the following situations:

1. Functional abnormalities, especially of uretero-pelvic junction (inability to transmit peristaltic waves) will lead to pelvic dilatation. Similar situation occurs in vesicoureteral reflux.
2. Chronic high urine flow states e.g. primary diabetes insipidus. Under conditions discussed above, this will also be non-obstructing.

The induction of diuresis to diagnose obstruction is now in common use. Diuresis is usually induced with furosemide. It is important to note that in renal impairment, the capability of furosemide to induce diuresis is reduced and it has been found that if single kidney GFR is below 32 ml/min, the response may be poor and it may be difficult to produce urine flow rate of 10 ml/min as suggested above, and then results of these diuretic test may not give much information[54,55]. Secondly there is problem of bladder interactions with upper urinary tract. At high flow rates, ureter is behaving like an open pipe and if bladder is full at that time with high intravesical pressure, then this will cause hinderance to urine flow and may cause false positive obstructive response[55]. So during these studies bladder must be kept empty. Thirdly to ensure prompt diuresis, patient should be at least euvolumic or slightly over hydrated.

It should also be remembered that rise of pressure to 15 cm of H_2O, may not be entirely benign and can cause tubular abnormalities, without reducing RBF and GFR.

Evaluation of the urinary tract with a variety of diagnostic imaging techniques is essential to detect urinary tract obstruction, to assess its severity, and to determine its cause. In all patients with acute or chronic renal failure when no obvious cause is found, urinary tract obstruction must be excluded by appropriate investigations. A wide range of procedures is available and may be appropriate depending upon the circumstances, including abdominal plain films and tomograms, intravenous urography, ultrasonography, radionuclide scanning, computed tomography, and invasive techniques of retrograde or antegrade pyelography including perfusion pressure studies.

A) Plain films and tomograms

The plain abdominal radiograph (KUB) may provide clues to the presence of obstructive uropathy. A difference of more than 2 cm in size between two kidneys or the finding suggestive of intraabdominal calcification is an immediate clue to possible urinary tract obstruction[56]. Renal tomograms provide better definition of renal size and shape and also detect smaller calcifications (as small as 2 mm in diameter). A large urinary bladder suggests bladder outlet obstruction as occurs in prostatic disease and may give as accurate an indication of the residual volume as real time ultra sound[57].

B) Intravenous urography

The intravenous urogram (IVU) combines the features of accurate anatomic demonstration of the kidneys, calyces, pelvis and ureters, bladder with an estimate of renal function[58,59] have shown good correlation of plasma clearance of contrast medium (as measured by x-ray flouresence analysis) with simultaneous and non-simultaneous Cr51-EDTA clearance as a measure of renal function.

Intravenous urography during diuresis by furosemide may be helpful in cases of suspected obstruction without dilatation and non-obstructed dilatation

Ultrasonography

1. Routine Ultrasonography

Diagnostic ultrasound is the procedure of choice

to determine the presence or absence of dilated calyces or renal pelvis and thus to suggest the presence of obstructive uropathy[60-62]. Since it is non-invasive and not dependent on renal function it is particularly useful to exclude hydronephrosis in patients with acute or chronic renal failure. Ultrasound is an extremely sensitive test for hydronephrosis, with a reported accuracy of greater than 90%. It can also be used to assess the degree of parenchymal atrophy accompanying hydronephrosis. Like all diagnostic procedures, it is not foolproof. Examples are conditions causing obstruction without dilatation. A major limitation of ultrasonography is, paradoxically, its extreme sensitivity in detecting. Small increases in volume of the renal pelvis. 26% false positive rate has been described[60]. Major entities that may cause false positive diagnosis of hydronephrosis include duplicated collecting systems, a full bladder, diseases producing high flow rates, inflammatory diseases, renal sinus cysts, vesicoureteral reflux, and arteries and veins coursing through renal sinus.
Ultrasonography is also useful to determine the residual volume of urine in prostatism and its deletrius effect on upper urinary tract.

2. *Diuretic Ultrasound*
Has been tried as a modification to properly diagnose non-obstructive dilatation and cases of obstruction without dilatation such as parial obstruction and intermittent obstruction and has shown good correlation with Whitaker's test[61]. Remember single kidney GFR should be more than 32 ml/min.

3. *Doppler Ultrasonography*
It may be useful in many ways.

   i) *Ureteric Jets-evaluation with color doppler sonography*
   Color doppler sonography holds promise as a non-invasive means of investigating urodynamics, and in particular, of confirming or excluding ureteric obstruction, by comparing peristaltic jets frequency and other parameters[63].

   ii) *Pulsed doppler ultrasonography*
   Has been found useful in differentiating minor degrees of hydronephrosis from conditions like arteries and veins coursing through the renal sinus[60].

   iii) *Doplex doppler sonography to diagnose significant obstruction*
   As significant obstruction is known to cause increase renal vascular resistance, which may be detected by measuring resistive index (RI) by duplex doppler sonography. A value above 0.70 is diagnostic of significant obstructive nephropathy, when associated with dilatation on routine ultrasound[64,7].

4. *Endoluminal Sonography of the Urinary Tract*
Very recently endoluminal sonography, using ultrasound transducers contained within 2 mm - diameter catheters, introduced through urethra, has been tried to detect cause of obstruction[65].

Radionuclide scanning

1) *Renography*
Radioisotope renogram will show delayed excretion in case of obstruction The third phase will be affected[66]. Conventional renography can give both false positive and negative results false positive in the case of non-obstructed dilatation and false negative in the case of partial or intermittent obstruction. To combat these problems, diuresis renography is used[67-72].

2) *Parenchymal transit time index (PTTI)*
Due to obstruction to urine flow, raised intratubular pressure and increased salt and water reabsorption in the proximal tubule prolongs the nephron transit time. This is well established and is the basis of parenchymal transit time increase in obstructive nephropathy, determined by the use of radionuclides[73].

3) *Radionuclide imaging of ureteric peristalsis*
Normal functioning ureters exhibit peristaltic contraction at a frequency of up to 3/min. Hyper-peristalsis that exceeds 4 contractions / min is associated with obstruction[74].

4) *Measurement of individual kidney GFR*
^{99m}TC-DPTA gives indication of severity of obstruction and in bilateral obstruction may

help to decide which kidney is going to be operated first.

Computed tomography and magnetic resonance imaging

Computed tomography (CT) may be useful as a secondary study to determine the etiology of previously diagnosed urinary tract obstruction. It is particularly helpful to determine potential causes of urinary tract obstruction in the retroperitoneal area. Non-radiopaque calculi composed of uric acid are also readily detectable by CT. Thus CT may increasingly replace the invasive diagnostic procedures of retrograde and antegrade pyelography in the evaluation of patient with urinary tract obstruction.

Nuclear magnetic resonance or magnetic resonance imaging is particularly useful in determining tissue densities and hence, like CT, may prove to be useful in determining etiology of obstruction.

Ureteropyelography

Pyelography by the retrograde or antegrade route provides detailed and rapidly available information about the location and cause of urinary tract obstruction previously detected by intravenous urography, ultrasonography, or radionuclide scanning[75]. Differentiating gold standard in dilated non-obstructed and obstructed urinary tract is perfusion pressure studies (Whitaker's test)[6].

RELIEF OF OBSTRUCTION: CHANCES AND PATTERN OF RECOVERY

Changes of recovery

Various experimental and clinical studies have shown that recovery after relief of obstruction depends on following factors:

A) Duration of obstruction

Prolonged duration of obstruction, decreases the chances of recovery after relief of obstruction[76-78].

B) Degree of obstruction

The greater the degree of obstruction, more intense will be changes in renal vascular resistance, leading to marked ischemic atrophy. Complete obstruction demands early relief, if renal function is to the preserved.

C) Age of the patient

Older studies in children have shown conflicting reports about the beneficial effect of release of obstruction. However more recent studies have shown that damage is more, and chances of recovery are less in children as compared to adults. While the mechanisms underlying these observations remain speculative, unique characteristics of the developing kidney, including greater activity of the intrarenal renin-angiotensin system, and increased renal vascular resistance may all contribute to impaired recovery from obstructive injury.

D) Infection

Infection by causing more severe inflammatory infiltrate will cause more severe damage[78,79].

E) Location of pelvis

Presence of an intrarenal or extrarenal pelvis and degree of pyelolymphatic and pyelovenous back flow may also affect degree of damage and chances of recovery[76].

Prediction of recoverability

1. *Intravenous urogram*

Prediction of recoverability using preoperative intravenous urogram has not been successful, although it has been found that if a kidney is able to concentrate the contrast, return of function may be expected, even with apparently thin cortex.

2. *Renal ultrasonography*

Renal ultrasonography, may be helpful by showing the renal cortical thickness.

3. *Renal scanning*

Renal scanning with ^{99}mtechnitium DTPA, ^{131}iodine-hippurate and ^{99}mtechnitium DMSA has been found useful to predict recovery[80] have shown that patients in whom DMSA uptake was 10% or more greater than hippurate uptake, chances of recovery were greater, as compared to those who had no significant difference in the uptake of two radionuclides.

4. *Renal biopsy*

Has not been found useful.

5. *Temporary nephrostomy*
   Temporary nephrostomy and measurement of renal functions after this, is the only certain way to predict recovery[78,81]

Pattern of recovery after relief of obstruction

A precise knowledge of the changes to be expected during functional recovery in the post-obstructed kidney would greatly assist a urologist and nephrologist in managing patients with obstructive renal failure.

A) Changes in tubular function after relief of obstruction

Jones et al.[81] have studied the handling of various solutes and water after relief of partial bilateral obstruction. During obstruction absolute urinary volume, sodium, potassium, phosphate, urate and urea were appropriate for the reduced level of GFR. Fractional excretion of potassium was lower than would be expected for the level of GFR. On the first day following relief of obstruction a significant increase was observed in the absolute and fractional excretion of urinary volume and sodium indicating an alteration in the tubular handling of these urinary constituents. Values then fell so that by 2 weeks there was a significant reduction from day 1 level. Between 2 weeks and 3 months no significant change in absolute excretion occurred, but fractional excretion was reduced, reflecting improvement of GFR over this period. These marked changes were accompanied by return to normal of blood pressure and reversal of other clinical signs of salt and water retention. Similar changes have been described by Jones et al[48].

The absolute and fractional excretion of phosphate and urate followed the same pattern, as sodium and water excretion suggesting that these changes occurred as a result of altered electrolyte handling in the proximal tubule. It has also been shown that increased sodium and water excretion may also be due to defective distal nephron segments[82].

The excretion of potassium did not follow the same pattern, no significant changes were noted in either absolute or fractional excretion at 2 weeks and 3 months, however fractional excretion was once again lower than would be expected for the level of GFR, similar to the situation during obstruction.

B) Changes in glomerular function

Jones et al.[81] also studied glomerular functions. During obstructed phase, ^{99m}TC-DTPA and iohexol clearance as measure of GFR were reduced[66]. No significant improvement occurred by 2 weeks, following relief of obstruction, but was evident by 3 months.This study suggests that functional recovery from obstructive nephropathy occurs in 2 phases. During first two weeks marked changes in tubular handling of water and electrolyte take place. Important to remember is that no change occurs in the potassium excretion, rather fractional potassium excretion decreases. This may be the cause of fatal hyperkalemia, that has been reported even during diuresis[83].

Interesting to note is the comparison of GFR measured by creatinine clearance and GFR measured with radionucleides. This can be explained by the phenomena of pyelovenous and pyelolymphatic back flow. Early fall in serum creatinine when GFR measured with radionucleide was unchanged may once again be explained on the same basis. In addition tubular secretion might have played a role in this initial tubular recovery phase.

Post-obstructive diuresis (POD)

Due to the defective solute and water reabsorption, diuresis and natriuresis occurs. This is especially marked in BUO or UUO of solitary kidney[76,25,84].

This a physiological phenomenon to get rid of fluid retained during obstruction, but if excessive, may lead to excess loss of salt and water. There may also be excessive loss of phosphate and magnesium. Decreased reabsorptive capacity of ascending limb of Henle's loop has been suggested as the explanation for increased excretion of magnesium.

TREATMENT OF OBSTRUCTIVE NEPHROPATHY

Following are the main principles guiding the treatment of urinary tract obstruction.

1. Elimination of any life threatening aspect of the disorder; This will include treatment of Gram negative septicemia, papillary necrosis associated with acute pyelonephritis and

obstruction, acute or acute on chronic renal failure with its complications like hyperkalemia, metabolic acidosis, encephalopathy or pulmonary edema and pericarditis, electrolyte imbalance and volume depletion associated with postobstructive diuresis or chronic partial obstruction with inadequate fluid intake.

2. Attempts should be made to preserve renal function. Surgical intervention that will decrease elevated intrarenal pressure or correct progressive anatomical abnormality is essential. Meticulous management of urinary tract infection is also essential. Treatment of other complications, such as renal calculi or hypertension, and detection of recurrent obstruction by careful follow-up, may also contribute to preservation of renal function.
3. The cause of obstruction should be determined and specific treatment provided when necessary.

Long term follow-up

Patients who have had surgical treatment for obstruction or who have chronic obstruction require careful long term follow-up by a physician. Such follow-up involves careful clinical assessment, urinalysis and urine culture, periodic radiological evaluation, and most important an assessment of renal function.

Importantly, obstructive nephropathy is a disease that can be effectively treated by simply removing the obstructing lesion. Thereafter, depending upon various factors, renal function may improve or remain stable, and the need for some form of dialytic therapy may be postponed, or eliminated.

REFERENCES

1. **Campbell MR.** *Urinary obstruction.* In: **ed. M.F. Campbell and G.H. Harrison.** Urology 3rd ed. Philadelphia Saunders 1970. pp. 1772.
2. **Klahr S.** New insights into the consequences and mechanisms of renal impairment in obstructive nephropathy. *Am J Kid Dis 1991;* **XVIII:** 689-99.
3. **Wilson DR.** Pathophysiology of obstructive nephropathy. *Kindey Int 1980;* **18:** 281-92.
4. **O'reilly PH.** Idiopathic hydronephrosis: diagnosis, management and outcome. *Br J Urol 1989;* **63:** 569-74.
5. **Gordon I, Mialdea-Fernandez RM, Peters AM.** Perlviureteric junction obstruction. The value of post-micturation view in ^{99m}technetium-DPA diuresis renograpy. *Br J Urol 1988;* **61:** 409-12.
6. **Ryan PC, Maher K, Hurley GD, Fitzpatrick JM.** The whitaker test: experimental analysis in the canine model of partial ureteric obstruction. *J Urol 1989;* **141:** 387-90.
7. **Platt JF.** Duplex doppler evaluation of native kidney dysfunction: obstructive and non-obstructive disease. *AJR 1992;* **158:** 1035-42.
8. **Morrison AR, benabe JE.** prostaglandins and vascular tone in experimental obstructive nephropathy. *Kidney Int 1981;* **19:** 786-90.
9. **Kekomaki M, Vapaatalo, H.** *J Urol 1989;* **141:** 395-97.
10. **Yanagisawa H, Morrissey J, Morrison AR, Klahr S.** Eicosanoid production by isolated glomeruli of rats with unilateral ureteral obstruction. *Kidney Int 1990;* **37:** 1528-35.
11. **Canton AD, Corradi A, Stanziale R, Maruccio G, Migone L.** Glomerular hemodynamics before and after release of 24 hours bilateral ureteral obstruction. *Kindey Int 1980;* **17:** 491-96.
12. **Purkerson ML, Klahr S.** Prior inhibition of vasoconstrictors normalize GFR in postobstructed kidneys. *Kindey Int 1989;* **35:** 1306-24.
13. **Balint P, Laszlo K.** Effect of imidazole and indumethacin on hemodynamics of the obstructed canine kidney. *Kindey Int 1985;* **24:** 892-97.
14. **Chevalier RL, Peach MJ.** Hemodynamic effects of enalapril on neonatal chronic partial ureteral obstruction. *Kindey Int 1985;* **28:** 891-98.
15. **Gasparich JP, Mayo ME.** Comparative effects of four prostaglandin synthesis inhibitors on the obstructed kidney in the dog. *J Urol 1986;* **135:** 1088-90.
16. **Pettersson BA, Aperia A, Elinder G.** Pathophysiological changes in rat kidneys with partial ureteral obstruction since infancy. *Kindey Int 1984;* **26:** 122-27.
17. **Wilson DR.** Pathophysiology of obstructive nephropathy. *Kidney Int 1980;* **18**: 281-92.
18. **Rays AA, Martin D, Settle S, Khalr S.** EDRF role in renal function and blood pressure of normal rats and rats with obstructive uropathy. *Kidney Int 1992;* **41:** 403-13.
19. **Gerber IJ, Olson RD, Nies AS.** Inter relationship between prostaglandins and renin release. *Kidney Int 1981;* **19:** 816-21.
20. **Harris KPG, Schreiner GF, Klahr S.** Effect of leukocyte depletion on the function of the post-obstructed kidney in the rat. *Kindey Int 1989;* **36:** 210-15.
21. **Morsing P, Stenberg A, Persson AEG.** Effect of thromboxane inhibition on tubuloglomerular feed back in hydronephrotic kidneys. *Kidney Int 1989;* **36:** 447-52.
22. **Wahlberg J, Stenberg A, Wilson Dr, Persson AEG.** Tubuloglomerular feedback and interstitial pressure in obstructive nephrology. *Kidney Int 1984;* **26**: 294-302.
23. **Rector FC, Jr.** Obstructive nephropathy, . In: **Wyngaarden JB, Smith LH, Jr. (eds.)** Cecil text book of medicine. 18th edition, W.B. Saunders Company, 1988: 614-17.
24. **Brunskill N, Hayes C, Morrissey J, Klahr S.** Changes in lipid environment decrease Na-K-ATPase activity in obstructive nephropathy. *Kindey Int 1991;* **39:** 843-49.
25. **Bishop MC.** Diuresis and renal functional recovery in chronic retention. *Br J Urol 1985;* **57:** 1-5.
26. **Gillenwater JY, Westervelt FB, Jr. Vaughan ED, Jr. Howards SS.** Renal function after release of chronic unilateral, hydronephrosis in man. *Kindey Int 1975;* **7:** 179-86.
27. **Batlle DC, Arruda JAL, Kurtzman NA.** Hyperkalemic distal renal tubular acidosis associated with obsturctive nephropathy. *N Engl J Med 1981;* **304:** 373-79.
28. **Huland H, Gonnermann D, Werner B, Dossin U.** A new test to predict reversibility of hydronephrotic atrophy after stable partial. Unilateral ureteral obstruction. *J Urol 1988;* **140:** 1591-94.

29. **Bulger RE, Hebert SC.** Stucture functional relationship in the kidney. In: **Schrier RW, and Gottschalk CW (eds).** *Diseases of the kidney,* 1988; pp. 3-64.
30. **Hostetter TH, Rennke HG, Brenner BM.** Compensory renal hemodynamic injury: A final common pathway of residual nephron destribution. *Am J Kidney Dis 1982;* **1:** 310-10.
31. **Brenner BM, Meyer TW, Hostetter RH.** Dietary protein intake and the progressive nature of kidney disease: The role of hemodynamically mediated glomerular injury in the pathogenesis of progressive glomerular sclerosis in aging, renal ablation and intrinsic renal disease. *N Engl J Med 1982;* **307:** 652-57.
32. **Zelman SJ, Zenser TV, Davis BB.** Renal growth in response to unilateral ureteral obstruction. *Kidney 1983;* **23:** 594-99.
33. **Humphreys' MH, Lin SY, Wiedemann E.** Renal nerves and the natriuresis following unilateral renal exclusion in the rat. *Kidney Int 1991;* **39:** 63-70.
34. **Siegel NJ, Upadhyaya K, Kashgarian M.** Inhibition by indomethacin of adaptive changes in the contralateral kidney after release of unilateral ureteral occlusion. *Kidney Int 1981;* **20:** 691-94.
35. **Jones DA, George NJR.** Interactive obstructive uropathy in man. *Br J Urol 1992;* **69:** 337-45.
36. **Blok C, Von Venrodij GEPM, Coolsact BLRA.** Dynamics of the ureterovesical junction: effectiveness of its ureteral peristalsis in high pressure pig bladder. *J Urol 1985;* **134:** 825-27.
37. **George NJR, O'reilly PH, Bernard RJ, et al.** High pressure chronic retention. *Br Med J 1983;* **286:** 1780-83.
38. **George NJR, O'reilly PH, Bernard R, et al.** Practical management of patients with dilated upper tracts and chronic retention of urine. *Br J Urol 1984;* **56:** 9-12.
39. **Hackler RH, Hall MK, Zampieri TA.** Bladder hypocompliance in the spinal card injury population. *J Urol 1989;* **141:** 1390-93.
40. **Weston PMJ, Robertson LQ, Williams S, et al.** Poor compliance early in filling in the neuropathic bladder. *Br J Urol 1989;* **63:** 28-31.
41. **McGuire FJ, Woodside JR, Thomas AB.** Upper urinary tract deterioration in patients with myelody splasia and detrusor hypertonia: A follow up study. *J Urol 1983;* **129:** 823-26.
42. **Mundy AR, Borzyskowski M, Saxon HM.** Videourodynamic evaluation of nephropahtic vesicourethral dysfunction in children. *Br J Urol 1982;* **54:** 645-49.
43. **Turka A.** Urinary tract obstruction, In: **Rose BD, (ed.)** *Pathophysiology of renal disease,* 1987: 447-67.
44. **Klahr S, Buerkert J, Morrison A.** Urinary tract obstruction, In: **Brenner BM, Rector FC, (eds.).** *The kidney,* 3rd edition, Philadelphia, W.B. Saunders Company, 1986: 1443-90.
45. **Ronald AR, Simonsen JN.** Infections of the upper urinary tract. In: **Schrier RW and Gottaschalk CW (eds.).** *Disease of the kidney* 1988: pp. 1065-1108.
46. **Andriole VT.** Renal and perirenal abscesses. In: **Schrier RW, and Gottschalk CW (eds).** *Diseases of the kidney,* 1988; pp. 1049-64.
47. **Drutz DJ, Fetchick RJ.** Fungal infections of the kidney. In: **Schrier RW, and Gottschalk CW (eds).** *Diseases of the kidney,* 1988; pp. 1014-48.
48. **Jones DA, George NJR, O'Reilly PH, et al.** Reversible hypertension associated with unrecognized high pressure chronic retention of urin. *Lancet 1987;* **11:** 1052-54.
49. **Rose BD.** Tubulointerstitial diseases. In: **Rose BD, ed.** *Pathophysiology of renal diseases.* 1987, pp. 387-432.
50. **Rascoff JH, Golden RA, Spinowitz BS, Charytan C.** Non-dilated obstructive nephropathy. *Arch Intern Med 1983;* **143:** 696-99.
51. **Laville M, Maillet PJ, Pelle-Francey D.** Non-dilated obstructive acute renal failure (abstract). *Kidney Int 1985;* **28**: 694.
52. **Wilson DR.** Urinary tract obstruction. In: **Schrier RW, and Gottschalk CW (eds).** *Diseases of the kidney,* 1988; pp. 715-46.
53. **O'Reilly PH.** Diuretic renography, recent advances and recommended protocols. *Br J Urol 1992;* **69:** 113-20.
54. **Brown SCW, Upsdell SM, O'reilly PH.** The importance of renal function in the interpretation of diuresis renography. *Br J Urol 1992;* **69:** 121-25.
55. **Upsdell SM, Leeson SM, Brooman PJC, O'reilly PH.** Diuretic induced urinary flow rates at varying clearance and their relevance to the performance and interpretation of diuresis renography. *Br J Urol 1988;* **61:** 14-18.
56. **Armstrong P, Wastie, ML.** *Diagnostic imaging.* 2nd edition, Blackwell Scientific Publications, 1987; 203-48.
57. **Timoney AG, Payne SR, Davis LAD, Abercrombie GF.** The "plain film" bladder shadow in outflow obstruction: as accurate a discriminant of residual urine as ultrasound. *Br J Urol 1989;* **63:** 363-66.
58. **Sjoberg S, hellstens S, Almen T, Golman K, Gronberg T.** Estimating kidney function during urography-comparison of contrast medium clerance are simultaneous ^{51}Cr-EDTA clearance. *Acta Rad 1987;* **28:** 587-92.
59. **Boijsen M, Tacobsson L, Tylen U.** Renal function measured by x-ray flourescence analysis - a comparison between contrast medium clearance and non-simultaneous ^{51}Cr-EDTA clearance. *Acta Rad 1987;* **28:** 581-85.
60. **Scola FH, Cronan JJ, Schepps B.** Grade I hydronephrosis: pulsed dupper vs evaluation. *Radiology 1989;* **171:** 519-20.
61. **Rosi P, Virgili G, Distasis SM, et al.** Diureteic ultrasound a non-invasive technique for the assessment of upper tract obstruction. *Br J Urol 1990;* **65:** 566-69.
62. **Courtney SP, Weightman JAK.** The value of ultrasound scanning of the upper urinary tract in patients with bladder outlet obstruction. *Br J Urol 1991;* **68:** 169-71.
63. **Cox IH, Erickson SJ, Foley WD, Dewire DM.** Ureteric jets; evaluation of normal flow dynamics with color doppler sonography. *AJR 1992;* **158:** 1051-55.
64. **Platt JF, Rubin JM, Ellis JH, Dipietro MA.** Duplex dopper us of the kidney: differentiation of obstructive from non-obstructive dilatation. *Radiology 1989;* **171:** 515-17.
65. **Goldberg BB, Bagley D, Liu JB, Merton DA, Alexander A, Kurtz AB.** *AJR 1991;* **156:** 99-103.
66. **O'reilly PH, Shields RA, Testa HJ.** Nuclear medicine in urology and nephrology, 1986.
67. **English PJ, Testa HJ, Lawson RS, Carroll RN, Edwards EC.** Modified method of diuresis renography for the assessment of equivocal pelviureteric junction obstruction. *Br J Urol 1987;* **59:** 10-14.
68. **Greenstein A, Chen J, Matzkin H, Baron J, Braff Z.** Potential pitfalls in the obstructive renal scan in patients with double-pigtail ureteral catheters. *J Urol 1989;* **141:** 283-84.
69. **Bahar RH, Saha M, Kouris K, et al.** ^{99m}Technetium-DTPA diuretic renography in the evaluation of surgery in chronic schistosomal and non-schistosomal obstructive uropathy. *Br J Urol 1990;* **60:** 137-43.
70. **Nauta J, Pot DJ, Kodij PPM, Nijman JM, Wolff ED.** Forced hydration prior to renography in children with hydronephrosis. An Evaluation. *Br J Urol 1991;* **68:** 93-7.
71. **Upsdell SM, Testa HJ, Lawson RS.** The F-15 diuresis renogramin suspected obstruction of the upper urinary

tract. *Br J Urol 1992;* **69:** 126-31.
72. **Lupton EW, Testa HJ.** The obstruction diuresis renogram: an appraisal of the significance. *J Urol 1992;* **147:** 9851-83.
73. **Britton KE, Nawaz MK, Whitfield HN, et al.** Obstructive nephropathy: comparison between parenchymal transit time index and frusemide diuresis. *Br J Urol 1987;* **59:** 127-32.
74. **Lewis CA, Coptcoat MJ, Carter S. STC, Hilson AJW, Wickham JEA, Shah PJR.** Radionuclide imaging of ureteric peristalsis. *Br J Urol 1989;* **63:** 144-48.
75. **Kunin M, Goodwin WE.** The encased ureter; bullet and bodkim pattern, a reliable radiographic sign. *Br J Urol 1990;* **66:** 471-74.
76. **Wilson DR.** The influence of chronic unilateral ureteral obstruction. *Kindey Int 1974;* **5:** 402-10.
77. **Bander SJ, Buerkert JE, Martin D, Klahr S.** Long term function in the rat. *Kindey Int 1985;* **28:** 614-20.
78. **Kumar A, Sharma SK, Vaidyanathan S.** Results of surgical reconstruction in patients with renal failure owing to ureteropelvic junction obstruction. *J Urol 1988;* **140:** 484-86.
79. **Jones DA, Gilpin SA, Holden D, Dixon JS, O'reilly PH, George NJR.** Relationship between bladder morphology and long term outcome of treatment in patients with high pressure chronic retention of urine. *Br J Urol 1991;* **67:** 280-85.
80. **Thomsen HS, Hvid-Jacobsen K, Meyhoff HH, Nilsen SL.** Combination of DMSA-scintigraphy and hippuran renography in unilateral obstructive nephropathy - improved prediction of recovery after intervention. *Acta Rad 1987;* **28:** 653-55.
81. **Jones DA, George NJR, O'reilly PH, Bernard RJ.** The biphasic nature of renal functional recovery following relief of chronic obstructive uropathy. *Br J Urol 1988;* **61:** 192-97.
82. **Jones DA, Atherton JC, O'reilly PH, Barnard RJ, George NJR.** Assessment of the nephron segments involved in post-obstructive diuresis in man, using lithium clearance. *Br J Urol 1989;* **64:** 559-63.
83. **O'Reilly PH, Upsdell SM, Brough RJ.** Life threatening hyperkalemia after decompression for high pressure chronic retention. *Lancet 1987;* **1:** 1859.
84. **Clarke NW, Jones DA, Tames SF, Laing I, George NJR.** Disturbance in sodium regulating hormones in chronic obstructive uropathy. *Br J Urol 1991;* **68:** 118-21.

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