INTRODUCTION

Laparoscopic procedures have evolved in the field of pediatric urology over the last 15 years, as visualization and instrumentation have improved. Laparoscopic techniques have now been successfully applied to pyeloplasty, renal ablative procedures, orchidopexy, lower urinary tract reconstruction, ureteral reimplantation, and varicocelectomy.[1]

Children are now able to benefit from the laparoscopic advantages as improved cosmesis and decreased postoperative pain.

Ureteral dilatation, or hydroureter, is a frequent cause of dilatation of the fetal urinary tract.[2] Under normal conditions, on fetal or postnatal US the normal ureter is not visualized, once it is visible, a urinary tract dilatation is present and must be investigated.[3]

Ureteral reimplantation has been described to be the management of choice in children with obstructive megaureter[4] and one of the main modalities in the management of vesicoureteral reflux (VUR) and refluxing megaureter.[5]

As laparoscopy has been rapidly evolved; laparoscopic ureteral reimplantation has become a feasible technique for managing megaureters either obstructive or refluxing.[6]

Embryology and development of ureter and ureterovesical junction (UVJ)

Ureteral development begins during the fourth week of gestation when the ureteral bud arises from the mesonephric duct.[7] The bud elongates cephalad, and forms the ureter, renal pelvis, calyces, and collecting tubules.

The distal end of the mesonephric duct; from the ureteral bud to the vesico-urethral tract, is called the common excretory duct and expands in trumpet fashion into the bladder and urethra to form half of the trigone.

The wolffian duct (early vas deferens) and early ureter can be thought of as forming the two upper arms of a Y with the distal mesonephric duct as the stem of the Y. While budding is occurring, the distal mesonephric duct is being drawn and incorporated into the region of the urogenital sinus, which later becomes the bladder. Incorporation continues until the entire stem is absorbed, leaving the two arms of the Y to enter the bladder separately, one as the ureter and the other as the vas and ejaculatory duct in the male prostatic urethra (or the vestigial Gartner duct in the female vagina). The two arms of the Y also rotate relative to each other once they contact the urogenital sinus(UGS)/ bladder wall resulting in the ureteral opening being proximal to the ejaculatory duct orifice.[8] If the ureteral bud reaches the UGS too soon (believed to be due to early budding), over-rotation draws it high and lateral in the bladder wall, leading to inadequate incorporation, insufficient intramural length in the bladder wall, and reflux.[9] If the ureteral bud reaches the UGS too late (due to late budding), insufficient rotation occurs, resulting in an ectopic ureter that is drawn distally and medially, often obstructing in the bladder neck region or elsewhere. Furthermore, early or late budding is also thought to mistarget the contact between bud epithelium and the metanephros, leading to renal malformations, dysplasia, hypoplasia, or even agenesis.[8]

Anatomy of lower ureter and ureterovesical junction (UVJ)

The ureter is divided anatomically into two major components—abdominal and pelvic—with lengths ranging from 12 to 15 cm each. The pelvic ureter descends over the pelvic brim as it courses over the iliac vessels at the bifurcation of the common iliac artery into the external and internal iliac arteries.(10)

The pelvic portion of the ureter, immediately after crossing the iliac vessels, runs backwards and laterally in a vertical course along the side wall of the true pelvis, just lateral and anterior to the internal iliac artery till the level of the ischial spine. Then it runs forward and medially towards the bladder in a horizontal course, just medial to the obturator nerve and superior vesical artery. (Figure 1)

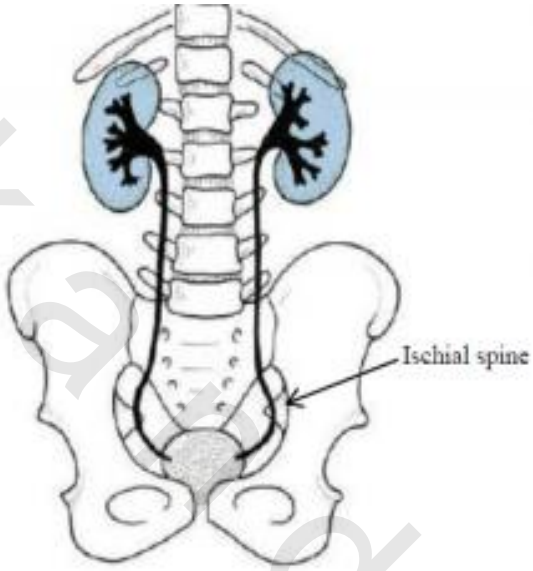


Figure (1): Course of the ureter.(11)

The anatomical relationships differ between male and female subjects for both the vertical and horizontal portions.(12)

The female pelvic ureter:

The relations of the female pelvic ureter are complex (figure 2). In its vertical portion, the ureter is dorsal to the ovary, medial to the ovarian vessels, and lateral to the sacrouterine ligament. The horizontal portion is located inside the broad ligament and runs close to the uterine artery**,** which goes obliquely forward.

After crossing the broad ligament, the ureter lies lateral to the uterine cervix and above the lateral fornix of the vagina. During its course, the ureter is accompanied by the vaginal artery, venous plexus, and lymphatics.(12)

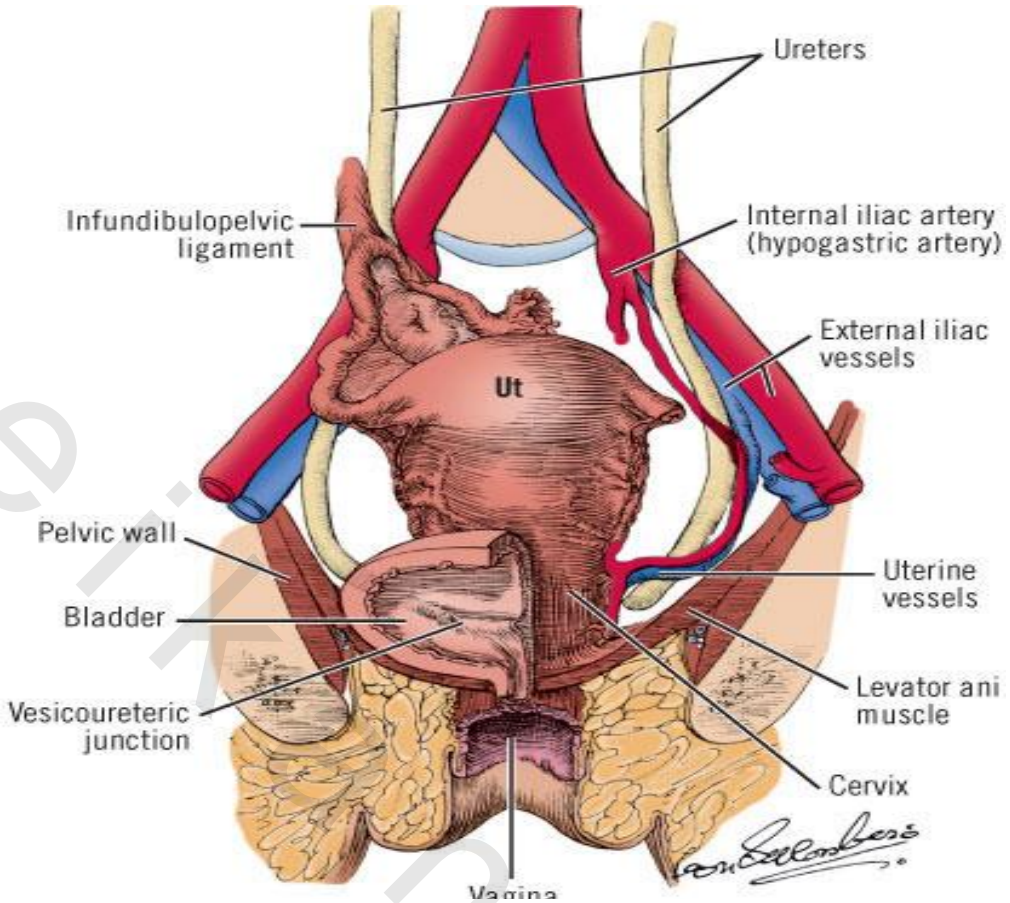


Figure (2): Pelvic relations of the ureter in females.[13]

The male pelvic ureter:

In its vertical portion, the male pelvic ureter is located lateral to the rectum and is crossed ventrally by the vas deferens at the level of ischial spine. In its horizontal portion, the ureter turns medially and enters the posterolateral wall of the bladder, lateral to the seminal vesicles. The vesicoprostatic arteries cross below the ureter.[12] (Figure 3)

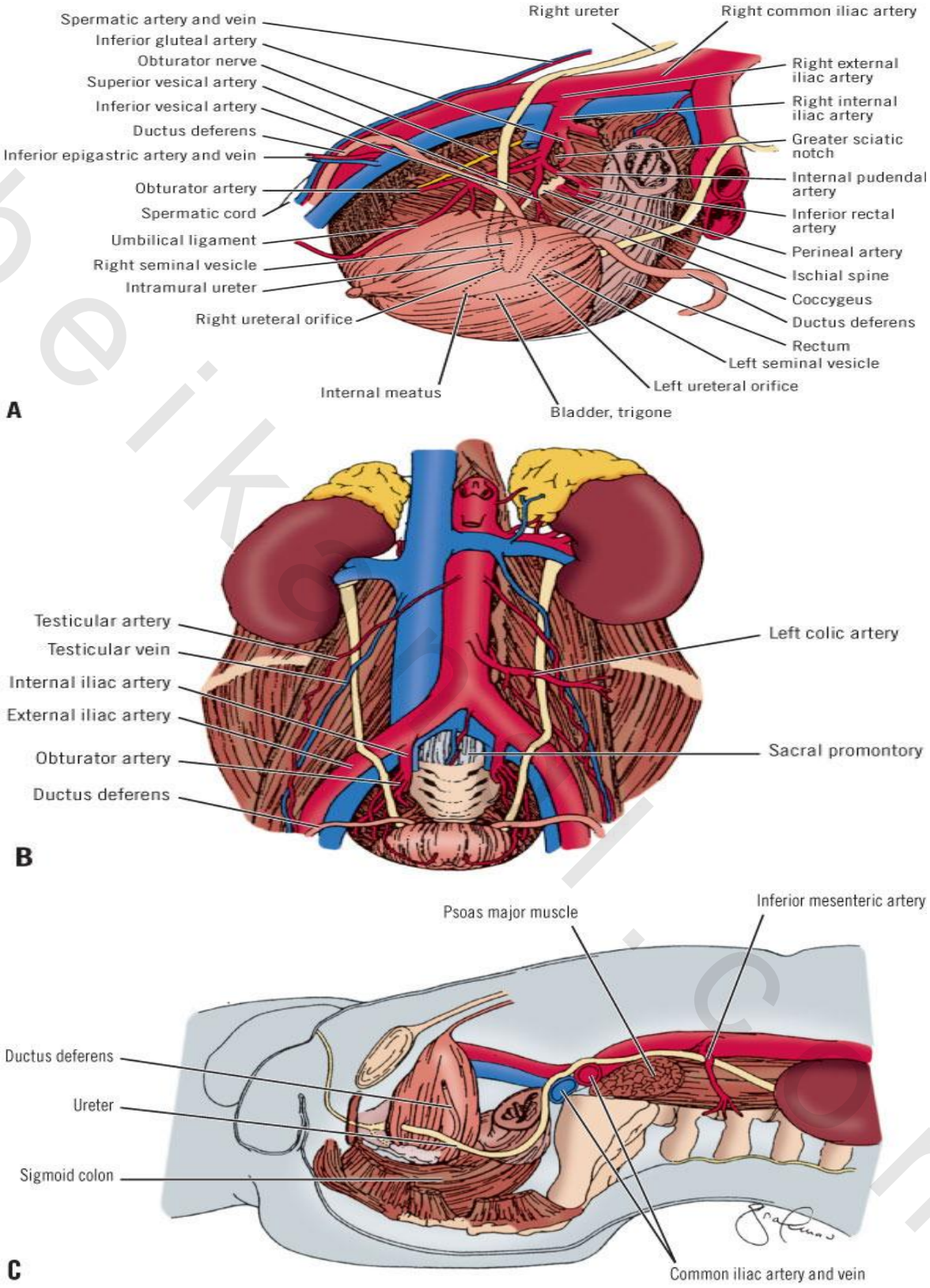


Figure (3): Pelvic relations of the ureter in males. **A)** Oblique view. **B)** Coronal view. **C)** Lateral view.[14]

The uretero-vesical junction (UVJ):

As the ureter approaches the bladder, its spirally oriented mural smooth muscle fibers become longitudinal. Two to 3 cm from the bladder, a fibromuscular sheath (of Waldeyer) extends longitudinally over the ureter and follows it to the trigone. The ureter pierces the bladder wall obliquely, travels 1.5 to 2 cm, and terminates at the ureteral orifice (Figure 4). The intravesical portion of the ureter lies immediately beneath the bladder urothelium and therefore is quite pliant; it is backed by a strong plate of detrusor muscle. With bladder filling, this arrangement is thought to result in passive occlusion of the ureter, like a flap valve.[15]

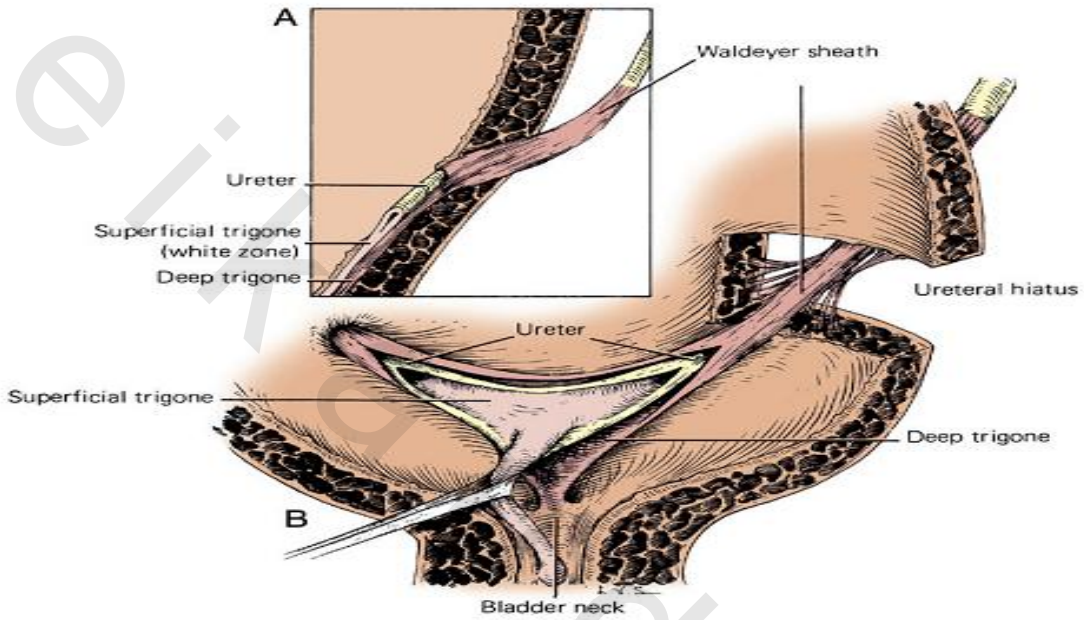


Figure (4): Normal ureterovesical junction and trigone.**A)** Section of the bladder wall perpendicular to the ureteral hiatus shows the oblique passage of the ureter through the detrusor and also shows the submucosal ureter with its detrusor backing. Waldeyer sheath surrounds the prevesical ureter and extends inward to become the deep trigone. **B)** Waldeyer sheath continues in the bladder as the deep trigone, which is fixed at the bladder neck. Smooth muscle of the ureter forms the superficial trigone and is anchored at the verumontanum.[15]

Laparoscopic anatomy:

There is a difference between conventional and laparoscopic surgical anatomy just as there is between surgical and gross anatomy. Surgical anatomy is always concerned only with structures that are surgically important, but what is seen when the pelvis is looked at through the laparoscope is not always the same as what is seen through an abdominal incision .The umbilical ligaments; for example, are easily visible laparoscopically whereas they cannot be seen through a Pfannenstiel incision. These ligaments, therefore, have little surgical significance during laparotomy, but are key landmarks for the laparoscopic dissection of the retroperitoneum.[16]

The key laparoscopic land mark for dissecting the retroperitoneum and hence the ureter is the pelvic sidewall triangle. In females; the base of this triangle is formed by the round ligament, the lateral border by the external iliac artery, the medial border by the infundibulo-pelvic ligament, and the apex at the point where the infundibulo-pelvic ligament crosses the external iliac artery.[16](Figure 5)

In males; the base of the triangle is formed by the vas deferens, the lateral border by the external iliac vein and the medial border by the medial umbilical ligament.[17](Figure6)

Understanding of the anatomical relationships at the apex of the pelvic sidewall triangle at the pelvic brim is critical to dissection of the ureter.

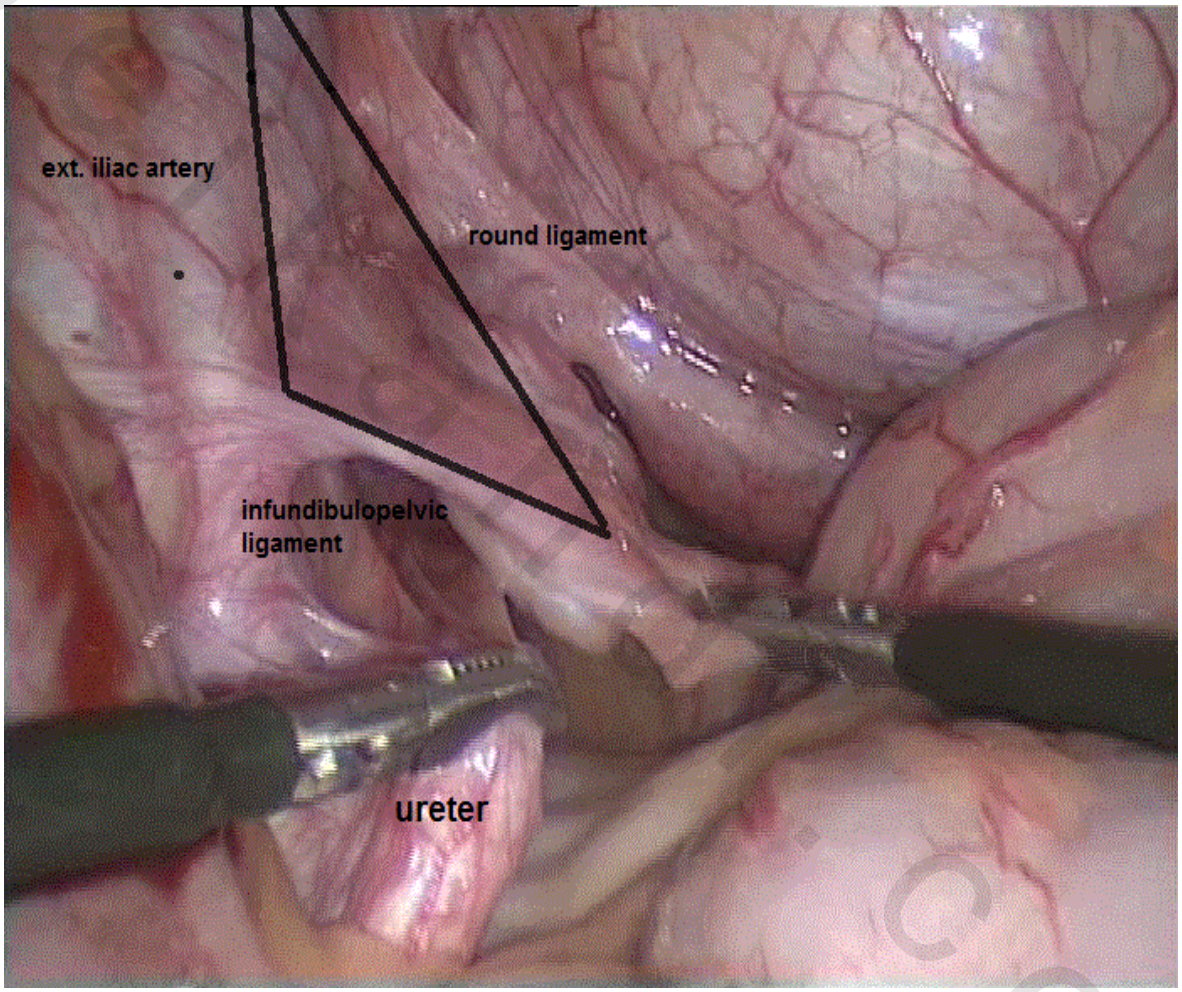


Figure (5): Pelvic side wall triangle in a female pelvis.

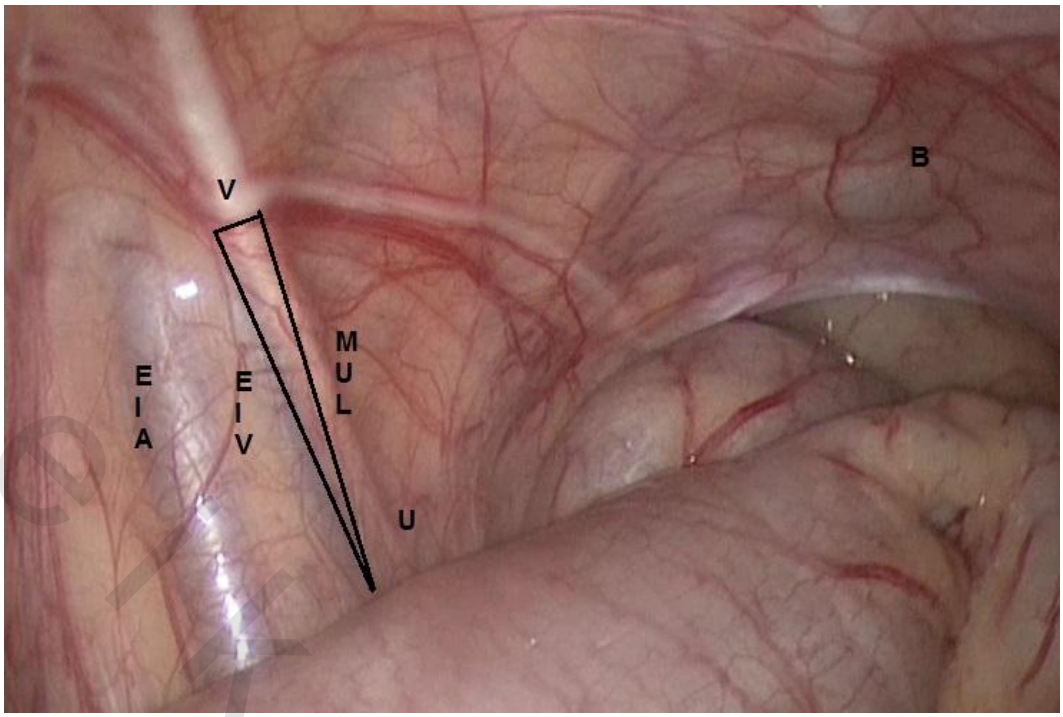


Figure (6): Pelvic sidewall triangle in a male composed of vas deferens (V) anteriorly, of medial umbilical ligament (MUL) medially and of medial aspect of external iliac vein (EIV) laterally. EIA = external iliac artery, B = bladder, U = ureter.

Functional Anatomy of the Anti-reflux Mechanism

The anti-reflux mechanism represents a balance between several factors. Abnormality in any of these factors alone or in combination will allow or cause the retrograde flow of urine from the bladder up the ureter and ultimately to the renal pelvis and tubules. These factors include the functional integrity of the ureter, the anatomic composition of the ureterovesical junction (UVJ), and the functional dynamics of the bladder. First, for purposes of reflux prevention, the ureter represents a dynamic conduit, which adequately propels the urine presented to it in a bolus fashion; antegrade, by neuromuscular propagation of peristaltic activity and so reflux is actively opposed. Moreover, if reflux were to occur; depending on its degree and timing, antegrade flow might be expected to keep refluxing urine from reaching the renal pelvis. The second component is the anatomic design of the UVJ, at the heart of this unique mechanism lies the intramural portion of ureter that travels within the detrusor muscle as it traverses the bladder wall. (Figure7)The intramural ureter remains passively compressed by the bladder wall during bladder filling, preventing urine from entering the ureter. Adequate intramural length and fixation of the ureter between its extravesical and intravesical points is required to create this antirefluxing compression valve.[8]As regard the third component, alterations in functional dynamics of the bladder "voiding dysfunction" can affect ureterotrigonal structure and result in vesicoureteral reflux (VUR). Increased intravesical pressure causes a spectrum of intravesical anatomical distortions that predispose to VUR.[18] There is a theory that reflux is a secondary phenomenon, appearing as a result of detrusor over activity rather than as a primary ureterovesical junction abnormality.[19]

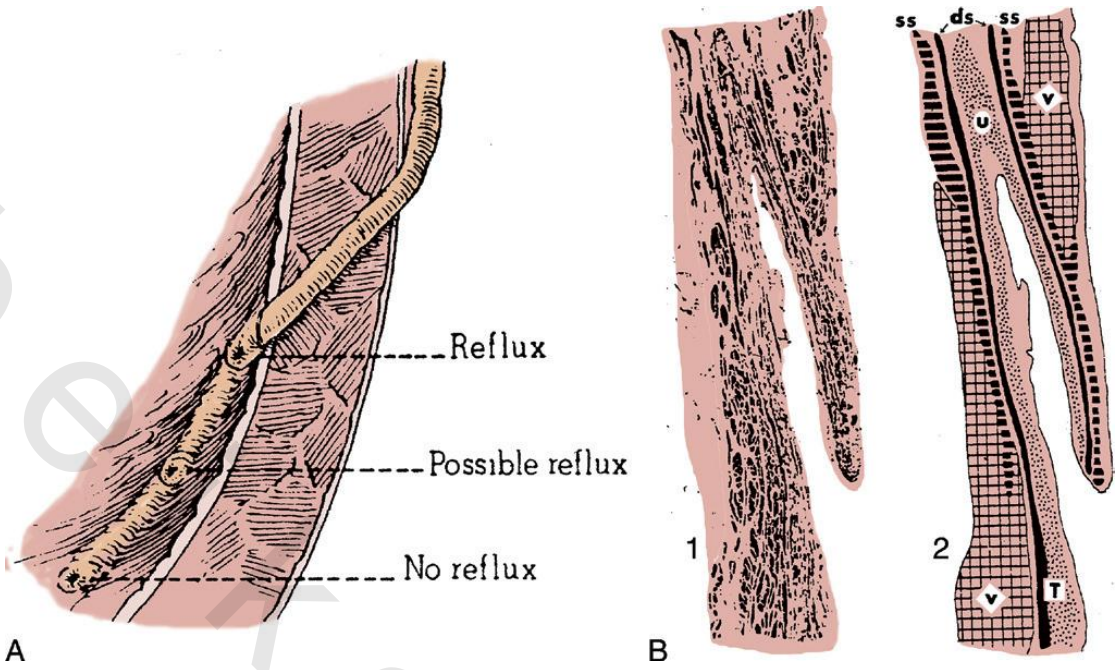


Figure (7): Functional anatomy of vesico ureteral junction.

A) A refluxing ureterovesical junction has the same anatomic features as a non-refluxing orifice, except for inadequate length of the intravesical submucosal ureter. Some orifices with marginal submucosal tunnels may reflux intermittently.(20) **B)** Ureterovesical junction in longitudinal section. 1, Photomicrograph; 2, diagrammatic representation. The ureteral muscularis (u) is surrounded by superficial (ss) and deep (ds) periureteral sheaths that extend in the roof of the submucosal segment and continue beyond the orifice into the trigonal muscle (t). The relationship of the superficial sheath to the vesical muscularis (v) is clearly seen. Transverse fascicles in the superior lip of the ureteral orifice belong to the superficial and deep sheaths. No true space separates ureter from bladder.(21)

Megaureter

History

In 1923, Caulk described a patient with distal ureteral dilatation without evidence of hydronephrosis and coined the term megaloureter. Thirty years later, Swenson postulated a neurologic etiology for megaureter and treated such patients with urinary diversion, ureteral substitution, and ileal augmentation to provide peristalsis. Clinical management of megaurete evolved over the next two decades. Stephens, Nesbitt, and Withycombe advised observation and double voiding to reduce the incidence of urinary tract infections (UTIs).(22)

Definition and classification

The term "megaureter" is simply a descriptive term about the diameter of the ureteral lumen. Retrovesical ureteric diameter ≥7 mm from30 weeks' gestation onwards is abnormal[4]and can be defined as megaureter. Smith classified megaureters into four categories, obstructive, refluxing, refluxing with obstruction, and non-refluxing/non-obstructed[23], later subdivided into primary and secondary by King.[24] The Pfistere Hendren classification established in 1978 was based on the morphological appearance: type I involved the distal ureter without associated hydronephrosis; type II extended to both ureter and pelvis; and type III was associated with severe hydroureteronephrosis and ureteric tortuosity.[25] It is important to distinguish between cases of primary or secondary causes of the pathology, as in the case of secondary causes, the treatment is directed at the initiating pathology and not at the ureter.[26]

Prevalence:

Primary obstructive megaureter:

Obstructive megaureter represents 23% of children with urinary tract obstruction.[26] The diagnosis is more common in boys than girls, and is more often on the left side. It can be bilateral in 25% of cases. There is no clear genetic pattern of inheritance, although some cases do appear to run in families.[26]

Most cases of megaureter are now first detected with prenatal ultrasound and then diagnosed afterbirth. Some cases present clinically during childhood with abdominal pain, hematuria, and/or urinary tract infections. Megaureter may also be incidentally discovered later in life on imaging studies. It rarely leads to renal insufficiency.[26]

Primary vesicoureteral reflux "VUR" and refluxing megaureter:

Since VUR can be asymptomatic and lower grades often spontaneously resolve, it is challenging to calculate a true prevalence rate .Older estimates of VUR prevalence among asymptomatic children have cited a rate of 0.4% - 1.8%.[27]

More recently, Sargent completed a review of studies of children undergoing voiding cystourethrography(VCUG) for various indications, estimating the prevalence of reflux in normal children without any urologic history at 0.9%, and as high as 30% for those presenting with a history of a urinary tract infection (UTI).[27]

Irrespective of the exact prevalence of VUR in children as a whole, there are distinct sex and race differences. Early in life, boys are more likely to have VUR; however, the majority of persistent reflux in older children is found in girls. A retrospective review of 15, 504 children by Chand et al reported a female-to-male ratio of reflux of 2:1.[28]

Additionally, they found that white children were three times more likely to be diagnosed with VUR than black children. Accordingly, VUR certainly has a genetic component, but the precise mode of inheritance remains unknown. Currently, researchers hypothesize that VUR is inherited in an autosomal dominant fashion with variable penetrance.[29]

Pathophysiology

The smooth muscle differentiation in the distal ureter may be the key to understand why reflux or obstruction develops in utero, persists in the newborn, and often subsides later in childhood. Smooth muscle bundles of the extramural ureter first appear in weeks 17-22 of gestation, and ureteral muscle development proceeds in a craniocaudal direction.[30] The distal ureter is the last portion to develop its muscular coat, and early muscular differentiation is primarily of the circular muscles. The process whereby the circular muscle pattern, which is typical of the fetal ureter, changes progressively into the double muscle layers of the full-term infant, may last up to 2 years, and may explain the transient functional disturbances of the UVJ during this period.[31]

Primary Obstructive Megaureter:

There is a general agreement that there is no true narrowing at the UVJ in most of cases, but a functional obstruction arising from an aperistaltic juxtavesical (adynamic) segment 0.5 – 4 cm long that is unable to transport urine at acceptable rates.[32](Figure8)

Other than the adynamic segment mentioned above, other anatomic causes can lead to a similar clinical scenario. Both congenital distal ureteral strictures and distal ureteral valves can be almost indistinguishable from the classic obstructive megaureter.[8]

This distal segment has been examined histologically and has been found to contain increased levels of collagen type I and III (predominantly type I). It is this increased fibrosis that is implicated in the disruption of intercellular communications and leads to uretero-arrhythmias and obstruction.[33]

Figure (8): Obstructive megaureter with distal aperistaltic segment.[34]

There are many other theories regarding the development of obstructive megaureters:

a- Atrophy of the inner longitudinal muscles in these ureteral segments(the longitudinal muscles are those that transmit peristalsis) and hypertrophy of outer, compressive circular muscle, leading to obstruction.[23]
b- A maturational cause of obstructive megaureters, as many obstructive megaureters resolve and develop into normal collecting systems over time signifies that perhaps the renal urine production began slightly prematurely, before the ureter is fully canalized at its caudal end, leading to hydroureter. The full canalization of the mature ureter could then explain their solution of the obstructive appearance of the ureter.[26]
c- Developmental evolution of the distal ureter from a single, circular muscle layer to the double layer (circular and longitudinal) of the child, represents another maturational theory.[26]

Other histologic findings claiming to display the causative aspect of the obstructive megaureter include distal ureteral segments with no muscle tissue present, but simply a fibrotic, static terminal end.[32]

Yet others have documented distal ureteral segments with thick sleeve of muscle forming a continuous layer surrounding the muscle bundles of the terminal ureter. The muscle forming this outer layer is distinct in its histological appearance and arrangement from the muscle bundles which form the ureteric muscle of the UVJ and is excessively not responsive to any adrenergic stimulus, leading to almost tonic contraction.[32]

Interestingly, the proximal, dilated ureteral segment has also been found to be composed of altered connective tissue, and this fibrosis and the dilation itself can lead to uretero arrhythmias and poor peristaltic wave transmission.[35]

It is important to note that the upper tract dilation (while appearing to be a significant pathology itself) does play an important role in the urinary tract response to the presence of obstruction. The infant collecting system is more pliable than in more mature patients and this dilation allows for the dampening of pressure, allowing the kidneys to produce urine into a collecting system in pressures as close to physiologic pressures.[26]

Primary vesicoureteral reflux "VUR" and refluxing Megaureter

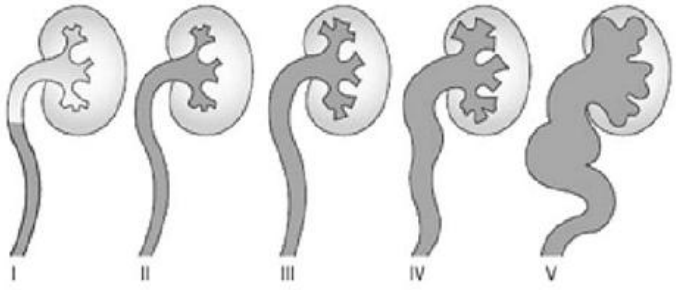
Refluxing megaureter simply represents a refluxing ureter that happens to be dilated.[26] The pathology of vesicoureteral reflux involves a congenital defect in the structure and therefore functions of the UVJ. Reflux occurs despite an adequately low pressure urine storage profile in the bladder. The length-to-diameter ratio of the intramural ureteral tunnel is almost always less than approximately 5: 1 ratio of tunnel length to ureteral diameter in non-refluxing junctions compared with a 1.4: 1 ratio in refluxing UVJs.[36] (Table 1)

They may be associated with abnormalities of the UVJ, making reflux more likely, such as periureteral diverticulae.[8]

The distal segment of refluxing megaureters also shows histologic derangement with increased fibrosis (much like the obstructive megaureters); however, in these cases, the predominant collagen is collagen type III.[33]

Grading of VUR

Vesicoureteral reflux is commonly graded according to the international study classification.[37] (Figure 9)



Grade	Description
I	Into a nondilated ureter
II	Into the pelvis and calyces without dilatation
III	Mild to moderate dilatation of the ureter, renal pelvis, and calyces with minimal blunting of the fornices
IV	Moderate ureteral tortuosity and dilatation of the pelvis and calyces
V	Gross dilatation of the ureter, pelvis, and calyces; loss of papillary impressions; and ureteral tortuosity

Figure (9): Grading of vesicoureteral reflux "VUR"[38]

Table (I): Mean ureteral tunnel length and diameter in normal children.[36]

Age (yr)	Intravesical Ureteral Length (mm)	Submucosal Ureteral Length (mm)	Ureteral Diameter at the Ureteroveical Junction (mm)
1 - 3	7	3	1.4
3 - 6	7	3	1.7
6 - 9	9	4	2.0
9 - 12	12	6	1.9

Management

It is well-known that the majority of congenital megaureters (either refluxing or obstructive) may be managed conservatively[35], and fortunately, up to 85% of prenatally detected cases are asymptomatic and may simply be observed.[39]

Primary obstructive Megaureter:

In cases of possibly obstructive megaureters, the decision to intervene surgically is a difficult one. Even in cases of obvious obstruction, early surgical intervention is fraught with a higher complication rate. The basic rule that should be followed is that no surgery should be performed as long as renal function is not significantly affected and urinary tract infections are not a major issue.[26] Instead, antibiotic suppression with close observation is

all that is required. Typically, surgical repair is warranted between 1and 2 years of age if an initial differential renal function (DRF)< 40%, especially when associated with massive hydroureteronephrosis, or failure of conservative management (breakthrough febrile UTIs, pain, worsening dilatation, or deteriorating DRF).[4]

In certain rare cases, early intervention is necessary. In order to prevent the complications, but the reimplantation of a grossly dilated ureter into a small infantile bladder could be a challenging operation in babies below 1 year of age, and a number of alternative temporizing or definitive options are available which include:[4]

- Temporary double-J stenting.
- Endoscopic balloon dilatation.
- Endoureterotomy.
- Cutaneous ureterostomy.
- Refluxing ureteral reimplantation.

In terms of forming algorithms to decide which patient will require surgery, no good parameters dictate patients who will resolve and those who will worsen. In general, over 70% of cases resolve over 2 years of follow-up. While there is no correlation with any definable factors (such as degree of hydronephrosis) with regard to which children will require surgery and which will not, there is a correlation of age of resolution and grade of dilation in infants.[40]

Primary VUR and refluxing Megaureter

All urologists are familiar with the standard treatment of reflux and the treatment of primary refluxing megaureter is no different. Initially, even with severe dilation and high-grade reflux, medical management (antibiotic prophylaxis) and observation are all that is necessary.[26]Surgical intervention; including either endoscopic injection, open or laparoscopic reimplantation, is recommended in the following conditions:[41]

- Persistent high grade reflux.
- Renal scaring.
- Recurrent pyelonephritis.
- Breakthrough febrile UTI while on continuous antibiotic prophylaxis (CAP).
- Parental preference.

Laparoscopy in pediatric urological surgeries

History:

Since the introduction of diagnostic laparoscopy for the evaluation of impalpable testes[(42)], laparoscopic urological surgery in children has developed steadily from a simple diagnostic maneuver to an integral part of complex reconstructive procedures. Despite this development; laparoscopy as a method for treating urological problems has been slower to be adopted in children than in adults. This might be attributed to the excellent success rates with traditional open procedures, the initial longer operating times with greater difficulty in learning, and increased hospital costs with no documented measurable differences in recovery time.[(43)]

With establishment of procedures, progress was made on several fronts including operative time, safety and decreasing age of the patient .This progress was also supported by improvements in technology, with smaller cannulas for smaller instruments, better tissue-handling devices such as smaller clip appliers and the harmonic scalpel, all of which are currently available in 5 mm sizes or even smaller 3 mm instruments and adaptable to any infant.[(44)]

Laparoscopy in children and adolescents shares many similarities to adult procedures, but experience with adult surgery is not sufficiently translated to safe surgery in pediatric patients. Pediatric procedures must be performed with understanding of the relevant anatomic and physiologic differences between the pediatric and adult populations.

Differences in instruments:

Many hospitals have created size-specific laparoscopic instrument kits for laparoscopic procedures (table 2). These sets vary in both the diameter and length of the instruments, and they are used according to the age and weight of the patient.[(45)]

Advances in digital imaging and the quality of laparoscopes have improved screen images; this provided a good quality video image while still allowing the surgeon to use the smallest laparoscope available.

Laparoscopes used in pediatric surgery vary in diameter (e.g., 3 mm, 5 mm) and are available with either a straight or angled lens. For pediatric laparoscopic procedures, it is difficult to use a 10-mm laparoscope.[(45)]

Constant research and development need to be conducted in the field of laparoscopic instrumentation with particular attention paid to the design and production of smaller instruments specific for pediatric use.

Table (II): Size specific laparoscopic instruments kits.[45]

LAPAROSCOPY KITS	
Infant laparoscopy kit	3 mm x 20 cm instruments
Pediatric laparoscopy kit	3 mm x 30 cm instruments
Adolescent laparoscopy kit	5 mm x 30 cm instruments
Adult laparoscopy kit	5 mm x 36 cm instruments

Differences in technique:

Achieving safe peritoneal access is a major concern in pediatric laparoscopic procedures. The thin and pliable abdominal wall in infants and children make injury to organs or vessels more likely than with adults. Entering the abdominal cavity can be achieved by using closed or open technique; where the open technique is being preferred as it gives more control when entering the abdomen.[46]

Safe intra-abdominal pressures in pediatric patients have not been well defined, nor have any standards been set for insufflation of the peritoneal cavity of pediatric patients.

Veyckemans states that insufflation pressures should be kept as low as possible and monitored by the anesthesiologist.[47]

The advantages of low insufflation pressures are reduced respiratory and hemodynamic consequences of the pneumoperitoneum, easier control of CO2 absorption by increasing alveolar ventilation, and a reduced risk of mortality in case of accidental CO2 embolism.

Insufflation is started slowly, and the flow is progressively increased up to when the desired intra-abdominal pressure (IAP) or good working conditions are obtained.[47]

Insufflation pressures vary in neonates, infants, and children. The volume of gas introduced affects the pressure created within the abdomen, but this is not the only factor; patient size, abdominal wall distensibility and degree of muscle relaxation also play important roles in determining intra-abdominal pressure. Therefore, it is preferable to set the pressure limits on pressure-regulated automatic insufflators. For infants, the pressure is set at 6 to 8 mmHg. For children, most procedures can be accomplished using pressures of 8 to 10 mmHg, with an increase to 12 to 15 mmHg for older, larger children. These pressures are only general guidelines, because the patient's medical condition may dictate a lower pressure.[46]

According to Beebe et al, "The surgeon selects the pressure desired in the abdomen for the surgical exposure as well as the maximal flow rate for insufflation".[48]

Complications in pediatric laparoscopy

Laparoscopic urological procedures in children have an acceptable rate of complications. In different series the complication rate was 1–2.7% (when excluding pre-peritoneal insufflation and subcutaneous emphysema as a complication).[49]Passerotti et al. reported lower complication rates when an open technique was used than for the Veress technique (0.8% and 2.3%, respectively, $P = 0.006$).[50]

To address concerns that there will be more complications as the level of complexity and number of surgeons using laparoscopic procedures increases, Farhatet al. showed that mentored laparoscopic teaching is a safe way to introduce advanced laparoscopic procedures to inexperienced residents or physicians.[51]

Laparoscopic ureteral reimplantation

When surgical intervention is indicated, Open ureteral reimplantation remains the gold standard for correcting obstructive megaureter and some cases of VUR.[52]

Recently, laparoscopic techniques have been developed for the surgical management of VUR, which have a success rate comparable to that of open ureteral reimplantation.[53] Still; in contemporary practice, the application of a laparoscopic approach for repairing congenital obstructive megaureter in children is rare.[52]

Laparoscopic ureteral reimplantation has never achieved popular consensus as it is a technically demanding procedure, especially when intracorporeal freehand suturing is required for tailoring and reimplantation; however, efforts have been directed toward reducing the perioperative morbidity period and the length of hospitalization.[53]

Laparoscopic extravesical transperitoneal approach following the modified Lich-Gregoir technique is feasible even in unilateral, bilateral simultaneous and double system. In most cases, laparoscopic extravesical transperitoneal approach is not associated with bladder dysfunction as in open surgery even in bilateral procedures.[53]

New aspects of the technology will shape its future uses, particularly robotic technology, yet the same questions will apply; how can the value and role of this technology be assessed objectively? What are the responsibilities of the practitioner to his/her patients in terms of interpreting and using these technologies? What should the basic criteria be for acceptance or rejection of a technology in pediatric urology?

As more surgeons enter the field with laparoscopic experience, and as technology continues to improve, pediatric urological laparoscopy will also continue to progress and minimally invasive approaches to urological conditions will become more available to children in the near future.