MANAGEMENT OF POSTERIOR URETHRAL VALVE – A REVIEW OF 104 CASES



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CERTIFICATE

This is to certify that this dissertation titled 'POSTERIOR URETHRAL VALVE – MANAGEMENT – REVIEW OF 104 CASES' is a bonafide work of Dr.K.MOHAN KUMAR, submitted for the qualifying examination in M.Ch., Paediatric Surgery, Branch-V, to be held in August 2007 by the Dr. M.G.R. Medical University.

Signature of the H.O.D

Signature of the Dean.

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This study would not have seen the light of the day, had not our patients showed the kind co-operation they extended. I sincerely thank them.

OUR EXPERIENCE WITH THE MANAGEMENT OF POSTERIOR URETHRAL VALVES

A REVIEW OF 104 CASES

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OUR EXPERIENCE WITH THE MANAGEMENT OF POSTERIOR URETHRAL VALVE

A REVIEW OF 104 CASES

INTRODUCTION

Posterior urethral valve (P.U.V) represents the most common cause of congenital obstructive uropathy leading to childhood renal failure. The incidence of P.U.V. is approximately 1:5000 to 1:8000 infant males^{24,27}. Challenges posed by children with P.U.V are multiple. Obstruction by valves is a process which involves the entire urinary system. So the surgeons involved in the acute and long-term care of these patients are faced with the challenging management problems that are multiple and interdependent.

Appropriate clinical suspicion remains the key to diagnosis which is confirmed by standard imaging techniques. Initial therapy usually consists of skillful endoscopic work to remove the obstruction²⁴. The outcome is however far from determined at this point. The risk of renal compromise and ultimate renal failure is a potential problem for each patient. This outcome may be altered by appropriate intervention, but in most cases the renal development in utero determines the need for eventual dialysis or transplant.

In the past, the treatment of P.U.V was based primarily on the mechanism of obstruction and its relief. The current treatment of P.U.V. is based upon our evolving knowledge of the consequences of bladder outlet obstruction on our patients renal health and is enhanced by continuous, long term follow-up.

AIMS OF THE STUDY

- 1. TO ANALYZE OUR EXPERIENCE WITH THE MANAGEMENT OF PUV AND TO STUDY THE SHORT TERM OUTCOME OF PATIENTS MANAGED AT OUR INSTITUTION.
- 2. TO ANALYZE THE DIFFERENT MODALITIES OF TREATMENT AND TO CHOOSE THE BEST AMONG THEM.
- 3. TO DETERMINE THE COMPLICATIONS AND FOLLOW-UP CRITERIA OF PUV.
- 4. TO DETERMINE THE PROGNOSIS OF OUR ANTENATALLY DETECTED PATIENTS.

MATERIALS AND METHODS

Study Type : Retrospective and

prospective study

Study Group : 104 patients of P.U.V

were analyzed regarding presenting symptoms, signs and the different modalities of treatment

instituted

Study Period : January 1998 to

January 2007 (9 years)

Study Center : Coimbatore Medical

College & Hospital, Coimbatore-18.

METHODOLOGY

In our study of 104 patients of P.U.V, 34 (32.70%) cases presented in the neonatal period, 31 (29.80%) cases between 1 to 12 months of age, 24 (23.08%) patients in the 1 to 5 year age group and 15 (14.42%) were of 5 to 12 years of age. 8 patients were diagnosed antenatally.

Among the 104 patients, 47 were treated by Primary endoscopic valve fulguration and the remaining cases underwent urinary diversion either in the form of vesicostomy in 45 patients or ureterostomy in 9 patients. We have analyzed the results and have presented the discussion with a brief review of literature, follow-up and recommendations.

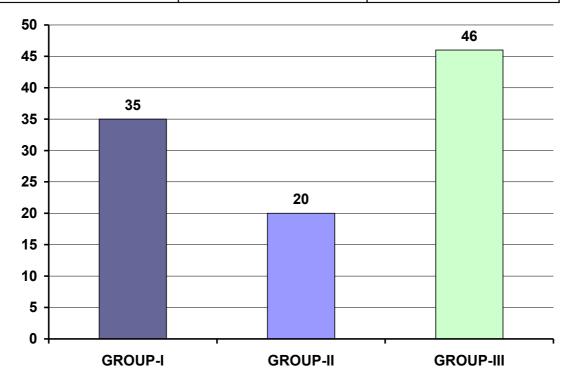
MANAGEMENT

Our patients were grouped as follows

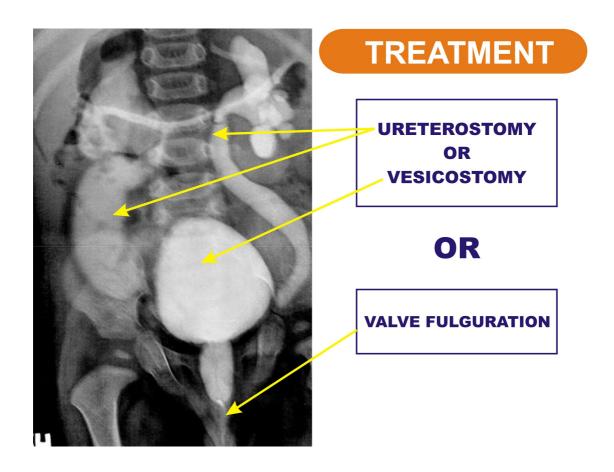
| Group | General Condition | Infection | Urea & Creatinine |
|---------|-------------------|-----------|-------------------|
| Group-1 | Good | Absent | Normal |
| Group-2 | Poor | Absent | High |
| Group-3 | Poor | Present | High |

TABLE – 1
GROUP DISTRIBUTION

| Group | No. of Patients | Percentage |
|-----------|-----------------|------------|
| Group-I | 35 | 34.65 |
| Group-II | 20 | 19.80 |
| Group-III | 46 | 45.55 |



POSTERIOR URETHRAL VALVE



RESULTS

Study was carried out over a period of 9 years from January 1998 to January 2007. Patients were followed up monthly after intervention.

DEMOGRAPHY:

Total number of cases : 104

Age : Birth till 12 years

Duration of Symptoms : 1 Day to 5 years

Duration of Follow-up : 3 Months to 7 years

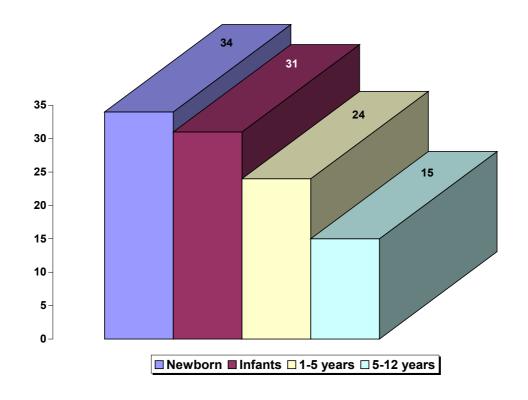
AGE DISTRIBUTION

104 patients came to our department from between January 1998 to January 2007 (9 years). 34 were neonates, 31 were in 1-12 month.age group, 24 were in 1- 5year age group and 15 were in 5-12 year. age group. 8 presented primarily in 5-12 age group.

TABLE – 2

AGE DISTRIBUTION

| Age | No. of Cases | Percentage |
|-------------|--------------|------------|
| New Born | 34 | 32.70 |
| 1-12 months | 31 | 29.80 |
| 1-5 years | 24 | 23.08 |
| 5-12 years | 15 | 14.42 |

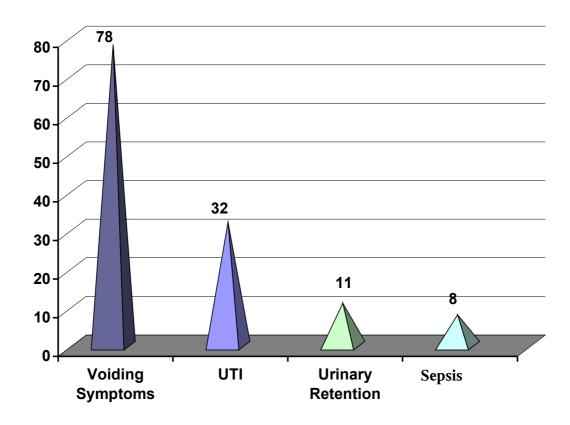


PRESENTATION: CLINICAL SYMPTOMS

78 patients had voiding symptoms, 11 had urinary retention and 32 had urinary tract infection. Organisms isolated from them were predominally E.coli., proteus. 8 patients were admitted with sepsis.

TABLE – 3
PRESENTATION: CLINICAL SYMPTOMS

| Symptoms | No. of Patients | Percentage |
|-------------------|-----------------|------------|
| Voiding Symptoms | 78 | 60.47 |
| UTI | 32 | 24.80 |
| Urinary Retention | 11 | 8.53 |
| Sepsis | 8 | 6.20 |



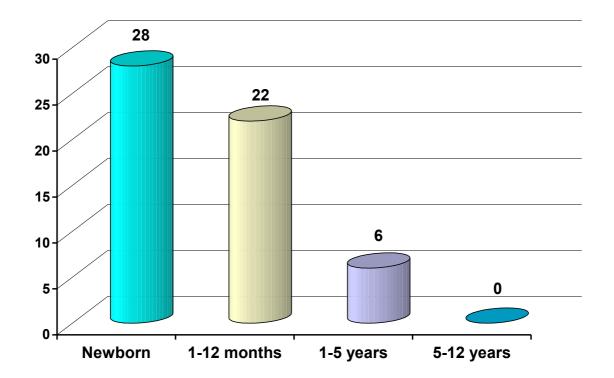
PRESENTATION: SIGNS

On presentation bladder was palpable in 62 patients. In 11 patients kidneys were also

palpable.

TABLE – 4
BLADDER PALPABILITY

| Palpable Bladder | No. of Patients | Percentage |
|------------------|-----------------|------------|
| Newborn | 28 | 82.35 |
| 1-12 months | 22 | 70.97 |
| 1-5 years | 6 | 25 |
| 5-12 years | 0 | 0.00 |

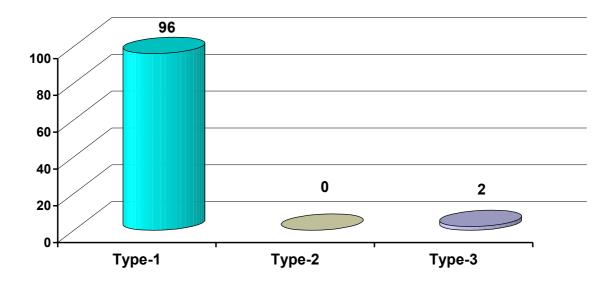


TYPE OF VALVE AND CYSTOSCOPY FINDINGS

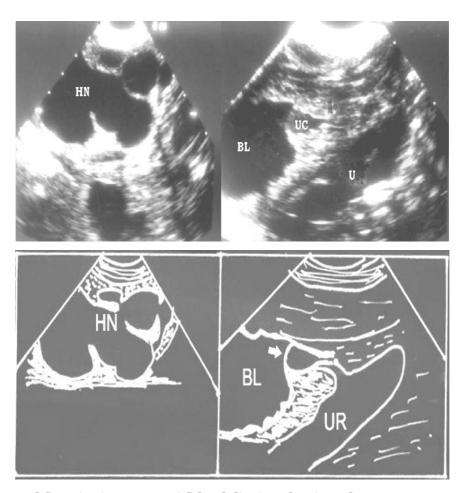
Type-1 valve was seen in 96 patients, Type-3 valve was seen in 2 patients. In 3 patients cystoscopy was not done. In 3 patients the typical valve was not seen. Bladder trabeculations and sacculations were seen in 38 patients and features of cystitis were seen in 22 patients.

VALVE FREQUENCY

| Valve Type | No. of Patients | Percentage |
|------------|-----------------|------------|
| Type-1 | 96 | 95.05 |
| Type-2 | 0 | 0.00 |
| Type-3 | 2 | 1.99 |



PUV



POSTNATAL -ULTRASONOGRAM OF ABDOMEN – PUV



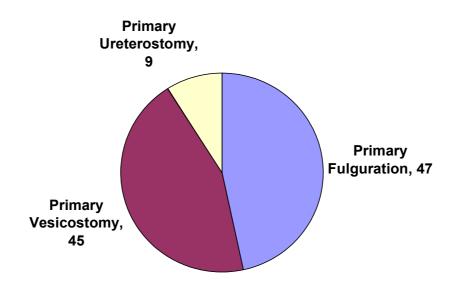
MICTURATING CYSTOURETHROGRAM – PUV

TREATMENT – PRIMARY

Primary fulguration was done in 47 patients including 16 neonatal fulgurations. Facilities for newborn fulguration have been available for the past 4 years. 54 diversions of which 45 primary vesicostomies and 9 primary ureterostomies were also done.

TREATMENT DISTRIBUTION – PRIMARY

| Types of Treatment | No. of Cases | Percentage | | | |
|-------------------------|-----------------------------------|------------|--|--|--|
| Primar | Primary Fulguration – 47 Patients | | | | |
| Newborns | 16 | 45.10 | | | |
| 1 month-12yrs | 31 | 45.19 | | | |
| Diversion – 54 Patients | | | | | |
| Primary Vesicostomy | 45 | 54.01 | | | |
| Primary Ureterostomy | 9 | 54.81 | | | |



■ Primary Fulguration ■ Primary Vesicostomy □ Primary Ureterostomy

We fulgurated 31 patients after diversion. We also did 2 ureterostomies and 1 vesicostomy after fulguration. Initially dry fulguration was done in 22 of our patients. The present policy is to do simultaneous fulguration and diversion closure in one sitting, ultimately avoiding dry fulguration. 7 patients required refulguration for residual valve.

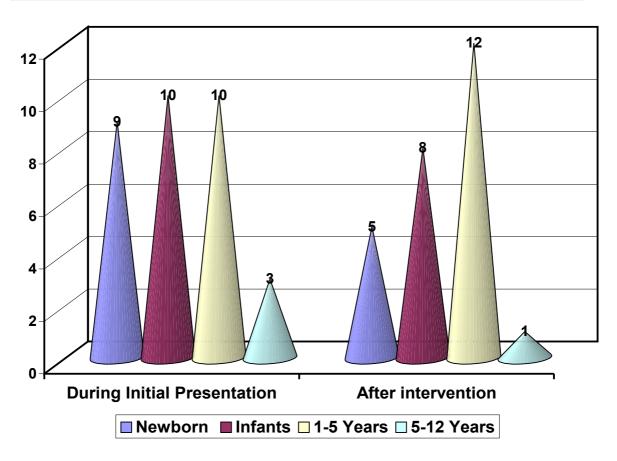
In our series 20 patients were catheterized initially for stabilization. Suprapubic cystostomy was done in 1 patient in a private hospital and referred .Also valvotome was used to check the adequacy of fulguration in 5 patients.Fogarty's dilator was also used for the same in 10 patients. In 12 patients peritoneal dialysis had to be done to stabilize the general condition.

URINARY TRACT INFECTIONS

Urinary tract infection was seen in 32 of our patients during the initial period before treatment and in 26 patients after intervention. Organisms isolated were mostly E-coli and proteus.

AGE DISTRIBUTION – UTI

| Period | Newborn | 1mon-1yr | 1-5 Years | 5-12 Years |
|----------------|---------|----------|-----------|------------|
| During Initial | | | | |
| Presentation | 9 | 10 | 10 | 3 |
| After | | | | |
| intervention | 5 | 8 | 12 | 1 |

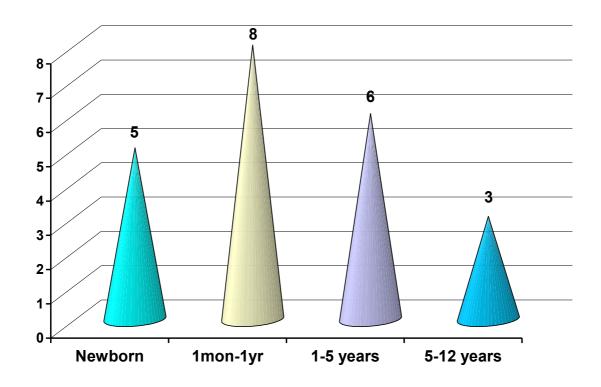


OCCURRENCE OF VESICOURETERIC REFLUX

Vesicoureteric reflux was seen in 22 of our patients of which 5 were newborns, 8 were in 1mon. to 1yr. age group, 6 were in age group 1-5 years and 3 were in 5-12 years group.

AGE DISTRIBUTION OF VUR

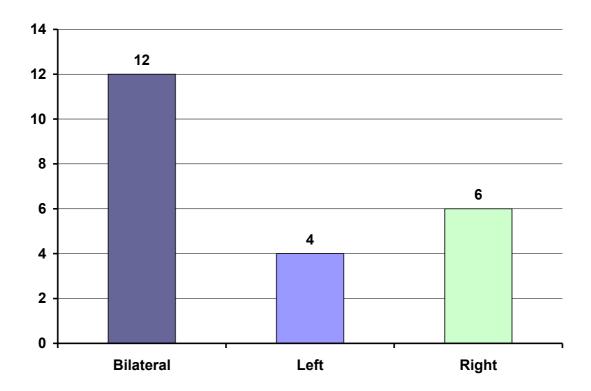
| Age | No. of Cases | Percentage |
|------------|--------------|------------|
| Newborn | 5 | 14.71 |
| 1mon-1yr | 8 | 25.81 |
| 1-5 years | 6 | 25.00 |
| 5-12 years | 3 | 20.00 |



Out of them 12 patients had bilateral VUR, 4 had reflux on the left and 6 had reflux on the right.

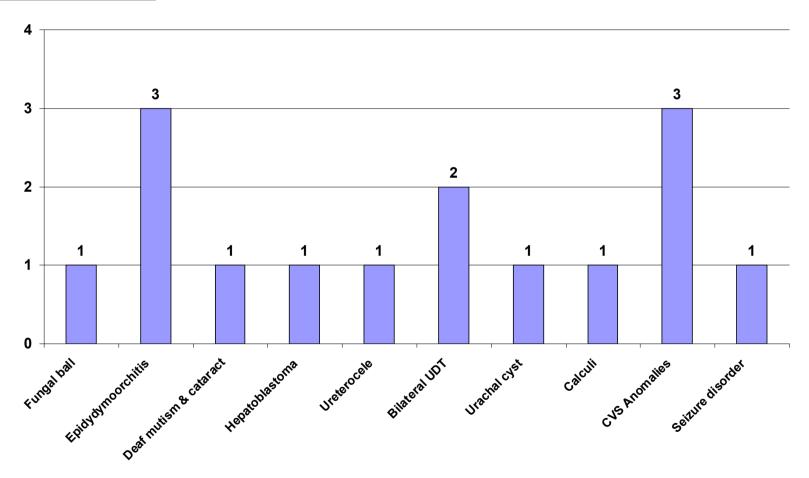
SIDE DISTRIBUTION OF VUR

| Sides | No. of Cases |
|-----------|--------------|
| Bilateral | 12 |
| Left | 4 |
| Right | 6 |



ASSOCIATED CONDITIONS

ASSOCIATED CONDITIONS:

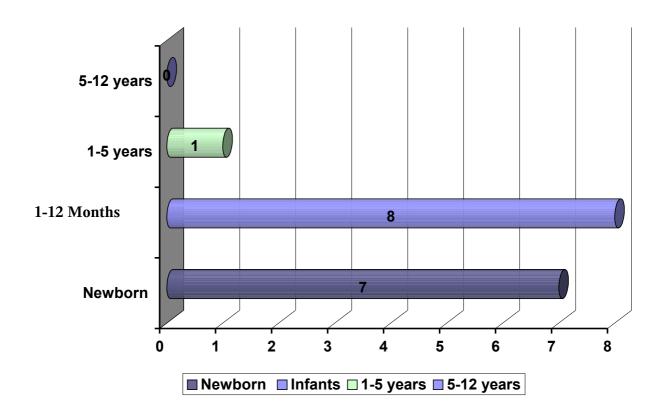


MORTALITY

There were 16 deaths in our study. 7 were newborns, 8 were 1mon-1yr of age group and 1 three and a half year old child.

AGE DISTRIBUTION – MORTALITY

| Age | No. of Death | Percentage |
|------------|--------------|------------|
| Newborn | 7 | 20.59 |
| 1mon-1yr | 8 | 25.81 |
| 1-5 years | 1 | 4.17 |
| 5-12 years | 0 | 00.00 |



FOLLOW UP

Patients followed up with

- 1. Monthly urine for culture and sensitivity
- 2. Quarterly blood urea and S. Creatinine
- 3. MCU and USG if warranted

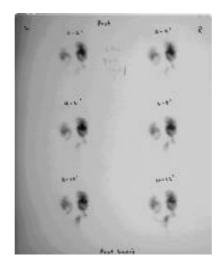
Of these followed up patients we had to repeat the fulguration for residual valves in 7 patients. We also did 2 ureterostomies and 1 vesicostomy after fulguration. Post operative stricture was seen in 7 of our patients and we had to do urethral dilatation. Some times they required repeated dilatations.

18 patients were lost to follow up. Now 67 patients are regularly on follow up.

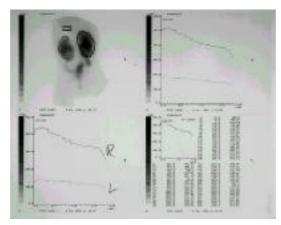
To evaluate the morbidity we did radio nuclide studies (DTPA / DMSA) in 10 of them. Urodynamic study was done in 11 patients. Subsequently 4 patients underwent nephrectomy (2 open and 2 laparoscopic). We also did reimplantation in 2 patients. We did augmentation (Ureterocystoplasty) with Mitrofanoff procedure in one patient. We also attemped STING procedure in 1 patient.

PUV – RADIO NUCLEOTIDE STUDIES

DTPA demonstrate radiotracer accumulation within the dilated renal collecting systems and dilated ureters. The bladder remains empty because of catheter drainage.

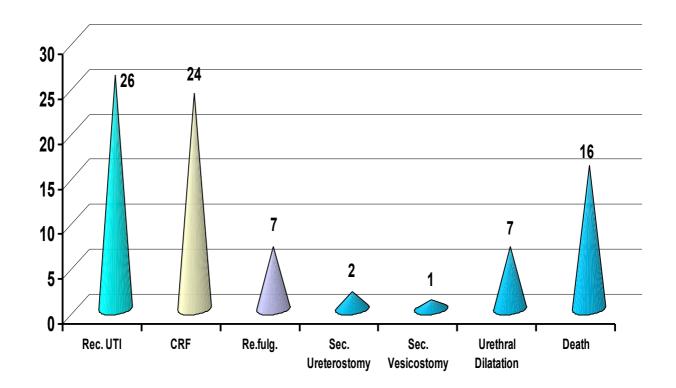


DTPA demonstrate accumulation of radiotracer within the renal collecting systems bilaterally; within the dilated ureters bilaterally; and within a small, irregular-appearing bladder. Renograms (bottom left) demonstrate poor clearance of contrast material from the renal collecting systems. The relatively poorer function in the left kidney reflects congenital renal dysplasia.



FOLLOW UP

| Rec. UTI | CRF | Re.fulg. | Sec. Ureterostomy | Sec. Vesicostomy | Urethral Dilatation | Death |
|-------------|-----|----------|----------------------|---------------------|------------------------|-------|
| 26 | 24 | 7 | 2 | 1 | 7 | 16 |



ANTENATALLY DETECTED PATIENTS

Out of 8 patients detected antenatally 2 patients expired and 3 were lost to follow-up (probably expired). Out of the remaining 3, 1 patient had nephrectomy and the other had ureterocele with VUR so reimplanted on one side and STING tried on another side. Only one patient is at present without morbidity.

The prognosis of antenatally detected patients has been dismal in our study.

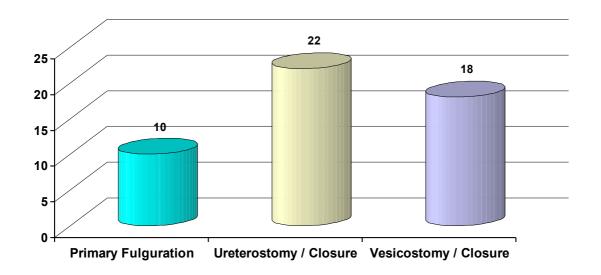
LENGTH OF HOSPITAL – STAY

Finally the length of hospital stay for primary fulguration was on an average 7 to 10 days whereas for ureterostomy and closure the patient had to get admitted two times and total

length of stay was 15 to 20 days. For vesicostomy and closure the average length of stay was 14 to 20 days for 2 admissions. So therefore in our study the outcome of patients with PUV depended upon the initial renal status irrespective of the management options used. So it is better to do fulguration primarily than diversion. Fulguration is also cost effective, with lesser no. of surgeries, scarless and also economical for the Government.

LENGTH OF HOSPITAL – STAY

| Primary Fulguration | Ureterostomy / Closure | Vesicostomy / Closure | |
|---------------------|------------------------|-----------------------|--|
| 10 days | 22 days | 18 days | |

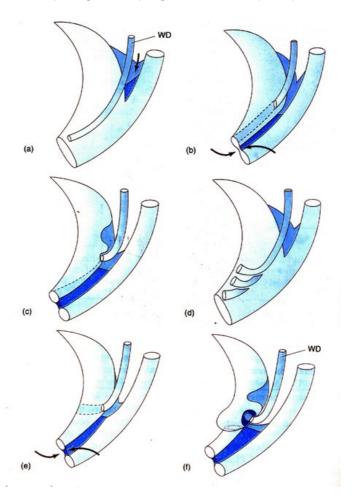


REVIEW OF LITERATURE

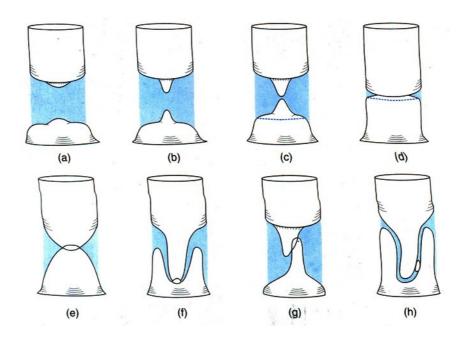
ANATOMY AND EMBRYOLOGY

Located in the posterior midportion of prostatic urethra is the verumontanum, containing paired ejaculatory duct openings and urogenital sinus remnant known as prostatic utricle. Extending distally from the veru in the midline is the crista-urethralis and diverging from this are the plicae colliculi, which merge into the membranous urethral area into 2 oblique folds. Tolmatchew in1870 proposed these to be obstructing in some infants. Stephens postulates that these plicae are normal remnants of the terminal mesonephric ducts which regress during embryogenesis, leaving only the ejaculatory duct openings. Incomplete regression of the plicae leads to fusion dorsally to form the typical PUV. Stephens theory is supported by observations of twins with valves. Livne et al suggests a mesonephric duct origin with possibility of polygenetic transmission and other undefined factors. B.W. Young maintains that P.U.V. obstruction represents abnormal development of urogenital sinus itself (persistence of the sino-vaginal bulb) and not the distal mesonephric duct.

DEVELOPMENT OF TYPE – 1 VALVE



DEVELOPMENT OF TYPE – 3 VALVE



DISCUSSION

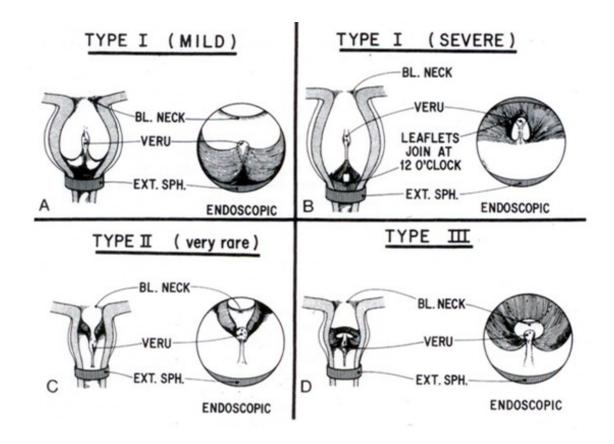
CLASSIFICATION

Dr.H.Hampton Young's long standing system of classification is still employed(1919)^{4,10,29,40}.

In Type-1 valves are membranes that originate at the verumontanum and travels distally to insert in the anterior proximal membranous urethra with an opening present posteriorly at the verumontanum. The etiology is probably a result of the mesonephric ducts entering the cloaca more anteriorly than normal and fusing in the midline. Such valves offer no resistance to retrograde instrumentation but co-opt during micturition to obstruct urinary flow. It gives characteristic the sail-in-the wind finding in MCU.

In Type-2 folds extend from upper extremity of verumontanum to bladder neck. They are never obstructive and are of historical value only.

In Type-3 valves consist of a ring-like membrane distal to the verumontanum with a perforation present centrally. Field and Stephens describe a variation of type 3 valves in which the membrane becomes stretched so that during micturition it prolapses like a windsock, into bulbar urethra. The cause of these valves is an incomplete dissolution of the urogenital membrane.95% of P.U.V. are type 1 with variations in leaflet thickness and in the degree of obliquity and coalasence at 12'O clock position. The other 5% are type 3.



Stephens (1983) suggested **type-4** valves in prune-belly syndrome⁴ Exaggeration of normal folds (plicae) without anterior fusion has been deemed partially obstructive by some (mini-valves). Despite former classifications of PUV, Devan recently stated that most likely there is a single obstructive membrane that may be altered by the passage of urethral catheters or cystoscopes resulting in variable tears of the membrane. This may be perceived as Type I or III valve. This concept of single type of valve is referred to as congenital obstructive posterior urethral membrane (COPUM)¹⁴

Also type 3 is referred as COBB'S COLLAR or MOORMAN'S RING (bulbar urethral obstruction)²⁵. We had 96 type 1 valve patients and 2 type 3 valve patients in our cystoscopy findings.

PATHOPHYSIOLOGY

Above the valves back-pressure effects are nearly always present. These take the form of a widely dilated posterior urethra, a thick walled and usually trabeculated bladder, widely dilated tortuous ureters and bilateral hydronephrosis. V.U.R is common and frequently

associated with varying degrees of dysplasia of affected kidney.

In the past, secondary narrowing at the bladder outlet was also felt to contribute to the obstruction in some cases. Y-V plasties and other operations directed to bladder neck were felt to be important. This hypertrophy is now understood to be part of the overall detrusor wall thickening that results from the inferior obstruction, and needs no specific therapy, since it is not a true point of obstruction. Infact many children found to have stress incontinence later in life have undergone a concomitant bladder neck ablative procedure. Since this approach has been abandoned, stress incontinence has eventually been eliminated.

Children with P.U.V manifest renal abnormalities that can include varying degrees of dysplasia or hydronephrosis. The renal abnormalities seen in these patients have atleast two theoretical etiologies, pressure and ureteral bud problems. The first etiology states that the bladder pressure is related to the severity of original obstruction. Dysplasia & /or hydronephrosis develops, based on gestational timing and degree of pressure transmission to the upper tracts, by means of ureteral changes that follow unrelieved bladder pressure elevations. This occurs with or without breakdown of antireflux mechanism at the U.V junction. Meguire has shown by his work in patients with myelomeningocele that chronic intravesical pressure of >40cms of water are associated with upper tract deterioration in a majority of patients. It seems likely that this same threshold is accurate in the fetus with valvular obstruction. Even in the absence of reflux, intrapelvic renal pressures reflect the underlying high pressure bladder, when the normal urinary system is first filled to capacity with urine. GFR decreases in a similar setting. The high pressure bladder therefore must play a key role in determining renal compromise but the initial pressure threshold or range that affects renal development in the fetus is yet to be determined.

The second theoretical etiology for renal abnormalities, ureteral bud induction problems, has been examined carefully by Henneberg & Stephens. The severity of renal dysplasia

was correlating with the degree of ureteral orifice malposition. They deduced that the abnormality of the ureteral bud & metanephros interaction is causative^{4,11}.

PRESENTATION

Most children are now fortunate enough to be discovered on prenatal ultrasound and fetal MRI^{3,7,9}. Treatment started early in life prevents the potential renal damage due to sepsis and pressure. At the other end of the spectrum are those fetuses with oligohydramnios, Potter facies and such severe pulmonary hypoplasia that even the most intense supportive measures cannot sustain them. Despite the fact that the obstruction is the primary problem, these infants may not be labeled as P.U.V patients because of severity of their pulmonary compromise and very early mortality.

More than half of the children with P.U.V are discovered in neonatal period. (PUV=1:5000 to 8000 live male births) and 10% are detected prenatally. Presentation in the neonates includes distended bladder, bilateral flank masses, dribbling or weak urinary stream. However a full urinary stream doesn't preclude significant urethral obstruction. Subtle signs of urethral obstruction can include failure to thrive and in some children it may be the only indication of underlying urinary difficulties. An acutely ill baby with P.U.V may have rapid respirations resulting from acidosis. Neonatal urinary ascites or urinary extravasation from kidney is usually considered to be a pressure related phenomenon.

Infants in whom the diagnosis has been missed usually present with urinary infection and acute on chronic renal failure. This is generally accompanied by hyperkalemia and severe metabolic acidosis which may lead to respiratory arrest. Water and sodium balance are often also profoundly disturbed. Septicaemia is common and may be complicated by consumptive coagulopathy.

Older boys may also present with urinary infection but often the main complaint is of a poor urinary stream with straining or urinary incontinence. It is important to maintain a high degree of suspicion until urinary abnormalities are excluded in such a child. The less severely affected child may be overlooked in the neonatal period.

DIAGNOSIS & PROGNOSTIC SIGNS

Antenatally, sonography and fetal MRI are useful in diagnosis. In sonography the typical keyhole sign of bladder and urethral dilatation is seen.12,13,40

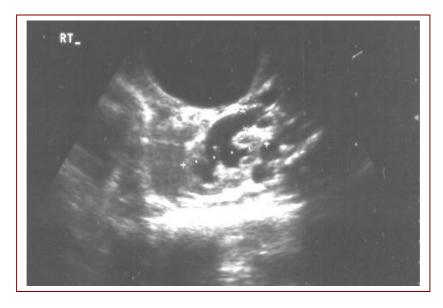
In the newborn also, standard imaging studies usually provides an unequivocal diagnosis of posterior urethral obstruction. Sonography again is the preferred initial study when obstruction is suspected, clinically and will show the typical hydroureteronephrosis, thick walled bladder and wide prostatic urethra associated with PUV.A loculated urinoma or urinary ascites can also be identified. The MCU usually confirms the diagnosis and demonstrates the reflux present in half of the patients. In our series among the 104 patients 22 had reflux (bilateral -12 left – 4 and right – 6). The dilated posterior urethra, hypertrophied bladder neck, and thick-walled sacculated bladder are typical features. At times this typical appearance can be confused with Prune Belly syndrome in which a dilated posterior urethra narrows significantly at membranous urethra, but with a widely opened bladder neck and, sometimes with a urachal remnant.

In doubtful cases diagnosis can be confirmed by cysto-urethroscopy. Since valve cusps co-opt only during antegrade flow, they usually cannot be demonstrated during retrograde passage of an instrument. The valve is best observed with the bladder filled and the endoscope placed well distal to the veru with the water source removed and the connection open. This allows irrigant to flow from the bladder through the instrument, bellowing the cusps demonstrating their coopting margins.

Functional assessment of the kidneys is best obtained with radionuclide renography, allowing quantitation of renal function. As this test is not helpful in the first month and also if done in the first few days or prior to relief of obstruction, it offers very little help in clinical management, it is postponed till 3-4 weeks. The extraction factor is a simple calculation from the renogram, that reflects the GFR of the individual kidney, based on % uptake of radionuclide in each kidney during the 2-3 minutes of study. Bilateral VUR has usually been associated with both increased morbidity and decreased renal function.

Measure of nadir serum creatinine in the first few months after treatment has also proven to be a useful sign. If the lowest post treatment creatinine is <0.8mg/dl then renal function (with upto 8 years of follow-up) has remained in a normal range. Ducket et al have recently shown that there is a subset of PUV patients that has a good renal function prognosis, due to a "POP-OFF" mechanism (Large bladder diverticulum, VURD syndrome, urinary ascites, patent urachus).^{4,11,38}. Infants with VURD syndrome (valves, unilateral reflux and dysplasia in a non-functioning unit) seems to spare the function of contralateral renal unit by a pressure "pop-off" mechanism which apparently protect the nephrons not subject to reflux. This group of patients has significantly better renal function as measured by serum creatinine.

NATURE'S PRESSURE POP- OFF MECHANISMS



PERIRENAL URINOMA



BLADDER DIVERTICULUM



UNILATERAL VUR

PROGNOSTIC VARIABLES – ANTENATAL 4,11,25

| Variable | Good predictors | Poor predictors | |
|--|---|--|--|
| In – utero presentation (weeks) | > 24 | <24 | |
| Amniotic fluid volume | Normal to moderately increased | Moderate to severly decreased | |
| Sonographic appearance of renal parenchyma | Normal to slightly increased echogenicity | Increased echogenicity to frankly cystic | |
| Fetal urinary values | | | |
| Sodium (mEq/L) | < 100 | >100 | |
| Chloride (mEq/L) | <90 | >90 | |
| Osmolality (mOsm) | <210 | >210 | |
| Urinary output (ml/Hr) | >2 | <2 | |
| Beta 2 microglobulin | <6 | > 6 | |

PROGNOSTIC VARIABLES – AFTER BIRTH

| Variable | Good predictors | Poor predictors | |
|---------------------------------------|---------------------------------|---------------------------|--|
| Sonography – | -Present | -Absent | |
| identification of CMJ differentiation | -Pyramids in atleast one kidney | -Hyperechoic, no pyramids | |
| S. Creatinine | < 0.8 at one year | >0.8 at one year | |
| Reflux | No reflux | Bilateral reflux | |
| Continence | At 5 years | incontinence | |
| Pop off mechanisms | | | |
| Urinary ascites | Present | Absent | |
| Bladder diverticulum | Present | Absent | |
| VURD | Present | Absent | |
| Patent urachus | Present | Absent | |
| | | | |

DIFFERENTIAL DIAGNOSIS

A variety of other conditions may masquerade as PUV. Prominent infra-collicular folds which can sometimes be made out on a good quality study in normal children. Hesitant voiding in a normal baby may cause an abrupt change in caliber of posterior urethra. Extrinsic compression by pelvic floor may cause one or more concentric indentation in urethral contour. They are considered as normal variants.

A neuropathic bladder may closely simulate a valve, but the thin stream below the obstruction will be seen emerging from the center of the external urethral sphincter rather than from posterior margin as seen with a valve. In such cases the spine should be carefully examined and other evidence sought of a neurological deficit in perineum or lower limbs. A posterior urethral stricture may cause a similar appearance but this will invariably be associated with history of urethral or pelvic trauma.

The Prune-Belly syndrome may closely mimic a valve, but the correct diagnosis should be suspected from the appearance of bladder which is horizontal and is invariably smooth walled, and the dog-leg configuration of posterior urethra which often bears a utriculus masculinus. A distended non-visualized ectopic ureter opening into ejaculatory duct may distort and partially obstruct the posterior urethra thus simulating a valve . Whilst dilatation of the posterior urethra may also be caused by a prolapsed ectopic ureterocele or posterior polyp careful examination of these films however will usually reveal a filling defect leading to correct diagnosis 4,11,24.

PRENATAL INTERVENTION

Is useful only for selected individuals and still experimental. Only few cases have been tried but a price have to be paid, in the form of fetal loss and maternal morbidity. At present second or third trimester discovery of the obstructive process that begins and may

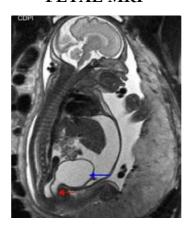
have had its major effects in the first trimester rarely warrants in-utero intervention. Progressive bilateral hydronephrosis with worsening oligohydramnios detected in second trimester (21-28 weeks) may however be an indication. When intervention is planned it is done in the tertiary centres. The potential for ECMO in those babies with borderline pulmonary hypoplasia is probably a treatment of the future. Endoscopic vesico-amniotic shunting and fetal cystocopy are being experimented^{3,7,9}.

FETAL ULTRA SOUND

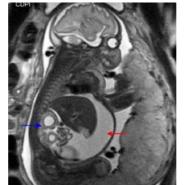


The dilated proximal urethra and dilated bladder (thickened) resembles a keyhole (arrow) extending from the bladder toward the fetal perineum

FETAL MRI



Sagittal T2 view showing a dilated bladder (arrow) and proximal urethra (arrow). $\mathbf{FETAL} \ \mathbf{MRI}$



Sagittal T2 view showing the dysplastic right kidney (arrow) and urinary ascites (arrow). Note the oligohydramnios.

INDIAN SCENARIO OF PUV

The incidence of antenatal diagnosis is only 10% in India. Urinary diversion is done in 50% of cases in most centers. In tertiary centres 80% of cases are ablated primarily. The results of laser fulguration of valves from AIIMS, New Delhi¹⁸ and the primary newborn fulguration from other centers in India are encouraging. Abraham from Kerala, Gopal from Varanasi and Kulasekar from Colombo have developed hooks for vale ablation but the use is limited to their own centers only^{31,32}. Fetal surgery is not done in any of our tertiary centres.

PRE OP PREPARATION

On suspicion 5 fr to 8 fr plastic infant feeding tube is inserted for continuous bladder drainage for selected patients. The self retaining catheter is avoided as the hypertrophied bladder tends to clamp down around the balloon and obstruct the ureters. In our series we had to catheterize 20 of our patients immediately on admission. One patient was referred with supra pubic cystostomy.

If the catheter is curled in the dilated PUV so that there is failure to drain, withdrawing the catheter for a few centimeters and repassing it with a finger in the rectum will usually ensure its passage through the hypertrophied bladder neck²⁶. Persistent difficulty can usually be resolved by injecting a few ml of contrast medium through the catheter and manipulating it under fluoroscopic control.

Hyper kalemia and severe metabolic acidosis should be corrected. Hydration is assessed and in severe cases peritoneal dialysis is warranted as a resuscitative measure. In our series peritoneal dialysis was done for 12 of our patients.

As a routine, blood and urine culture are taken. Then amino glycosides or third generation cephalosporin IV are started. When in sepsis, blood coagulation studies are also carried out. Chest X-ray is taken in respiratory distress to exclude pneumothorax

secondary to pulmonary hypoplasia. Even after this, if serum creatinine doesn't begin to fall percutaneous drainage of upper urinary tracts is considered. MCU is done on the day of operation to confirm PUV and to detect if VUR is also present. MCU in newborn is a major procedure and done under strict aseptic precautions. MCU is skipped only in special situations where all information is obtained with ultra sound or radio isotope or rarely when baby is very sick and needs emergency diversion as a life saving procedure. MCU is done later when the baby's condition is stable.

MANAGEMENT PLAN

It is a surgical emergency, not like intestinal obstruction but cannot be delayed as an elective case. Ideally treated within few days of diagnosis. As mentioned earlier, we divided our patients into 3 groups.

In Group - I

Once investigations were completed, end fulguration was done. MCU was done just before operation. No need for pre-op IVF or bladder drainage. 35 patients were of this group.

<u>In Group – II</u>

IV fluids started on admission, bladder drained using 5 fr feeding tube, prophylactic antibiotics given and re-evaluation of the baby was done after 48 hours. 20 patients came under this group.

Sub-Group-A

Marked improvement in bio-chemical status, MCU done just before operation. Valve fulguration using endoscope was done. 12 patients came under this group.

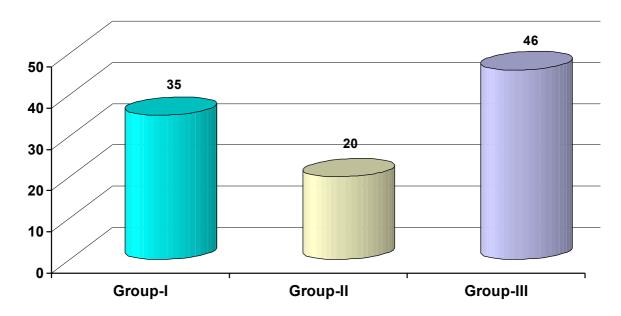
Sub-Group-B

No improvement, so urinary diversion like vesicostomy / ureterostomy was done.

8 patients came under this group.

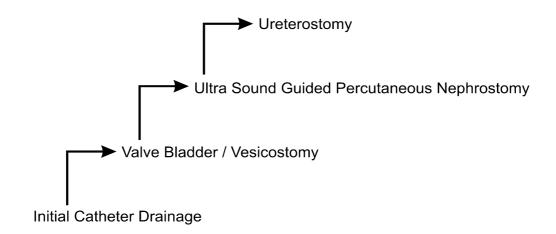
In Group - III

Vesicostomy/ureterostomy was done as soon as resuscitation was completed and further treatment of valve was done after baby was stabilized. 46 patients came under this group.

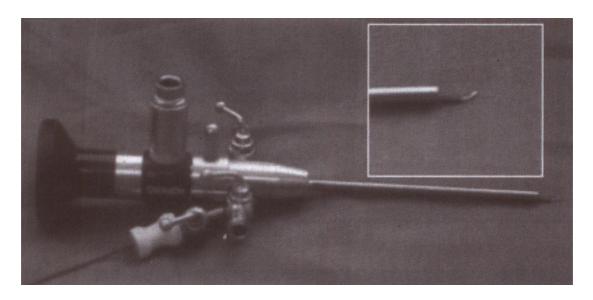


In new born – IV antibiotics was given before any procedure. In older children without urinary infection primary resection of valves and further surgery was considered later. Hendren et al performed total urinary tract re-construction in newborn period for patients with severe obstructive uropathy due to PUV and included ureteral tapering and re implantation. Primary total re construction is generally abandoned nowadays because of high risk of obstruction associated with ureteral re impanation into thick and obstructed bladders.

STEP LADDER PROTOCOL 25



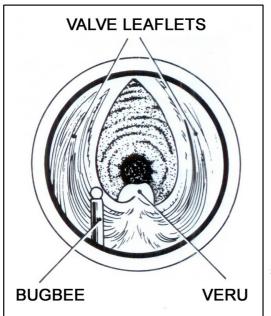
CYSTOSCOPE FOR FULGURATION



TECHNIQUE OF SURGICAL PROCEDURE – VALVE

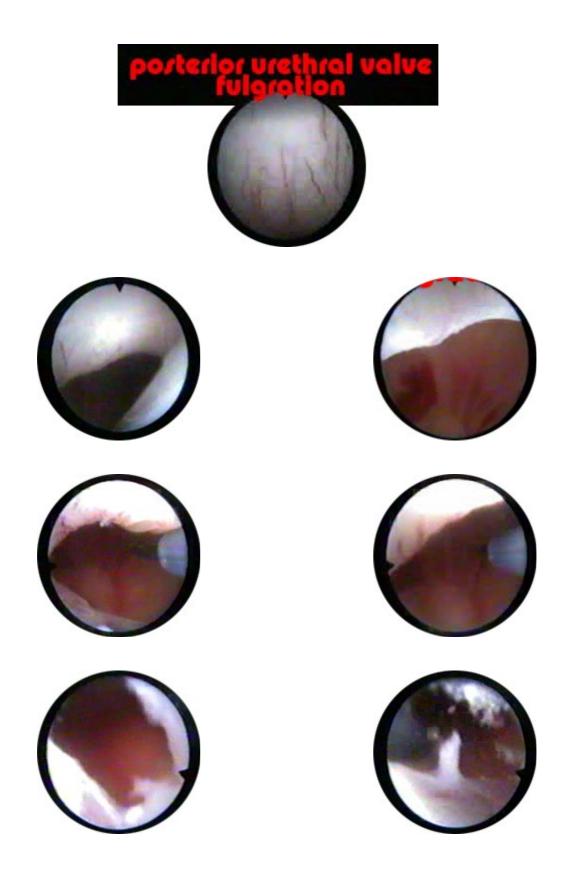
FULGURATION

The calibre of penile urethra should ideally be checked with a well lubricated 10 fr sound



introduced for 1 to 2 cms²⁶. If necessary a meatotomy can be performed but no attempt should be made to dilate the urethra. Diagnosis is confirmed using a well lubricated 9.5 fr or 10fr. Cystoscope introduced under vision.

Resection of valves is undertaken using a 6 fr cystoscope fitted with bugbee electrode in newborns with the 3 F working channel^{4,11}. For older children 10 fr cystoscope is used. Some times a guidance wire is introduced through the 3F ureteric stent so that only the tip is exposed. Instrument is first assembled and the alignment of working parts is checked using 0 degree telescope. The sheath is then dried and thoroughly coated with water soluble lubricant and with introducer in place is gently inserted through the meatus. Introducer is now removed and the instrument is re assembled and gently advanced under vision towards the bladder neck. It is frequently necessary to angle the eye piece end of the instrument downwards to allow the beak to move interiorly to pass through the bladder neck. Once in the bladder the shape and position of ureteric orifices if possible are noted and presence of any periureteric diverticulum is noted.



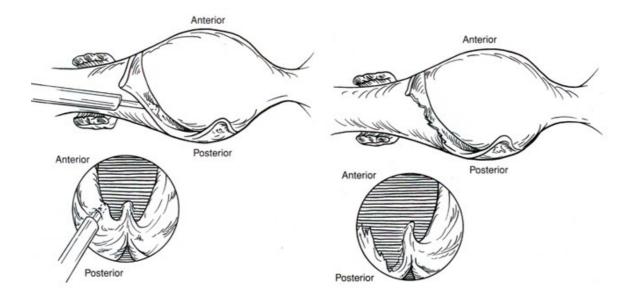
The instrument is now rotated through 180 degrees and with irrigation flowing in under low pressure it is progressively withdrawn. Once through the bladder neck, scope is run down along the anterior wall of posterior urethra until just beyond the veru, the valve will suddenly snap across the anterior portion of the field of view, like a curtain. With further withdrawal of the instrument and manipulation the valve will be clearly seen. A short burst of cutting current is then applied.

There is no need to excise the valves, but it is necessary to divide the valves at 2 points so that valvular obstruction is destroyed and urinary obstruction is removed. We divide the valves at 2 points 5 and 7'O clock position. In a rare instance of a diaphragm, we also divide the valve interiorly at two more sides 10 and 12'O clock positions. Attempts to excise the valve leads to increased incidence of stricture formation. Any remaining free floating tags do not require treatment. It is better to under cut rather than over cut. Cystoscopy is now removed and the presence of unobstructed urethra is confirmed by manual expression of bladder (BUDDS TEST). Finally a 8 fr feeding tube is passed and fixed with 3/0 silk suture to the prepuce. It is removed after 24-48 hours. If any significant bleeding occurs, attempts at valve ablation is discontinued and situation is reassesed after 2 to 3 days of catheter drainage. In older children 10 fr cystoscopy is used employing similar technique.

If urethra is too small for cystoscope some centers use perineal urethrostomy to divide the valves. They also use a trans vesicle route using angled scope. Others use 9 fr examining scope (with an irrigation channel) and use an electrode or a ureteric catheter through the channel.

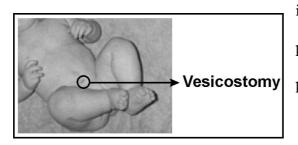
Antegrade valve ablation through an established vesicostomy or temporary percutaneous cystostomy, has been described. Neodymin – Yag laser energy instead of electrical

current is used. Other methods of valve destruction have included open resection, pressure necroses from an indwelling urethral catheter, destruction by dilatation or fulguration through a perineal urethrostomy or rupture of valve by a balloon catheter either Foleys or Fogarty. In an improvement on an old theme, Whitaker has reintroduced the use of an insulated "Crochet Hook" type of instrument for blind valve ablation at 12'O clock position²⁰. Using a similar approach, rupture of the valve has been accomplished blindly and without anesthesia using valvotome in a retrograde manner. We have used check valvotome in 5 of our patients after fulguration under anaesthesia. We have also used check fogarty dilator for 10 of our patients. Although direct endoscope ablation is preferable, the usefulness of these modifications is apparent.



<u>URINARY DIVERSION – VESICOSTOMY AND URETEROSTOMY</u>

In uraemic and infected children we do vesicostomy described by Blocksom in our centre^{27,28} BLOCKSOM VESICOSTOMY is fashioned through a small transverse



incision half way between the umbilicus and pubis. The fascia is incised and the peritoneum is pushed superiorly off the dome

of the bladder. The urachal remnant is divided and the dome of the bladder is pulled up to the skin. The fascia is secured to the bladder wall to form the required defect (No 22 Fr) and the bladder is matured as flush stoma. We did vesicostomy primarily in 45 patients and secondarily in 1 patient.

We did URETEROSTOMY primarily in 9 of our patients and secondarily in 2 patients. With infant in lateral decubitus position, a postero lateral oblique skin incision is made in the lower flank region. Muscle layer is divided and retroperitoneum is entered. Dilated tortuous ureter is identified and mobilized enough to reach the skin without tension and divided completely. An infant feeding tube is passed proximally and distally to ensure that there are no angulations. Then ureteral adventia is sutured to the external oblique fascia anteriorly and posteriorly. Muscle and fascial layers are re approximated on both sides of exteriorized ureter.

POST OP TREATMENT- 48 HOURS

IVF, antibiotics and bladder drainage is needed in immediate post OP period. These babies developed transient diuretic phase following release of obstruction (Post obstructive diuretic phase) and urinary output may vary between 300 to 1000 ml or even more. Adequate amount of IV fluids is necessary for 3 to 7 days depending upon status of upper tract. Bladder is catheterized for 3 to 5 days if necessary.

A prophylactic dose of antibiotic usually trimethoprim – sulphamethoxazole is given for 1 month to guard against infection in the healing posterior urethra. This is continued for 6 months in those infants who cystogram reveals VUR. Sodium bicarbonate supplements are also frequently necessary to correct a persistent metabolic acidosis and these may be continued for a year or more. Polyuria is also common and parents are advised to give supplementary clear feeds early in the event of a diarrhoel illness.

FOLLOW – UP

We usually do urethral calibration if there is a stricture formation¹⁹. Then we do a post-op MCU 4 to 6 months after valve fulguration if warranted to confirm the adequate resection of valve and to determine if VUR still persists. If adequate and if VUR has disappeared then the vesicostomy or ureterostomy closure is done. If there is some massive reflux, that needs corrective surgery. We have done anti reflux surgery in 2 cases.

AT 3 MONTHS

The GFR of each kidney is measured by the slope clearance method using 99m TC DTPA if needed, and an IVU carried out. Also serum creatinine, electrolytes and acid base status checked. We had to do DTPA in 4 of our patients for the consequences of PUV.

AT 6 MONTHS

MCU is done. Normally in one-third cases, VUR disappears. If VUR is still persistent 99m TC DMSA done. We have done DMSA for 6 patients.

PROBLEMS WE ENCOUNTERED AFTER INITIAL INTERVENTION

<u>Reflux</u> – seen in 50% of valve patients. Spontaneous resolution of reflux expected in atleast 20% of these patients after valve ablation. So a period of watchful waiting on prophylactic antibiotics warranted. Reflux has been demonstrated to resolve upto 3 years after treatment.

Indications for surgical intervention during this period include break -through UTI or massive reflux that interferes with the adequate emptying of system. If re implantation is necessary care must be taken to avoid problems with a thick walled non compliant bladder. Post-op complication rates of persistent reflux or obstruction in the range of 15 to 30% can be expected. Trans uretero urethrostomy with a unilateral re implant and psoas hitch may give better results.. Bladder dysfunction will also compromise the results. We had VUR in 22 patients and we have done 2 reimplantations and tried STING procedure in one patient.

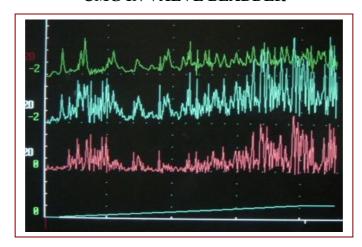
VALVES, UNILATERAL REFLUX, DYSPLASIA (VURD SYNDROME)

Ducket et al have emphasized this syndrome of renal dysplasia associated with unilateral reflux. Spurious function on IVP or renal scan with delayed films may be misleading due to reflux. When recognized one can avoid re-implantation of a non functioning system. This syndrome is visualized normally in left, but right also involved some times. Fortunately this seems to be an effective pressure relieving adaptation which protects the uninvolved side. To improve voiding dynamics

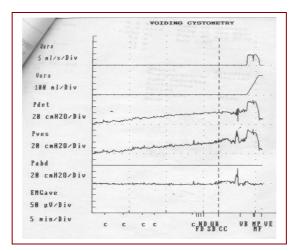
- 1) Nephrectomy but not Ureterectomy
- 2) Unilateral loop Ureterostomy
- 3) Ureterocystoplasty
- 4) Trans Uretero Ureterostomy can be done.

We have done 4 nephroureterectomies (2 open and 2 laparoscopic) in our patients, 2 ureterostomies and 1 ureterocystoplasty in our study.

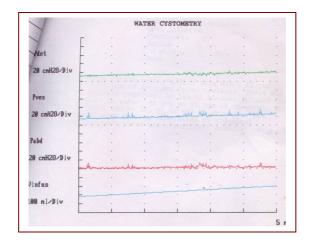
CMG IN VALVE BLADDER



UNSTABLE



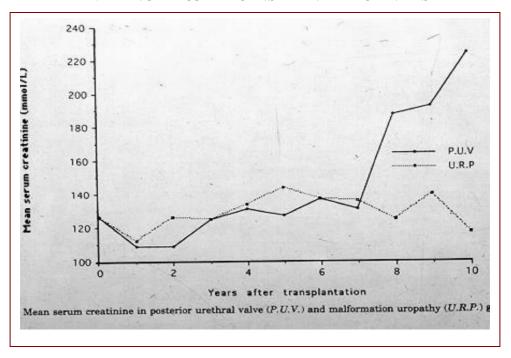
HYPOCOMPLIANT



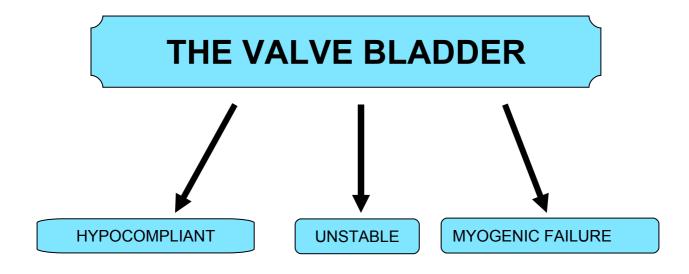
MYOGENIC FAILURE

DOES THE VALVE BLADDER MATTER?

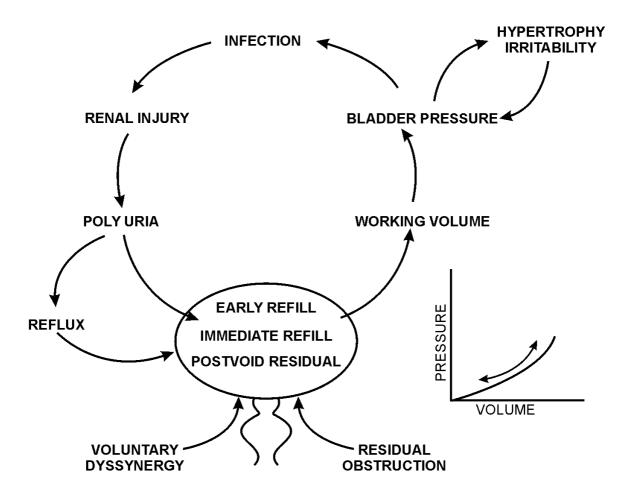
EVIDENCE FROM TRANSPLANTED KIDNEYS



The graph shows that renal transplant following PUV fare less well than other conditions



PATHO PHYSIOLOGY OF VALVE BLADDER 24



BLADDER DYSFUNCTION - VALVE BLADDER SYNDROME

Even after relief of obstruction, a significant number of patients will continue to have detrusor thickening and poor compliance. Manifestations of this problem include persistent ureteral dilatation associated with full valve bladder syndrome, a physiological obstruction of ureters associated with bladder filling.4,11,25 Even in the absence of reflux, persistent ureteral dilatation secondary to poor compliance of thickened bladder wall can be a source of alarm. When bladder is empty drainage from the ureters proceeds unimpeded. This abruptly changes with filling of the poorly compliant bladder. Impaired drainage of the upper tracts can be demonstrated with ultra sound, with relief upon voiding or catheterization. This valve bladder syndrome may be managed by complete

urinary tract emptying at least twice daily either with double or triple voiding regimen, or CIC along with timed voiding during the day. The use of anticholinergics to reduce the adverse effects of bladder and ureter is also desirable. Ureteral changes can be seen in approximately 30% of older valve patients. Attempts to improve the radiographic appearance by tapered re-implantation surgery are too often not with disheartening results when bladder dysfunction is ignored. That is why we had to augment one of our patients (ureterocystoplasty with mitrofanoff) and many are to follow⁵.

1. TREATMENT OF VALVE BLADDER

1. HYPOCOMPLIANT (EARLY POST FULGURATION)

POP OFF DIVERSIONS

NIGHT DRAINAGE

ANTICHOLINERGICS +/- CIC

2. HYPOCOMPLIANT (LATE)

AUGMENTATION

3. UNSTABLE

ANTICHOLINERGICS +/- CIC

4. MYOGENIC FAILURE

CIC AND NIGHT DRAINAGE

2. TREATMENT OPTIONS ACCORDING TO URODYNAMIC FINDINGS

- 1. BLADDER PRESSURE LESS THAN 20 cm $\rm H_20$ WITH VOLUME EQUAL TO 3-4 HR URINE OUTPUT THEN 3-4 HOURLY CIC WILL SUFFICE
- 2. LOW PRESSURE LEAKAGE

DRUGS + CIC

3. HYPERREFLEXIA

ANTICHOLINERGICS + CIC

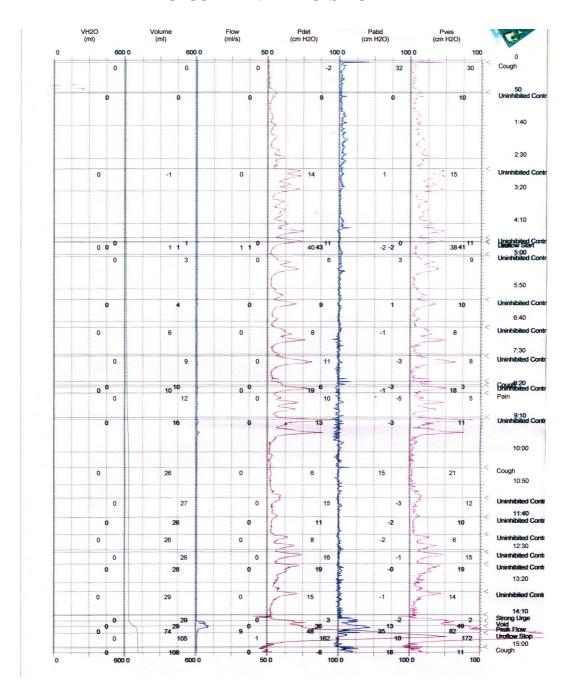
4. POOR COMPLIANCE HIGH PRESSURE LEAKAGE

AUGMENTATION + CIC

5. TOTALLY INCOMPETENT BLADDER NECK

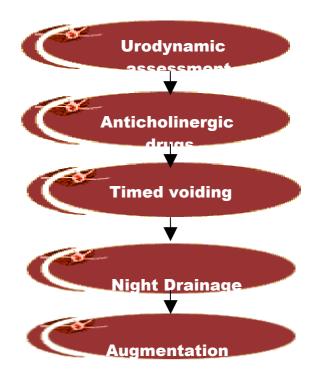
BLADDER NECK PROCEDURE + AUGMENT + CIC (MITROFANOFF)

URO DYNAMIC STUDY



Shows leak due to gross instability. This is also causing gross reflux. Hence bladder augmentation with reimplantation may be an option provided the Sr. creatinine remains stable (around 1.6 mg/dl now) and provided the patient is willing for self c.i.c..

LONG TERM BLADDER MANAGEMENT



URINARY INCONTINENCE

This is common event when there has been no bladder neck surgery or sphincter damage is seen. Our review has 25% of urinary incontinence mostly minor. Stricture or injury to external sphincter or bladder neck was not seen in our study. Although urge incontinence improves at time of puberty presumably secondary to prostatic growth, this is not always the case. Incontinence may be due to detrusor instability, increased urinary output (Fixed tubular concentrating defect secondary to previous obstruction) or inadequate sensation of a full bladder.

Using formal urodynamic studies 15% of valve population have bladder dysfunction, despite successful valve ablation. Bladder Hypertonia Hyperreflexia and Myogenic failure are the 3 main urodynamic findings and these may be overlapping in individual

patients.

Day time urinary incontinence after the age 5 has been a consistent clinical sign in one-third of patients with bad outcome regarding renal function. These findings serve as a warning for early attention to voiding dynamics. Simple relief of obstruction will alter the pathophysiology of the condition in most patients, but sophisticated intervention for the persistent effects caused by secondarily hyper trophied bladder may be necessary. Many of the method now used to control the neurogenic bladder are applicable including CIC, anticholinergics, bladder augmentation with bowel or more recently auto augmentation by creating a large bladder diverticulum to lower the high bladder pressure and night drainage. In our series one patient underwent augmentation with Mitrofanoff.

Patients with ESRD awaiting transplant must have their bladders evaluated carefully. Failure to address and correct under lying bladder dysfunction could result in ureteral obstruction, reflux, infection or even graft loss.

CHRONIC RENAL FAILURE

About 10% of patients with PUV will be stillborn or not survive the neonatal period. Ultimately upto 35% of children with PUV will develop some degree of CRF, generally associated with azotemia, anemia and acidosis. A more precise operational definition is renal insufficiency for more than 3 months with evidence of decreased GFR, anemia, osteodystrophy and electrolyte disturbances. However transplanted valve patient do not do well when compared to transplanted patients with other causes of renal failure. Abnormal bladder dynamics may play a role but clearly more study is warranted. In our study nearly 40 percent of our children developed chronic renal failure.

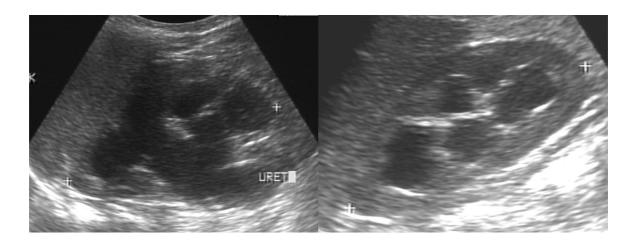
AUGMENTATION (URETEROCYSTOPLASTY) WITH MITROFANOFF PROCEDURE



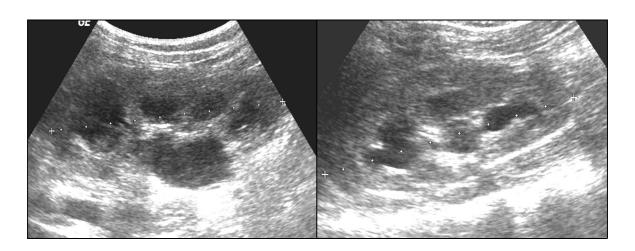
NIGHT DRAINAGE



ULTRASOUND – VOIDING PROGRAMME



BEFORE



6 MONTHS LATER

SEXUAL FUNCTION AND FERTILITY

Kruger reported an increased incidence of UDT in 12% of PUV patients. 2 patients had UDT in our study. A recent study indicates that the number of spermatogonia in fetuses with PUV and prune-belly syndrome are decreased when compare to a control group of normal fetuses. Wood house et al reported no evidence of testicular or sexual dysfunction in 20 adults originally treated for PUV. Hence there was a high incidence of retrograde ejaculation and poor ejaculatory force, suggesting persistent bladder neck incompetence. Previously undiagnosed PUV in adults have been reported in infertility patients. These studies emphasis sterility from retained ejaculate in dilated PU and symptoms of chronic prostatitis and bladder neck contracture in others. Adult PUV presentation remains infrequent.

LONG TERM

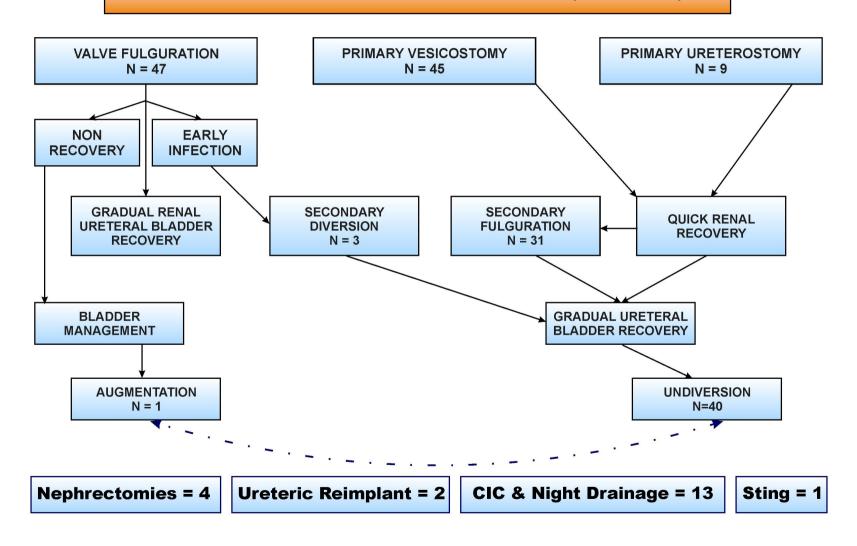
Prognosis depends upon

- 1. Presence of renal dysplasia before birth.
- 2. Status of upper tracts on admission.
- 3. Presence of infection.
- 4. Upon the presence or absence of VUR.

All infants and those older children with impaired renal function at presentation will require close supervision until adult life is reached. A progressive rise in serum creatinine is often seen during childhood and in most severe cases renal transplantation may be required before puberty. Persistent urinary incontinence is an indication for cystometrogram. Bladders showing severe hyperreflexia or very poor compliance may require augmentation prior to transplantation. At present none of our patients required renal transplantation.

In the antenatally diagnosed children in our study out of 8 patients, 7 of them had either mortality or morbidity in them. Only one patient is at present without problems. So the prognosis of antenatally detected patients has been dismal in our study.

A BIRD'S EYE VIEW OF LAST 104 CASES (1998-2007)



CONCLUSION

By means of this study the following conclusions were arrived at

- 1. Though technically demanding primary fulguration seems to be a promising method for managing PUV irrespective of age. The benefits of primary fulguration are:
 - a. Length of hospital stay is reduced
 - b. Cost effective
 - c. Lesser number of surgeries
 - d. Scarless
 - e. Parent's psychological trauma is lessened
- 2. We could avoid urinary diversion in 42.30 percentage of our cases.
- 3. Urosepsis and increased serum creatinine are not absolute contraindications for primary fulgurations
- 4. Outcome of patients with PUV depends upon initial renal status irrespective of management options used
- 5. So it is better to fulgurate as a single stage than going in for diversion which involves many stages
- 6. The prognosis of antenatally detected patients was dismal in our study.
- 7. Patients should be referred at the earliest to tertiary centres which have facilities for fulguration
- 8. Long term follow-up is a must going by the prognostic indicators with special attention to bladder dynamics.

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PROFORMA

| Name: | | Age : | I.P. No. | |
|----------------|--|---------------------|-------------------------|---|
| Symptoms: | Dribbling Failure to t Vomiting AN Detect Others | | D.O.A D.O.S D.O.D | : |
| Duration of Sy | ymptoms | : | | |
| Treatment Ob | tained : | Yes / No | | |
| AN History - | Delivery | : | | |
| Consar | nguinity | : | | |
| AN Sc | an | : | | |
| Significant Al | N problem: | | | |
| Family Histor | y | : | | |
| Examination | : | | | |
| Investigations | : | Bl. Urea | : | |
| | | S. Creatinin | e: | |
| | | Bl. Sugar | : | |
| | | USG Abd | : | |
| | | MCU | : | |
| Treatment Giv | ven : | Primary Fulguration | n | |
| | | Diversion | | |

Follow-up

| Bl.Urea | : |
|---------------------------|---|
| S.Creatinine : | |
| Hb% | : |
| Urine RE | : |
| Urine C/s | : |
| USG abd (Residual Urine): | |
| MCU | : |

Outcome:

Bl.Urea

MASTER CHART P.U.V PATIENTS

| S. NO | NAME | AGE | DOS | PRI. TRT. | SEC. TRT | OTHERS | OUTC OME |
|----------|------------------|---------|----------|------------------|-------------|--------------------------|-------------|
| 1 | B/o Manonmani | 2 mon. | 1-1-98 | b/l ureterostomy | | vur | expired |
| 2 | Raghavendran | 2 yrs | 28-3-98 | vesicostomy | Cyst.fulg | Seizures catheterised | ok |
| 3 | Sudarshan | 1 mon | 14-5-98 | vesicostomy | | | No fu |
| 4 | Daniel | 4.5 yrs | 26-3-99 | Rt ureterostomy | Cyst.fulg | | ok |
| 5 | B/o Poomany | 22 days | 24-4-99 | vesicostomy | | | No fu |
| 6 | Devaraj | 3yrs | 15-7-99 | vesicostomy | Cyst.fulg | | Ok |
| 7 | Joshua | 1yr | 30-4-99 | vesicostomy | Cyst.fulg | | ok |
| 8 | Gurupriya | 13 days | 13-8-99 | vesicostomy | | catheterised | No fu |
| 9 | Pommudurai | 6yrs | 31-8-99 | b/l ureterostomy | Cyst.fulg | Stricture | ok |
| | | | | | | Dilatation | |
| 10 | B/o rani | 30 days | 24-9-99 | vesicostomy | | catheterised | No fu |
| 11 | yoshua | 10 days | 20-8-96 | vesicostomy | Cyst.fulg | | ok |
| 12 | kumar | 6yrs | 29-10-99 | vesicostomy | Cyst.fulg | Stricture dilatation | ok |
| 13 | B/o vijaya | 4 days | 24-11-99 | vesicostomy | | | expired |
| 14 | arulmani | 10yrs | 26-11-99 | Cyst.fulg | | | ok |
| 15 | ramu | 5yrs | 30-5-00 | vesicostomy | Cyst.fulg | | ok |
| 16 | B/o krishnaveni | 15 days | 19-6-05 | | | Peritoneal dialysis | expired |
| 17 | hariharan | 8 mon | 12-8-00 | vesicostomy | | | No fu |
| 18 | murugan | 8yrs | 19-9-00 | b/l ureterostomy | Cyst.fulg | Stricture dilatation | ok |
| 19 | vadivelu | 7yrs | 13-10-00 | b/l ureterostomy | Cyst.fulg | Vur | ok |
| 20 | Pradeep kumar | 3yrs | 17-10-00 | vesicostomy | Cyst.fulg | | No fu |
| 21 | jeevanathan | 5yrs | 14-11-00 | vesicostomy | Cyst.fulg | | No fu |
| 22 | harinarayanan | 9yrs | 30-1-01 | Cyst.fulg | | crf | Ok |
| 23 | murugan | 7yrs | 21-6-01 | Cyst.fulg | | | Ok |
| 24 | B/o anandhi | 2 mon | 21-6-01 | vesicostomy | Cyst.fulg | catheterised | ok |

| 25 | Jeevanathan | 5yrs | 22-6-01 | vesicostomy | Cyst.fulg | | ok |
|----|-------------------|--------------|----------|------------------|------------|-------------------------|---------|
| 26 | Arunprasad | 9 mon | 4-7-01 | vesicostomy | Cyst.fulg | vur | ok |
| 27 | B/o Elizabeth | 7days | 14-7-01 | vesicostomy | | Antenatal diagnosis | expired |
| 28 | Kadar basha | 5yrs | 14-9-01 | b/l ureterostomy | Cyst.fulg | Vur | ok |
| 29 | Amen akbar | 12yrs | 18-9-01 | Cyst.fulg | | | ok |
| 30 | B/o sunitha | 4days | 4-12-01 | vesicostomy | | | Lost fu |
| 31 | Mohan | 2yrs | 11-12-01 | Cyst.fulg | | | ok |
| 32 | Binnikumar | 11month s | 14-1-02 | vesicostomy | | Vur | expired |
| 33 | Rahamatullah | 62days | 26-1-02 | vesicostomy | | Vur catheterised | expired |
| 34 | Sabi | 4yrs | 14-2-02 | Cyst.fulg | | | ok |
| 35 | Sanjeevi | 6months | 8-4-02 | vesicostomy | | Vur peri.dial. | expired |
| 36 | B/o Maragatham | 21days | 2-5-02 | vesicostomy | Cyst.fulg | | ok |
| 37 | B/o Maheswari | 41 days | 17-5-02 | b/l ureterostomy | Cyst.fulg | Fungal ball nephrectomy | ok |
| 38 | Sankar | 4 mon | 14-7-02 | vesicostomy | Cyst.fulg | Vur | Lost fu |
| 39 | Gunalan | 4 mon | 9-8-02 | vesicostomy | | Vur | expired |
| 40 | Premkumar | 4 mon | 30-8-02 | Cyst.fulg | | Catheterized vur | ok |
| 41 | Yuvan | 2.5 yrs | 3-9-02 | Cyst.fulg | | Catheterized vur | ok |
| 42 | Murugesan | 12yrs | 30-9-02 | Cyst.fulg | | Vur | ok |
| 43 | Gunasekaran | 10 mon | 20-9-02 | Cyst.fulg | | | ok |
| 44 | B/o Ellamal | 2 days | | | | | expired |
| 45 | Ramesh | 2.5 yrs | 22-10-02 | vesicostomy | Cyst.fulg. | | ok |
| 46 | B/o Madammal | 17 days | 30-10-02 | Cyst.fulg | | catherised | ok |
| 47 | B/o Tamilselvi | 1day | 13-11-02 | vesicostomy | | Antenatal diag. | Lost fu |
| 48 | Nagendran | 1yr | 11-12-02 | vesicostomy | Cyst.fulg | | Lost fu |
| 50 | Guna | 3.5 yrs | 30-5-03 | vesicostomy | | Peri.dial. | Lost fu |
| 51 | Anilkumar | 1yr | 3-6-03 | vesicostomy | Cyst fulg | hepatoblastoma | expired |
| 52 | B/o Karpayee | 12 days | 25-7-03 | vesicostomy | Cyst fulg | Epi.orchi | ok |
| 53 | Saravanan | 50 days | 1-8-03 | Cyst. fulg | | catheterised | ok |
| 54 | B/o suguna | 1day | 12-8-03 | Cyst fulg | | catheterised | Ok |

| 55 | Gokul | 30 days | 16-9-03 | Cyst fulg | | Type 3 | Ok |
|----|----------------------|---------|----------|------------------|-----------------|--------------------------------|---------|
| 56 | Binnykumar | 11 mon | 30-9-03 | vesicostomy | Cyst fulg | | ok |
| 57 | Raja | 3.5 yrs | 2-12-03 | vesicostomy | | Peri. dial | expired |
| 58 | Dhinu | 1.5 | 10-2-04 | Cyst fulg | | Peri dial catheterised | Lost fu |
| 59 | B/o Loganayagi | 4 mon | 10-3-04 | Cyst fulg | | calculi | ok |
| 60 | Vignesh | 1.5yr | 2-3-04 | Lt ureterostomy | Cyst fulg | Vur | ok |
| 61 | B/o Sarojini | 4 mon | 17-3-04 | Cyst fulg | | Catheterized ante.diag. | ok |
| 62 | B/o Krishnaveni | 28 days | 24-3-04 | Cyst fulg | | Spc ante.diag.peri.di al | Lost fu |
| 63 | Ganesan | 12yrs | 24-3-04 | Cyst fulg | | Catheterized -cvs -c rf | Lost fu |
| 64 | Guhan | 4 mon | 6-4-04 | Cyst fulg | vesicosto my | catheterised | Lost fu |
| 65 | Bharathi | 3 mon | 27-6-04 | vesicostomy | Cyst fulg | | Lost fu |
| 66 | Udhayakumar | 3 mon | 3-8-04 | Cyst fulg | | Type 3 | Ok |
| 67 | Om prakash | 2yrs | 20-7-04 | vesicostomy | Cyst fulg | | ok |
| 68 | B/o Kalaivani | 16 days | 24-9-04 | vesicostomy | Cyst fulg | Peri.dial. | Ok |
| 69 | B/o Padmapriya | 10 days | 14-12-04 | Cyst fulg | | | Ok |
| 70 | Lalith | 1yr | 17-12-04 | Cyst fulg | | Peri.dialcrf | Ok |
| 71 | Mohideen Basha | 1yr | 4-1-05 | Cyst fulg | | Vur | Ok |
| 72 | Joyal Vinith | 25 days | 5-1-05 | Cyst fulg | | | Ok |
| 73 | Mohamed Iqbal | 5 days | 19-1-05 | Cyst fulg | | | Ok |
| 74 | B/o Ranjanadevi | 6 days | 1-2-05 | Cyst fulg | | | ok |
| 75 | B/o Anish Fathima | 1 day | 2-2-05 | Cyst fulg | | Ante,diaging hernia | Expired |
| 76 | B/o Usha | 27 days | 1-2-05 | Cyst fulg | | | Ok |
| 77 | Akash | 1yr | 19-4-05 | b/l ureterostomy | Cyst fulg | | ok |
| 78 | Devaraj | 9yrs | 20-5-05 | vesicostomy | Cyst fulg | | Ok |
| 79 | Magesh Kannan | 8 mon | 10-5-05 | vesicostomy | Cyst fulg | | Ok |
| 80 | Wilson | 1.5yr | 15-7-05 | vesicostomy | valvotomy | Vur –p.d. | Ok |

| | | | | | | deafmute- cataract | |
|-----|-----------------------|---------|----------|------------------|---------------------|------------------------------------|---------|
| 81 | Vignesh | 2.5yrs | 9-8-05 | Cyst fulg | | | Ok |
| 82 | Pommudurai | 12yr | 12-8-05 | b/l ureterostomy | Cyst fulg | Str.dilreimpl nephrect | Ok |
| 83 | Mohan | 6 mon | 30-8-05 | vesicostomy | Cyst fulg | | expired |
| 84 | Thilak | 10yrs | 8-11-05 | Cyst fulg | | fogarty | Ok |
| 85 | Jay Pratap | 2yr | 22-11-05 | vesicostomy | Cyst fulg | Vur- ante.diag.nephre ct | Ok |
| 86 | Ashwin | 1.5yr | 16-12-05 | Cyst fulg | | b/l udt | Ok |
| 87 | B/o.Radha | 18 days | 27-12-05 | Cyst fulg | | Catheterized-p.d. | Ok |
| 88 | Arulmani | 2.5 yrs | 12-1-06 | vesicostomy | Cyst fulg | p.dcrf | Ok |
| 89 | Sankaran | 10yrs | 31-1-06 | vesicostomy | Cyst fulg | | Ok |
| 90 | B/o.Sowmya | 22 days | 14-2-06 | Cyst fulg | | Valvotome- vur-?vurd | Ok |
| 91 | B/o Abirami | 2 days | 10-1-02 | vesicostomy | | vur | Lost fu |
| 92 | B/o.Dhanalaksh mi | 1day | 7-3-06 | Cyst fulg | | Valvotome-vur- ant.diag. | Ok |
| 93 | B/o.Nagamani | 7 days | 2-5-06 | Cyst fulg | | | Ok |
| 94 | Gowrishankar | 10 mon | 29-8-06 | Cyst fulg | Sting tried reimpl. | Ureterocele- ant.diagvur | ok |
| 95 | Arunkumar | 5yrs | 1-12-06 | Cyst fulg | vesicosto my | Vur- catheterised | ok |
| 96 | B/o.Veerammal | 19 days | 2-1-07 | Cyst fulg | | Vur- catheterised- valvotome | ok |
| 97 | B/o.Maheswari | 10 days | 17-9-04 | Cyst fulg | | Vent.hernia | ok |
| 98 | B/o.Chamundes wari | 12 days | 21-6-94 | b/l ureterostomy | Cyst fulg | | ok |
| 99 | B/o.Sangeetha | 2 days | 22-4-05 | Cyst fulg | | Urachal cyst | expired |
| 100 | B/o Jaya | 17 days | 9-1-07 | Cyst fulg | | Valvotome- cathe | ok |
| 101 | Venkatesh | 4 mon | 2-2-06 | | | Cathp.d-crf. | expired |
| 102 | Krishnaveni | 26 days | 22-12-05 | | | crf | expired |
| 103 | Santhosh Kumar | 5yrs | 3-1-07 | ?cyst fulg | | | ok |

| | 104 | Dhandapani | 20 days | 29-12-94 | Vesicostomy | Cyst fulg | Augmentation- mitrofanoff- nephrectomy | ok |
|---|-----|------------|---------|----------|-------------|-----------|--|----|
| 1 | | | | | | | nepinectomy | |