POSTERIOR URETHRAL VALVE

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INTRODUCTION

- The first description of posterior urethral valves (PUVs) was made by Hugh Hampton Young.
- PUVs represent a spectrum of severity, ranging from disease incompatible with postnatal life to disease that is minimal and may not manifest until later in life
- Commenest cause of congenital bladder outlet obstruction
- Only occurs in males
- Incidence is 1 per 5000-8000 male births

Anatomy

- Embryology- Speculative

  The most caudal end of the Wolffian duct is absorbed into the primitive cloaca at the site of the future verumontanum in the posterior urethra.

  In healthy males, the remnants of this process are the posterior urethral folds, called plicae colliculi.

  These are missing in patients with PUV

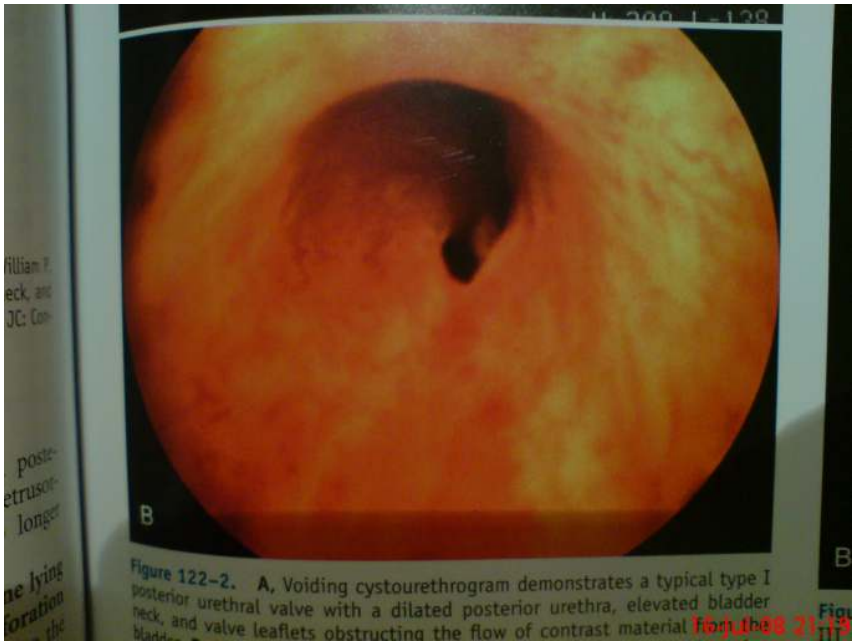
  Histological studies suggest that PUVs are formed at approximately 4 weeks' gestation, as the Wolffian duct fuses with the developing cloaca.

Gross anatomy

- Membranous valve like tissue
- Not functional but ‘structural’ in its disease causation.
- Cause passive obstruction
- Young et al initially categorized posterior urethral valves into 3 types.

Classification

- The type I valve –
- Bicuspid valve that radiates distally from the posterior edge of the verumontanum to the anterior proximal membranous urethra. There is a variable aperture to allow urine flow during voiding; however, the fused portion fills with urine and bulges into the membranous urethra.
- Sail-in-the-wind finding commonly seen on voiding cystourethrography (VCUG).
- Type I valves account for 95% of all valves.



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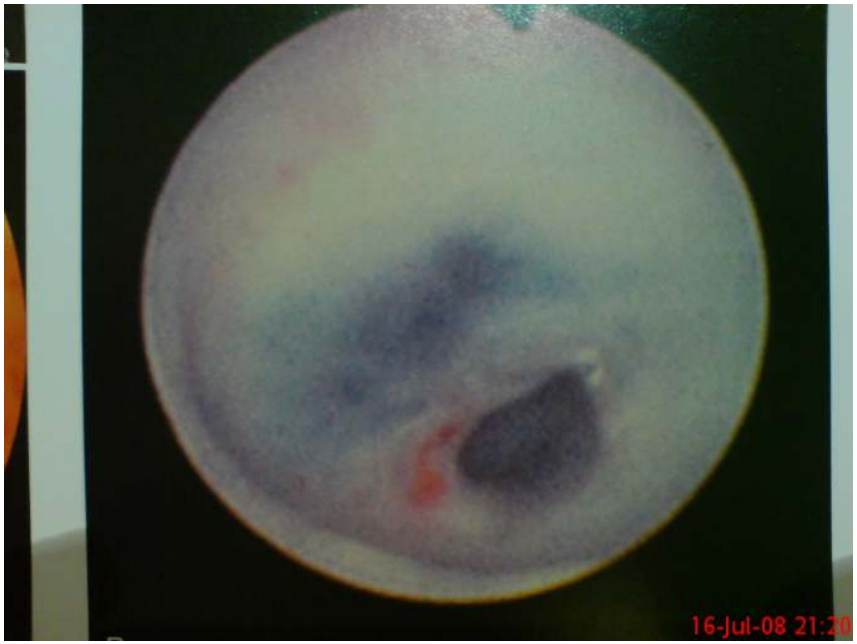


- The type III valve - Circumferential membrane or diaphragm that is located at the membranous urethra.
- There is a central aperture, and the central portions of the ring may prolapse into the more distal urethra during voiding.
- Wind-sock appearance on VCUG.
- Type III valves account for almost 5% of all valves.

- The type II valve is no longer considered an obstructing valve; rather, it is thought to be a sequela of voiding dysfunction.
- Despite all this a theory holds that there is most likely a single obstructive membrane that is altered by the passage of urethral catheters or cystoscopes, which results in variable tears of the membrane; these membranes may be perceived as a type I or type III valve. This concept of a single type of valve is referred to as **congenital obstructing posterior urethral membrane.**

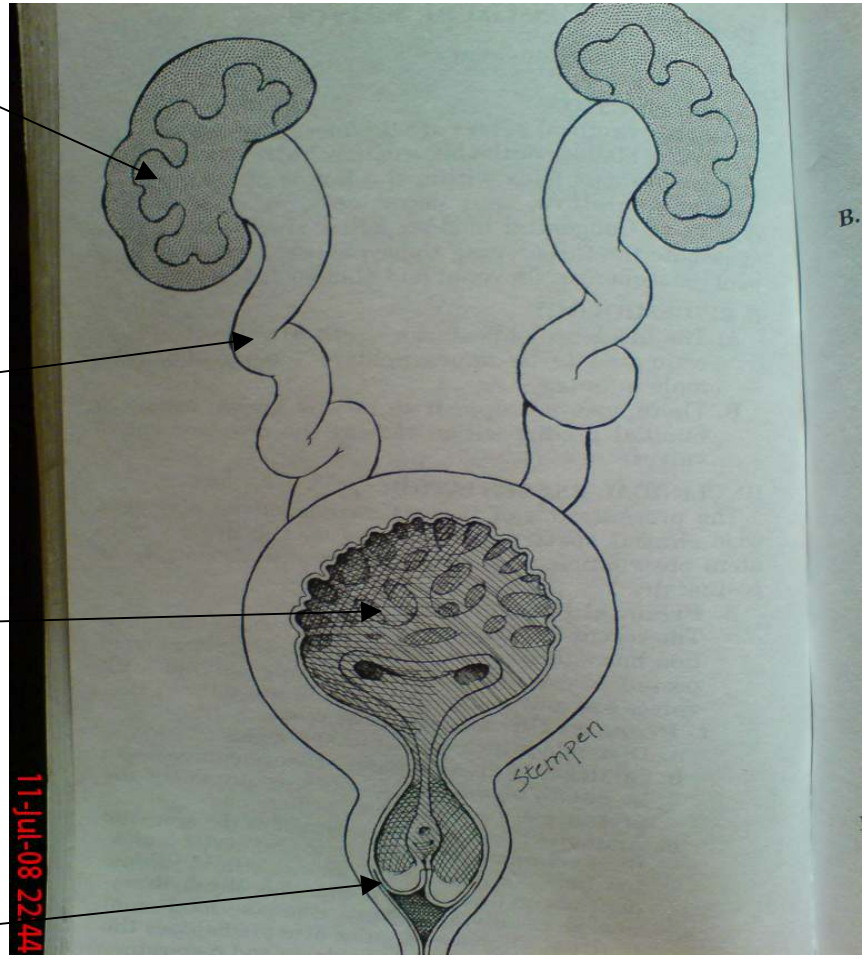
Pathophysiology

Hydronephrosis

Hydroureters

Diverticuli

Posterior urethral valves



Pathophysiology

PUV appear at early stage

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High intraluminal pressure due to mechanical obstruction

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May result in permanent damage of kidneys,ureters and bladder due to abnormal prenatal dev.

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The compenatory mechanism themselves i.e. VUR and diverticula may complicate things further.

RENAL FAILURE

Renal parenchymal dysplasia

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Maldevelopment of metanephric blastema

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Renal tubular dysfunction 2nd to poor concentration ability leading to diuresis

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Bladder and urethral dysfunction due to increased amount of urine

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Chronic pyelonephritis a/w VUR, Glomerulosclerosis etc

BLADDER DYSFUNCTION

- Thought to be caused by alterations in collagen deposition and the development of detrusor smooth muscle cells.

  → Myogenic failure.

  → In mild cases, incontinence may be present.

  → In severe cases, ongoing deterioration of renal function occurs.

LUNGS

- Can cause pulmonary hypoplasia
- Main cause of neonatal death!

REFERANCES
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