Obstructive Uropathy: Diagnosis and Management of BPH, Neurogenic Bladder, and Hydronephrosis

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Learning Objectives

By the end of this session, students will be able to: - Evaluate benign prostatic hyperplasia (BPH) and lower urinary tract symptoms - Differentiate obstructive uropathy causes (lower vs. upper urinary tract) - Recognize and manage neurogenic bladder in various neurologic conditions - Assess hydronephrosis and determine clinical significance - Diagnose acute kidney injury without hydronephrosis (NDOU) - Manage post-obstructive diuresis safely - Identify indications for nephrostomy tube or ureteral stent placement - Recognize urologic malignancy presentations and initial management

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I. Benign Prostatic Hyperplasia (BPH) and Lower Urinary Tract Obstruction

Epidemiology and Pathophysiology

Epidemiology: - **Prevalence:** 25% of men age 40–49; 50% by age 60; >80% by age 80 - **Symptomatic BPH:** 10–15% of men age 50; increases with age - **Risk factors:** Age, male sex, testosterone, family history, metabolic syndrome

Pathophysiology: - Age-related hyperplasia of prostatic stromal and epithelial tissue - ☐ Dihydrotestosterone (DHT) ☐ androgen-receptor activation ☐ cellular growth - ☐ Smooth muscle tone (α_1-adrenergic) ☐ dynamic obstruction - Static obstruction from enlarged gland + dynamic obstruction from smooth muscle = total resistance

Clinical Presentation

Lower Urinary Tract Symptoms (LUTS):

Symptom Category	Examples	Mechanism
Obstructive (Voiding)	Weak stream, hesitancy, incomplete emptying, straining to void, prolonged voiding	Outlet obstruction from enlarged prostate
Irritative (Storage)	Frequency, urgency, nocturia (>2×/night), dysuria	Bladder irritation from outlet obstruction and/or detrusor overactivity
Post-void residual	Dribbling, incontinence after standing	Incomplete bladder emptying

Severe symptoms can include: - Urinary retention (acute or chronic) - Upper tract involvement (AKI, hydronephrosis from back-pressure) - Recurrent UTIs - Bladder stones - Hematuria (usually minor; can be massive if bleeding from prostate)

Assessment Tools

International Prostate Symptom Score (IPSS) A validated 7-question symptom assessment (each scored 0–5) plus 1 quality-of-life question:

Question	Scoring	Mild (0–7)	Moderate (8–19)	Severe (20–35)
Incomplete emptying	0–5	Mild sx	Moderate sx	Severe sx
Frequency	0–5	<2 voids/day	2–4 times	>7 times
Intermittency	0–5	±	Frequent	Nearly always
Urgency	0–5	Mild	Moderate	Severe
Weak stream	0–5	Adequate	Weak	Very weak
Straining	0–5	Never	Sometimes	Most of time
Nocturia	0–5	≤1/night	2–4/night	>5/night
Total Score	0–35	Mild	Moderate	Severe

Use: IPSS guides treatment decisions (see Section I.D below).

Uroflowmetry

- Non-invasive measure of urine flow rate
- Normal flow: Peak flow 15–25 mL/sec; total voided volume >200 mL
- Reduced flow (<10 mL/sec) suggests obstruction
- Useful for documenting baseline before treatment

Post-Void Residual (PVR) Measurement

- **Ultrasound:** Non-invasive, accurate, operator-dependent
- **Catheterization:** Invasive but confirms obstruction; volume >100–150 mL abnormal
- **Elevated PVR:** Risk factor for retention, UTI, upper tract deterioration

Diagnostic Approach

History and Physical: - Symptom duration and severity (IPSS scoring) - Comorbidities affecting LUTS (diabetes, neurologic disease, medications) - Medications contributing to LUTS (anticholinergics, antihistamines, sympathomimetics, opioids) - Digital rectal exam (DRE): Prostate size (not well correlated with symptoms), nodules, hardness

Laboratory and Imaging: - **Urinalysis and culture:** Rule out UTI/hematuria - **Serum creatinine:** Assess renal function; elevated suggests upper tract involvement - **PSA (prostate-specific antigen):** Discussed but controversial; individualized decision based on life expectancy and patient preferences - **Ultrasound or bladder ultrasound:** Measure PVR - **Renal ultrasound:** If elevated creatinine or hematuria (screen for hydronephrosis, masses) - **Urodynamic testing (NOT routine):** Reserved for complex cases (mixed symptoms, failed treatment, preop evaluation for certain surgeries)

Management Strategy

Step 1: Patient Education and Lifestyle Modification Recommended for all patients with LUTS, especially mild-to-moderate symptoms:

Intervention	Mechanism
☐ **Fluid intake (especially evening)**	☐ Nocturia; avoid caffeine/alcohol
Timed voiding	Prevent post-void residual; improve bladder emptying
Perineal massage/double void	Facilitate complete emptying
Avoid NSAIDs, anticholinergics, OTC cold medicines	These worsen LUTS
Physical activity	☐ LUTS progression

Efficacy: 30–40% of men improve with lifestyle alone, especially mild symptoms.

Step 2: Pharmacologic Therapy (if lifestyle modification insufficient or moderate-severe LUTS) Alpha-1 Adrenergic Antagonists (α1-blockers) — First-line medical therapy

Mechanism: Block α1A-adrenergic receptors on prostatic smooth muscle ☐ relaxation ☐ ☐ dynamic obstruction

Agent	Dose	Half-life	Notes
Terazosin	1–10 mg daily	12 hours	Older agent; risk of syncope with first dose
Doxazosin	1–8 mg daily	22 hours	Older; risk of syncope; ☐ BP
Tamsulosin	0.4–0.8 mg daily	5–7 hours	Selective for α1A (less ☐ BP); preferred agent

Agent	Dose	Half-life	Notes
Alfuzosin	10 mg daily	10 hours	Uroselective; fewer orthostatic effects
Silodosin	8 mg daily	13 hours	Most uroselective; used less commonly

Efficacy: 30–50% reduction in IPSS; improves symptom score by 3–5 points; effect seen in 1–2 weeks

Side effects: Orthostatic hypotension, dizziness, headache, retrograde ejaculation (10–30%); rare: intraoperative floppy iris syndrome (IFIS) if cataract surgery needed

Contraindications: Severe hepatic disease; concurrent use with other α-blockers

Advantage over 5-ARIs: Faster onset of action (days vs. months)

5-Alpha Reductase Inhibitors (5-ARIs) — For larger prostate or at risk for progression **Mechanism:** Block 5-alpha reductase ☐ ☐ DHT ☐ ☐ prostatic epithelial growth

Agent	Dose	Selectivity	Half-life	Efficacy
Finasteride	5 mg daily	Type II reductase	6 hours	20–30% prostate reduction over 6–12 months
Dutasteride	0.5 mg daily	Type I + II reductase	5 weeks	25% prostate reduction; more potent

Efficacy: 20–30% reduction in IPSS; prostate volume reduction 20–30% over 12 months; takes 3–6 months for symptom improvement

Side effects: Erectile dysfunction (10–25%), ☐ libido (5–15%), gynecomastia (0.5%); ~50% ☐ PSA (affects cancer detection)

Indications for 5-ARIs: - Large prostate (>30 g on imaging) with moderate-severe symptoms - Risk of progression (age >60, elevated PSA, positive family history) - Recurrent UTI from outlet obstruction - Recurrent hematuria (may improve bleeding)

Comparison: α-blocker vs. 5-ARI: - **α-blocker:** Faster symptom relief; more side effects; all men benefit - **5-ARI:** Slower onset; fewer side effects; mainly benefit men with large prostate - **Combination therapy:** Superior symptom relief and reduced retention risk than either alone in men with large prostate and moderate-severe LUTS

Phosphodiesterase-5 Inhibitors (PDE-5i) **Agents:** Tadalafil (Cialis) 5 mg daily approved for LUTS/BPH

Mechanism: Smooth muscle relaxation; increases cGMP (improves vascular function and reduces obstruction)

Efficacy: 4–5 point IPSS improvement; less than α-blockers; benefit if concurrent ED

Use: Second-line; added to α-blocker or used alone if ED is primary complaint

Side effects: Headache, dyspepsia, myalgia; caution with nitrates (contraindicated)

Combination Medical Therapy **Indicated if:** - Moderate-severe LUTS despite monotherapy (3–4 months trial) - Large prostate (>30 g) with obstructive symptoms - Recurrent retention or UTI despite monotherapy

Efficacy: α-blocker + 5-ARI superior to either alone; reduces symptom progression and acute retention risk by ~30–50%

Step 3: Surgical/Interventional Management (if medical therapy fails or complications develop) **Indications for Intervention:** 1. **Refractory LUTS:** Minimal response to optimal medical therapy (IPSS still >19) 2. **Acute/chronic urinary retention:** Unable to void spontaneously 3. **Recurrent UTIs:** Due to outlet obstruction (>2 UTIs/year) 4. **Renal insufficiency:** AKI from outlet obstruction 5. **Large bladder stones:** Secondary to obstruction 6. **Recurrent hematuria:** Despite 5-ARI trial 7. **Patient preference:** After counseling on risks/benefits of medical vs. surgical

Surgical Options:

Procedure	Description	Success Rate	Recovery	Notes
TURP (Transurethral Resection of Prostate)	Endoscopic resection of prostate tissue via cystoscope	90% short-term relief	2–4 weeks	Gold standard; higher morbidity (TURP syndrome risk if prolonged resection); retrograde ejaculation
Laser vaporization (TUVP, Photoselective vaporization)	KTP or thulium laser ablation of prostate	85–90% relief	1–2 weeks	Less TURP syndrome risk; shorter operative time; good for anticoagulated patients

Procedure	Description	Success Rate	Recovery	Notes
Laser enucleation (HoLEP)	Holmium laser enucleation + morcellation	90–95% relief	Similar to TURP	Excellent durability; steep learning curve; best for very large prostates
Transurethral Microwave Thermotherapy (TUMT)	Microwave heating of prostate tissue	70–75% relief	Outpatient	Minimal invasiveness; lower efficacy; useful if poor surgical candidate
Transurethral Needle Ablation (TUNA)	Radiofrequency needle ablation	65–70% relief	Outpatient	Minimal invasiveness; lower efficacy
Robotic-Assisted Simple Prostatectomy	Open or robotic removal of entire prostate gland	95%+ relief	2–4 weeks	Excellent for very large glands (>100 g); more invasive; reserved for selected patients
Urethral Stent	Permanent mesh stent in urethra	70–80% relief	Variable	Palliative; option for poor surgical candidates; risk of stent migration, encrustation

Patient Counseling on Surgical Side Effects: - **Retrograde ejaculation:** 10–30% after

TURP/laser (fertility impact) - **Erectile dysfunction:** 5–10% (usually temporary) - **Incontinence:** <5% (usually transient) - **Urinary retention:** 5–10% in early post-op (usually resolves with catheterization) - **Reoperation:** 15–25% within 10 years (depends on procedure, gland size) - **TURP syndrome:** Rare (<1%) but serious (hyponatremia, seizure, pulmonary edema) if prolonged resection

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II. Neurogenic Bladder

Pathophysiology and Classification

Definition: Loss of normal bladder function due to neurologic disease affecting spinal cord, nerve roots, or peripheral nerves.

Classification by Neurologic Level:

Neurologic Lesion	Bladder Pattern	Characteristics	Upper vs. Lower Motor Neuron
Spinal cord above S2–S4 (e.g., transection, MS, myelopathy)	Spastic/Reflex	Uninhibited detrusor contraction; no voluntary control; ☐ residuals	Upper motor neuron (UMN)
Spinal cord at/below S2–S4 (e.g., spinal dysraphism, cauda equina)	Flaccid/Atonic	Weak/absent detrusor contraction; large PVR; ☐ pressure	Lower motor neuron (LMN)
Peripheral nerves (e.g., diabetes, pelvic injury)	Autonomous	Variable; depends on which nerves affected	LMN

Etiologies of Neurogenic Bladder

Common Causes: 1. **Spinal cord injury (SCI):** From trauma, ischemia, inflammation 2. **Spinal dysraphism:** Tethered spinal cord, meningomyelocele 3. **Multiple sclerosis:** 50–90% develop neurogenic bladder over time 4. **Parkinson's disease:** ☐ bladder pressure, poor relaxation, retention 5. **Cauda equina syndrome:** Compression of nerve roots 6. **Peripheral neuropathy:** Diabetes (sensory loss ☐ urinary retention) 7. **Trauma:** Pelvic fracture, birth injury (obstetric fistula) 8. **Tumor:** Spinal cord compression 9. **Tethered spinal cord:** Restricted spinal movement 10. **Surgical injury:** Hysterectomy, colorectal surgery (nerve injury)

Clinical Presentation

Symptoms vary by underlying physiology:

Symptom	Spastic/Reflex Bladder	Flaccid/Atonic Bladder
Continence	Often present; reflex leakage	Usually absent; continuous leakage
Voiding	Spontaneous (triggered by stimulation)	Absent; requires catheterization or Valsalva
Sensation	Absent	May have some sensation depending on level
Post-void residual	Moderate-large (200–500 mL)	Very large (500–1500 mL)
Infection risk	Moderate	Very high
Upper tract risk	High if high pressures	Moderate if well-managed

Complications of neurogenic bladder: - Recurrent UTI/pyelonephritis - Hydronephrosis and renal failure (from high pressures or obstruction) - Bladder stones - Vesicoureteral reflux (VUR) - Bowel dysfunction (neurogenic bowel if same level injury)

Management Approach

Goals: 1. Preserve renal function 2. Maintain continence 3. Prevent UTI/sepsis 4. Improve quality of life

Step 1: Assessment Key tests: - **Urinalysis and culture:** Rule out UTI (even if asymptomatic, if fever/systemic symptoms) - **Serum creatinine:** Assess renal function - **Ultrasound or CT:** Screen for hydronephrosis, bladder stones, residual urine volume - **Urodynamic testing (cystometrography):** Measures bladder compliance, capacity, leak point pressures - Critical for determining risk of upper tract deterioration - **Safe parameters:** Capacity >300 mL, compliance normal, leak point pressure <20 cm H_2O - **Unsafe parameters:** Compliance <25 mL/cm H_2O, leak point pressure >20–40 cm H_2O (risk of reflux/hydronephrosis)

Step 2: Intermittent Catheterization (CIC) — Preferred first-line Mechanism: Regular (4–6× daily) sterile catheterization empties bladder completely, mimicking normal voiding

Advantages: - Preserves bladder capacity and compliance - ☐ Infection risk vs. indwelling catheter - Maintains continence between caths - Better quality of life than indwelling catheter - Lower cost long-term

Technique: - Sterile or clean technique (clean more practical at home) - Size 12–16 Fr catheter typically - Timing: Every 4–6 hours during day; once at night if needed - Patient-taught; good for cognitive/manual dexterity

Complications: - Urethral trauma (15–25%); hematuria, false passages, strictures - UTI if technique contaminated (10–20% annually) - More common in women than men

Modifications: - **Suprapubic catheterization:** If can't self-cath (severe hand weakness, high-level SCI); avoids urethral trauma - **Mitrofanoff procedure:** Surgical creation of continent catheterizable channel using appendix (provides continent reservoir)

Step 3: Pharmacologic Therapy (Adjunct to CIC) Anticholinergic agents (if bladder spasticity or hyperreflexia causing leakage between catheterizations):

Agent	Dose	Effect	Notes
Oxybutynin IR	2.5–5 mg q6h PO	☐ detrusor contractions	Immediate effect; anticholinergic side effects (dry mouth, constipation)
Oxybutynin ER	10–20 mg daily	Sustained delivery	Better tolerated; fewer dose-related effects
Oxybutynin topical gel	10% gel daily	Local effect	Avoids systemic side effects; good for sensitive patients
Trospium chloride	20 mg BID	Long-acting anticholinergic	Less CNS penetration; avoided in renal failure
Mirabegron	25–50 mg daily	β3-agonist; increases compliance	No anticholinergic side effects; good alternative if constipation issue
Botulinum toxin	100–200 units intra-detrusor	Paralyzes detrusor muscle	Lasts 3–6 months; repeated injections needed; excellent for spasticity

Indications: Significant spasticity causing leakage, high-pressure bladder, inadequate capacity despite CIC

Step 4: Surgical Intervention (if conservative management inadequate) Indicated for: - High-pressure bladder unresponsive to anticholinergics/botox - Severe sphincter incompetence causing incontinence - Large post-void residuals despite CIC - Recurrent UTI despite optimal CIC - Upper tract deterioration (hydronephrosis, reflux)

Options: - **Bladder augmentation (enterocystoplasty):** Add bowel segment to bladder ☐ ☐ capacity, ☐ pressure - Reduces upper tract complications - Requires lifelong CIC; risk of stone formation, malignancy - **Ileal conduit (ureterosigmoidostomy):** Diversion of ureters to ileal segment via stoma - For very high-pressure bladders or severe incontinence refractory to other measures - Morbidity risk; need external pouch - **Continent catheterizable pouch (Mitrofanoff):** Use appendix/other tissue to create continent channel for CIC - Excellent QoL improvement - Technically complex

Special Populations with Neurogenic Bladder

Spinal Cord Injury: - Acute phase: Often retention; may need indwelling catheter temporarily - Chronic phase: Transition to CIC best practice - Autonomic dysreflexia risk: If bladder overdistension ☐ severe HTN, bradycardia; medical emergency

Multiple Sclerosis: - Variable presentation depending on MS type and lesion location - Often presents with frequency/urgency; may progress to retention - Anticholinergics, CIC as above - Re-assess frequently as MS progresses

Tethered Spinal Cord: - Surgical correction (detethering) can halt/reverse neurogenic bladder if caught early - Earlier treatment = better outcomes - Loss of motor/sensory function may be irreversible if severe/prolonged

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III. Hydronephrosis and Upper Urinary Tract Obstruction

Definition and Grading

Hydronephrosis (HN): Dilation of renal pelvis and/or calyces

Etiology: Result of obstruction of urine flow OR vesicoureteral reflux, but not all HN = obstruction

Imaging-Based Grading (Ultrasound/CT):

Grade	Ultrasound Findings	Clinical Significance	Action
0	Normal	No obstruction	Reassurance
1 (Mild)	Renal pelvis dilation only; calyces normal	Minimal obstruction; usually asymptomatic	Observe
2 (Moderate)	Renal pelvis + minor calyceal dilation	May indicate obstruction; depends on clinical context	Assess renal function; imaging f/u in 3–6 months
3 (Severe)	Major and minor calyceal dilation; renal sinus enlargement	Significant obstruction; risk of renal damage	Assess renal function; consider intervention
4 (Grade 4+)	Massive dilation; renal parenchymal thinning; loss of corticomedullary differentiation	Likely chronic obstruction; significant renal damage	Urgent imaging; assess renal function; likely intervention needed

Common Causes of Obstructive Hydronephrosis

Upper Urinary Tract Obstruction (Unilateral): - **Kidney stones** (50% of obstruction) - **Ureteropelvic junction (UPJ) obstruction** (congenital or acquired) - **Ureteral stricture** (post-surgical, radiation, infection, trauma) - **Ureteral cancer** - **External compression:** Retroperitoneal fibrosis, pelvic mass, AAA

Lower Urinary Tract Obstruction (Bilateral or single kidney): - **BPH** (common; see Section I) - **Neurogenic bladder** (see Section II) - **Urethral stricture** - **Bladder**

cancer/prostate cancer - **Pelvic mass** compressing urethra

Non-Obstructive Hydronephrosis: - **Vesicoureteral reflux (VUR):** Retrograde flow of urine into ureters - **Pregnancy:** Physiologic dilation from ureteral relaxation + hormones; "physiologic hydronephrosis" - **Diabetes insipidus:** Large urine volume ☐ mild dilation - **Polycystic kidney disease:** Functional obstruction

Clinical Approach to Hydronephrosis

History and Exam: - Flank pain (suggests acute obstruction, stone) - LUTS (suggests lower tract obstruction) - Renal insufficiency or AKI (suggests significant/bilateral obstruction) - Fever (suggests infected obstructed system — **urologic emergency**) - CVA tenderness - Palpable bladder (outlet obstruction with retention)

Diagnostic Workup: 1. **Non-contrast CT or ultrasound:** Confirm HN; identify stone, mass, or other cause 2. **Serum creatinine:** Assess renal function 3. **Urinalysis:** Hematuria, pyuria, crystals 4. **Urine culture:** If fever/pyuria 5. **Post-void residual measurement** (if outlet obstruction suspected) 6. **Diuretic renal scan (Mag-3):** Controversial; helps differentiate obstructive HN from non-obstructive - Useful if imaging shows HN but clinical picture unclear - Assesses split renal function (important for surgery decisions)

Management

Acute Obstruction (stone, infected system, AKI): - Urgent decompression (ureteral stent or nephrostomy) if infected, in sepsis, or severe AKI - Pain control, antibiotics if infected - See Nephrolithiasis section (handout part 1) for stone management

Chronic/Subacute Obstruction without Infection or AKI: - Depends on cause and renal function - Regular monitoring (ultrasound q3–6 months, creatinine) - Intervention if: (1) Progressive renal function decline, (2) Recurrent infection, (3) Symptomatic, or (4) Obstruction confirmed on diuretic scan

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IV. Acute Kidney Injury Without Hydronephrosis (NDOU)

Definition and Pathophysiology

NDOU (Non-Dilated Obstructed Uropathy): AKI from urinary obstruction **without ultrasound evidence of hydronephrosis**

Why no HN despite obstruction? - **Acute, complete obstruction:** Before dilation develops (takes hours–days) - **Retroperitoneal/pelvic mass obstruction:** Ureters compressed externally; may not dilate - **Severe bilateral ureteral strictures:** Partial obstruction; not enough pressure to cause significant dilation - **Pyonephrosis:** Infected, obstructed kidney; infected material may prevent dilation - **Posterior urethra obstruction (males):** Lower tract obstruction ☐ filled bladder compresses ureters; ☐ HN

Clinical Presentation

Often missed because "normal ultrasound" falsely reassures clinicians

Classic scenario: - Patient with AKI + rising creatinine - Ultrasound ordered, shows **no hydronephrosis** - Clinician assumes renal cause (prerenal, intrinsic) and delays urology consult - Renal function deteriorates

Red flags for NDOU despite normal HN: - Bilateral AKI with normal ultrasound - AKI + urinary catheter placed acutely - AKI + history of pelvic surgery/radiation/mass - Acute ☐ creatinine + large post-void residual - Sepsis without apparent source + AKI + elevated lactate (pyonephrosis)

Diagnostic Approach

CT is superior to ultrasound for NDOU: - Shows ureteral caliber (dilated = obstruction; narrow = stricture) - Identifies masses, fibrosis, other causes - Hydration on CT can artificially create HN (overdiagnosis)

Diuretic renal scan (Mag-3): - Can show obstruction without HN - Helpful if CT equivocal

Urinalysis and culture: - If infected ☐ pyonephrosis (emergency)

Management

Immediate actions if NDOU suspected: 1. Remove urinary catheter if just placed (often precipitates obstruction) 2. Gentle bladder scan or catheterization for PVR 3. Hydrate carefully (may improve renal perfusion; avoid overload if retention) 4. Urology/IR consult for intervention if obstruction confirmed

Intervention (ureteral stent or nephrostomy) indicated if: - Bilateral obstruction + AKI - Obstruction to solitary kidney + AKI - Infected obstructed system (pyonephrosis) - Symptomatic or progressive renal function loss

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V. Post-Obstructive Diuresis (POD)

Pathophysiology

Definition: Excessive urine output (often >200–400 mL/hr) occurring after relief of bilateral urinary obstruction or obstruction to solitary kidney

Mechanisms (usually multiple):

1. **Osmotic diuresis:**
   - Retained urea, creatinine, electrolytes accumulate during obstruction
   - After relief, filtered at once ☐ high osmolar load
   - Osmolarity » normal ☐ osmotic diuresis (similar to hyperglycemic diuresis in DKA)
2. **Extracellular fluid (ECF) expansion:**
   - During obstruction, fluid retained ☐ ECF volume ☐
   - ANP release + decreased proximal tubule reabsorption
3. **Tubular dysfunction:**
   - Obstructed kidneys have ☐ collecting duct sensitivity to ADH
   - Reversible nephrogenic DI-like state
   - Takes days–weeks to resolve
4. **Natriuresis:**

- ☐ GFR recovery + ☐ tubular reabsorption of Na
- Leads to continued Na wasting

Clinical Presentation

Timing: Usually begins within hours of obstruction relief; peaks at 24–48 hours; resolves within 1–7 days (rarely longer)

Symptoms: - Massive urine output (1–3 L/hr possible, though rare) - Polyuria, thirst - Dehydration despite high output (if fluid intake not matched) - Orthostatic hypotension, tachycardia, weak - Electrolyte abnormalities (hyponatremia, hypokalemia, hypophosphatemia)

Dangerous scenarios: - **Hypovolemia:** If excessive diuresis not matched by fluid intake - Risk: AKI recurrence, hypotension, syncope, cardiac arrhythmia - **Electrolyte derangement:** Rapid K+, Na, or PO_4 loss - Risk: Cardiac arrhythmia, seizure, respiratory muscle weakness - **Cerebral edema:** Rapid hyponatremia (paradoxically, initial HN relief causes rapid fluid shifts)

Management of POD

Key principle: Monitor closely; avoid aggressive IV hydration but ensure adequate oral intake

Fluid Management **Goal:** Balance urine output to prevent both hypovolemia AND fluid overload

Monitoring: - **Urine output q1–2h initially** (especially first 24–48 hours) - **Daily weight** (goal: stable or modest loss) - **Vital signs q4h:** Watch for orthostasis - **Serum electrolytes q4–12h** (especially in first 24 hours) - **Creatinine** to ensure improving

IV fluid strategy: - **NOT standard:** Avoid routine IV hydration (contributes to overload) - **IF needed:** Only for hypotension/symptomatic dehydration - Use 0.5 NS at rate = 50% of prior hour's urine output - This allows gradual rehydration without worsening ECF overload - Discontinue IV fluids once PO intake adequate - **Preferred:** Encourage PO intake (water, sports drinks for electrolytes)

Electrolyte Management

Electrolyte	Normal POD Response	Management
Sodium	☐ (Na loss exceeds water loss initially)	Monitor; restrict free water if Na <125 (risk of seizure); oral saline if symptomatic hyponatremia
Potassium	☐ (K loss in diuresis)	Replace orally (K+ drink, banana, OJ); check levels q12h; risk of arrhythmia if K <3.0

Electrolyte	Normal POD Response	Management
Phosphate	☐ (PO_4 loss)	Usually mild; phosphate supplementation rare; monitor
Magnesium	☐ (Mg loss)	May need supplementation (oral or IV); check level
Calcium	May ☐ (in relief diuresis)	Usually self-limited

When to Restrict Fluids (Rare)

- Only if hyponatremia (Na <125 mEq/L) developing acutely
- Risk of seizure or cerebral edema
- Fluid restriction 1–1.5 L/day
- Monitor Na hourly; correct slowly (8 mEq/L per 24 hours max to avoid central pontine myelinolysis)

Medications Generally NOT recommended but may be used if:

Medication	Indication	Dose	Caution
NSAIDs	Excessive POD (>500 mL/hr sustained) + euvolemia	Indomethacin 25–50 mg TID	Inhibits prostaglandin-mediated diuresis; watch renal function
Spironolactone	Persistent hyperkalemia (rare in POD)	Usually not needed	Counteracts diuresis

Avoid: - **Loop diuretics:** Worsen electrolyte losses - **Aggressive IV hydration:** Perpetuates diuresis and ECF overload

Monitoring Duration

POD typically resolves within 3–7 days: - Urine output gradually decreases - Electrolytes stabilize - Creatinine plateaus - Patient can be discharged once stable and tolerating PO intake

Extended POD (>2 weeks): - Suggests incomplete obstruction relief - Or persistent renal dysfunction - Consider imaging to confirm obstruction removed - May need urology re-evaluation

VI. Indications for Nephrostomy Tube Placement

When Nephrostomy Is Indicated

Acute/Emergent (infection/sepsis): - Infected obstructed system (pyonephrosis) with sepsis - Untreated urosepsis with source not identifiable by other means - **Timing:** ASAP; before antibiotics if possible

Subacute/Chronic: - Obstructed kidney where ureteral stent placement not possible (failed attempt, severe stricture, ureter not crossed) - Extrinsic obstruction (mass compression) not amenable to stent - Post-operative management (after kidney stone PCNL) - Planned delayed treatment (e.g., patient unstable for definitive urology procedure) - Diversion before nephrostomy + PEG (for incontinent patient + feeding issues)

Permanent/Long-term: - Non-functioning obstructed kidney (renal function <15%) where patient refuses nephrectomy - Obstructed transplanted kidney (temporary bridge) - Palliative care for patient with metastatic cancer + bilateral obstruction

Technique

Placement: - IR guided (ultrasound or CT fluoroscopy) - Percutaneous puncture of renal calix - Guidewire advanced; tract dilated; pigtail catheter placed retrograde through renal pelvis, ureter

Post-placement care: - Secure with sutures/adhesive - Drain to external bag or internal stent (D-J stent) - Flushing q4–8 hours (10 mL sterile saline) to prevent clotting - Monitor output (color, volume) - Regular site care (dressing changes, clean technique)

Complications: - Bleeding (5–10%; usually minor) - Infection/sepsis (10–15% risk if long-term) - Catheter obstruction/migration - Urinoma (if catheter dislodges) - Chronic pain/irritation

Transition from Nephrostomy

Once obstruction resolved or infection cleared: - Can attempt antegrade ureteral stent (pigtail stent advanced antegrade from nephrostomy) - Or remove nephrostomy if obstruction permanently resolved - Trial clamping (clamp catheter; observe for fever/pain/elevated creatinine) before removal

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VII. Urologic Malignancy Overview

Renal Cell Carcinoma (RCC)

Epidemiology: - 3% of adult cancers; ~81,000 new cases/year in USA - Peak incidence: 60s–70s; male:female ~1.5:1 - Risk factors: Smoking, obesity, hypertension, hereditary syndromes (VHL, HREM, Birt-Hogg-Dubé)

Pathology: - 85% clear cell RCC (most aggressive) - 10% papillary - 5% chromophobe (better prognosis) - 5% rare variants (oncocytoma, Wilms, etc.)

Clinical Presentation: - **Classic triad** (rare, late disease): Hematuria + flank pain + abdominal mass (20% present this way) - **Asymptomatic** (most common now): Found incidentally on

imaging for other reasons - **Incidental small renal masses:** 15–20% of CTs; many benign (oncocytoma, cysts, angiomyolipoma) - **Metastatic presentation:** 25% present with mets (lung, bone, brain) - **Paraneoplastic:** Fever, weight loss, anemia (EPO-secreting), hypercalcemia

Diagnosis: - CT chest/abdomen/pelvis with IV contrast (enhancement >10 HU with contrast = RCC) - Biopsy rarely needed (except if cryoablation planned) - Staging: TNM system

Management: - **Small masses (<4 cm):** Active surveillance (many benign), biopsy, or ablation (cryo, RFA) - **Localized (≤Stage III):** Surgical nephrectomy (partial if possible to spare function; radical if large/solitary kidney not option) - **Advanced/metastatic:** Immunotherapy (checkpoint inhibitors) or targeted therapy (tyrosine kinase inhibitors)

Bladder Cancer

Epidemiology: - 4% of adult cancers; ~82,000 new cases/year - Risk factors: Smoking (80% of cases), schistosomiasis, radiation, chemotherapy

Pathology: - 90% urothelial carcinoma (transitional cell) - 5–10% squamous cell, adenocarcinoma (worse prognosis) - 75% non-muscle invasive (NMIBC) at diagnosis - 25% muscle invasive (MIBC; much worse)

Clinical Presentation: - **Gross hematuria** (most common; painless) - Dysuria, frequency, urgency (irritative symptoms) - Suprapubic/pelvic pain (advanced) - Obstructive symptoms (if trigone involved)

Diagnosis: - **Cystoscopy + biopsy:** Gold standard - Urine cytology (sensitive for high-grade, not low-grade) - CT urogram (staging if upper tract involvement)

Management: - **NMIBC:** TURBT (transurethral resection) + intravesical BCG or chemotherapy; frequent surveillance cystoscopy - **MIBC:** Radical cystoprostatectomy with urinary diversion (ileal conduit, neobladder, continent pouch) - **Metastatic:** Chemotherapy (cisplatin-based) or immunotherapy

Prostate Cancer

Epidemiology: - Most common cancer in men (15% of male cancers); ~268,000 new cases/year - Risk factors: Age (>65), black race, family history, BRCA mutations

Pathology: - 99% adenocarcinoma (acinar, atrophic, foamy variants) - Graded by Gleason score (6–10; higher = worse)

Clinical Presentation: - **Asymptomatic** (found by PSA screening) - Lower urinary tract symptoms (frequency, urgency, hesitancy — often from BPH, not cancer) - Hematospermia (blood in semen) - Metastatic pain (bone, especially spine; pelvic pain)

Screening and Diagnosis: - PSA screening controversial (risk of overdiagnosis) - Shared decision-making with patient (age <50, life expectancy) - Digital rectal exam (DRE) still recommended by some - Biopsy if PSA elevated or DRE abnormal (elevated PSA alone doesn't mandate biopsy; trajectory matters)

Management: - **Low-risk, localized:** Active surveillance (PSA monitoring, repeat biopsy q1–2 years) - **Intermediate-risk:** External beam radiation ± androgen deprivation - **High-risk, localized:** Radical prostatectomy or radiation + ADT - **Advanced/metastatic:** Androgen depriva-

tion therapy (ADT), LHRH agonists/antagonists, antiandrogens - **Castrate-resistant disease:** Docetaxel, cabazitaxel, abiraterone, enzalutamide, immunotherapy

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Practice Questions

1. **A 68-year-old man presents with LUTS and IPSS score of 22. Urinalysis normal; post-void residual 85 mL. DRE shows enlarged prostate without nodules. Serum creatinine normal. What is the most appropriate first-line therapy?**

    - A) Immediate TURP
    - B) Start tamsulosin + HCTZ
    - C) Start tamsulosin + finasteride + counseling on lifestyle modification
    - D) Start finasteride alone; no alpha-blocker needed

    Answer: C. This patient has **moderate LUTS (IPSS 22)** with evidence of obstruction (PVR 85 mL, enlarged prostate). First-line medical therapy combines: (1) α-blocker (tamsulosin) for rapid symptom relief; (2) 5-ARI (finasteride) for prostate regression and long-term benefit; (3) lifestyle modification (fluid restriction, timed voiding, avoid anticholinergics). HCTZ not indicated (not hypercalciuric). TURP reserved for medical therapy failure or complications (retention, UTI). Finasteride alone is slower and less effective than combination.

2. **A 34-year-old woman with spinal cord injury (T6 level, 5 years post-injury) manages her neurogenic bladder with intermittent catheterization (CIC) 5×/day. Recent cystometry shows: Bladder capacity 350 mL, compliance normal, detrusor leak point pressure (DLPP) 32 cm H_2O. She reports occasional incontinence between caths. What is the best next step?**

    - A) Continue CIC alone; no change needed
    - B) Add anticholinergic (oxybutynin) to reduce detrusor contractions
    - C) Increase CIC frequency to 6×/day
    - D) Recommend bladder augmentation surgery immediately

    Answer: B. This patient's urodynamic results show **elevated DLPP (32 cm H_2O; normal <20)**, indicating high bladder pressure with risk of reflux and hydronephrosis. While her capacity is adequate, the high pressures are unsafe. **Adding anticholinergic medication (oxybutynin or mirabegron)** reduces detrusor contractions ☐ lowers DLPP ☐ protects upper tract. If still symptomatic after optimizing anticholinergics, then consider botulinum toxin. Surgical augmentation is reserved for failure of medical therapy.

3. **A 58-year-old man with history of recurrent kidney stones presents with acute right flank pain, fever (38.5°C), CVA tenderness, and AKI (Cr 1.8, baseline 1.0). Ultrasound shows mild right hydronephrosis but no stone seen. What is the most urgent next step?**

    - A) Treat with antibiotics alone and recheck imaging in 24 hours
    - B) Arrange CT scan to look for stone
    - C) Urgent urology consultation for nephrostomy tube placement
    - D) Start NSAIDs for pain control

Answer: C. This patient has **fever + AKI + hydronephrosis** consistent with **pyonephrosis (infected obstructed kidney) — a urologic emergency**. Delaying intervention risks septic shock and death. Immediate actions: (1) **Urgent decompression (nephrostomy or stent)**—do not delay for CT; (2) Blood cultures; (3) IV antibiotics (fluoroquinolone or aminoglycoside pending cultures); (4) imaging (CT/ultrasound) parallel to intervention. Treating with antibiotics alone without decompression is dangerous—bacteria will continue proliferating in obstructed, infected kidney.

Clinical Pearls Summary

- **BPH is extremely common** (>80% of men >80); not all symptomatic ☐ individualize treatment decisions
- **Alpha-blockers act faster than 5-ARIs** (days vs. months); use α-blocker for faster relief
- **Neurogenic bladder best managed with CIC** if patient able ☐ preserves bladder function and prevents upper tract deterioration
- **Intermittent catheterization is better long-term than indwelling catheters** (lower infection, better QoL)
- **NDOU (no hydronephrosis) can occur with acute/severe obstruction** — don't falsely reassure on normal ultrasound; get CT if AKI + obstruction suspected
- **Post-obstructive diuresis is common but usually self-limited** — don't overtreat with IV fluids; monitor electrolytes carefully
- **Pyonephrosis (infected obstructed kidney) is a urologic emergency** — requires urgent decompression BEFORE definitive treatment
- **Nephrostomy tubes need regular flushing** to prevent obstruction; infection risk increases with duration
- **RCC is often found incidentally**; many small masses are benign ☐ not all need surgery
- **Bladder cancer and prostate cancer require long-term surveillance** after treatment; high recurrence/progression risk

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Clinical Resources

- Clinical Review: Uropathy Report — Comprehensive clinical review with PubMed references