GERIATRIC VOIDING DYSFUNCTION AND INCONTINENCE: 2016
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Background and Significance

Urinary Incontinence (UI) is a very common and costly problem for the elderly:
• Daily-weekly UI afflicts 10% of those at home, 30% in acute care, >50% in NHs
• Costs $26 billion/yr in ‘95, more than dialysis and coronary artery bypass surgery combined
But UI is usually treatable and never normal—despite age/setting, or even in demented pts.
Moreover, treatment involves addressing drugs and diseases outside the urinary tract.
Most patients don’t mention UI; thus, PCP detection, evaluation, and Rx are key.

Impact of Normal Aging on the Urinary Tract

Bladder sensation and contractility decrease with age in both sexes. In females, urethral length and sphincter strength also decrease. In males, the prostate enlarges. Involuntary bladder contractions develop in half of all elderly, and both nocturnal fluid excretion and post-voiding residual urine (PVR) increase with age – though the PVR only to 50-100 ml.

These normal changes predispose to incontinence, particularly when combined with urological or non-urological disease. Non-urological disease contributes to UI because continence requires intact mobility, cognition, motivation, and manual dexterity, as well as intact lower urinary tract function. Because non-urological causes usually respond readily to medical intervention, they are considered causes of transient UI.

Types of Incontinence and Their Causes

Transient UI (problem usually outside urinary tract—DIAPERS mnemonic, Tables 1,2)

Established incontinence (generally inside the urinary tract)—Underlying mechanisms:
• Detrusor contracts when it shouldn’t [detrusor overactivity, DO]
• Detrusor doesn’t contract adequately when it should [underactive detrusor, UD]
• Urethral resistance is too low [stress incontinence, SI]
• Urethral resistance is too high [bladder outlet obstruction, BOO]
• “Functional” impairment important, but likely contributes to—not causes—UI

A. Detrusor overactivity (DO), the cause of most cases of Overactive Bladder
Most common cause of geriatric incontinence (50-75% of cases)
Idiopathic, neurogenic, or assoc w/urethral obstruction or incompetence
Presents as urge incontinence or episodic gush without warning—in the absence of stress maneuver or urinary retention
Can coexist with bladder weakness (Detrusor Hyperactivity with Impaired Contractility [DHIC]); consider DHIC especially when PVR elevated
B. **Stress Incontinence (SI)**
Second most common cause in women (uncommon in men w/o radical prost)
Usually due to inadequate sphincter support (urethral hypermobility), so that increased abdominal pressure pushes urine out; much less commonly, due to intrinsic sphincter weakness/deficiency (ISD)
DO can accompany SI (mixed UI) or mimic SI if stress maneuvers trigger DO

C. **Urethral obstruction (Bladder Outlet Obstruction, or BOO)**
Second most common cause in men (rare in women); usually due to BPH
Presents as urge or overflow incontinence or post-void dribbling
Hard to diagnose clinically because sx (esp. nocturia [Table 5] post-void dribbling), prostate size, PVR, and cystoscopy are insufficiently specific

D. **Detrusor underactivity**
Least common cause (about 5% of cases in both sexes)
Idiopathic > autonomic neuropathy, LMN lesion, or prior obstruction
Usually presents as overflow incontinence w/PVR usually >400 ml

**Evaluation**

**Strategy:**
First office visit—Ask about UI and give out bladder diary (BD, see Figure)
Second visit—Hx/PEx w/pelvic/rectal, stress test, PVR, review bladder diary

- **Ask/Detect:** “Do you ever lose control of your urine and wet yourself?”

- **UI is rarely due to a single cause. Optimal approach is possible only by identifying and addressing all potential causes (including “DIAPERS”) and contributors.**

- Because multiple diseases and drug-use are common in older patients, ensure that impugned factor really contributes; e.g., Ca²⁺ blocker is irrelevant if PVR <100 ml and no edema (since UI link is via retention and edema). Similar reasoning for other drugs and diseases; e.g., ↑nocturnal fluid excretion is irrelevant for pt without nocturnal UI.

- **Clinically exclude serious conditions (by rectal, neuro exam; PVR; UA):**
  Brain mass; spinal cord injury/lesion; bladder stone/cancer; prostate cancer; urethral obstruction with hydronephrosis (renal ultrasound if man’s PVR ≥ 150 ml)

- **Perform stress test (in women) – Important caveats:**
  + Patient should be as close to upright position as possible
  + Bladder should be full, but patient should not have abrupt urge (i.e., DO)
  + Pelvic muscles should be relaxed (check gluteal creases)
  + Cough should be single and forceful
  + Positive test: Leakage initiation and cessation coincide **instantaneously** with cough; suggests SI (provided PVR <400 ml)
  + Negative test: No leak/delayed leak (≥ few secs) w/≥ 150-200 ml in bladder
• Measure patient’s void, then measure PVR by portable ultrasound or catheterization

• Refer patients with overflow UI (PVR ≥ 400 ml). In the rest:

  **Women without overflow:** Differentiate Stress [SI] from DO (BOO is rare):
  Stress test positive & PVR ≤ 200 ml → Presumed diagnosis is SI
  Stress test negative & PVR ≤ 50 ml → Presumed diagnosis is DO

  **Men without overflow:** Differentiate Obstruction [BOO] from DO (SI rare):
  Precipitant urgency & PVR ≤ 50 ml → obstruction unlikely, so treat as if DO
  All others, _conservative_ (only) Rx for both DO and obstruction (see below)

• Sophisticated (urodynamics [UDS]) testing indicated if:
  Need to know exact type of urinary tract dysfunction,
  Empiric therapy failed, or
  Surgery would be appropriate.
  _Note:_ Neither cystoscopy, IVP, prostate size, nor ease of cath’n can dx obstruction

**Treatment Strategies**

• Address all transient causes and functional impairments (see **Tables 1 and 2**).

• If UI persists, treat specific type of UI as determined above (see **Tables 3 and 4**)

• Successful treatment _rarely_ involves a _single_ intervention.

• Refer refractory cases, if clinically appropriate.

• Catheters: see **Tables 3, 6, and 7**.

**Bottom Line**

UI is never normal, no matter how old, demented, or immobile the patient—even among nursing home patients. Causes are disparate, usually >1 cause responsible, and most are in purview of PCP and can be diagnosed/treated without sophisticated testing. If UI persists, approach depends on PCP’s level of interest/expertise. Since most elderly UI patients are women—in whom the diagnosis is more straightforward—most can be treated empirically with behavioral interventions and/or medication. In men, the approach depends more on risk/benefit. A creative and positive approach cures many and improves most.

**References:**
Goode PS _et al._ Incontinence in older women. _JAMA_ 2010; 303: 2172-2181. (evidence-based review)
Marinkovic SP _et al._ Management of overactive bladder syndrome. _BMJ_ 2012; 344:e2365-72
# TABLE 1: CAUSES OF TRANSIENT INCONTINENCE*

<table>
<thead>
<tr>
<th>Cause</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>D</strong> delirium/confusional state</td>
<td>Results from almost any underlying illness or medication; incontinence is secondary and abates once the cause of confusion has been corrected</td>
</tr>
<tr>
<td><strong>I</strong> infection -- Urinary (only <em>symptomatic</em>)</td>
<td>Causes incontinence, but the more common asymptomatic bacteriuria does not</td>
</tr>
<tr>
<td><strong>A</strong> trophic urethritis/vaginitis</td>
<td>Characterized by vaginal erosions, telangiectasia, petechiae, and friability; may cause or contribute to incontinence. Now controversial but may be worth a 3-6 month trial of estrogen, especially topical application (if not contraindicated by breast or uterine cancer)</td>
</tr>
<tr>
<td><strong>P</strong> pharmaceuticals</td>
<td>Includes many prescribed and non-prescribed agents, because incontinence can be caused by diverse mechanisms (Table 2)</td>
</tr>
<tr>
<td><strong>E</strong> excess urine output</td>
<td>Results from large fluid intake, diuretic agents (including theophylline, caffeinated beverages, and alcohol), and metabolic disorders (e.g., hyperglycemia or hypercalcemia); nocturnal incontinence also may result from mobilization of peripheral edema (e.g., CHF, venous insufficiency, drug side effect [Table 2])</td>
</tr>
<tr>
<td><strong>R</strong> restricted mobility</td>
<td>Often results from overlooked, correctable conditions such as arthritis, pain, foot problem, postprandial hypotension, depression, or fear of falling</td>
</tr>
<tr>
<td><strong>S</strong> stool impaction</td>
<td>May cause both fecal and urinary incontinence that remit with disimpaction</td>
</tr>
</tbody>
</table>

Bladder diary of an incontinent 75 year old man. Urodynamic evaluation excluded urethral obstruction and confirmed a diagnosis of detrusor hyperactivity with impaired contractility (DHIC). Note the 24 hr urine output of nearly 3 liters due to the belief that drinking 10 glasses of fluid/day was “good for my health.” (He did not mention this until queried about the voiding record.) Given the typical voided volume of 150-250 ml and a measured PVR of 150 ml, excess fluid intake was overwhelming his usual bladder capacity of 400 ml (150 + 250 ml). Although involuntary bladder contractions were present, the easily reversible volume component of the problem – combined with the risk of precipitating urinary retention with an anticholinergic agent – prompted treatment with volume restriction alone. After daily urinary output dropped to 1500 ml, frequency abated and incontinence resolved.


TABLE 2: COMMONLY-USED MEDICATIONS THAT MAY AFFECT CONTINENCE

<table>
<thead>
<tr>
<th>Type of Medication</th>
<th>Examples</th>
<th>Potential Effects on Continence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedatives/Hypnotics</td>
<td>Long-acting benzodiazepines (e.g., diazepam, flurazepam)</td>
<td>Sedation, delirium, immobility</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Dicyclomine, disopyramide, antihistamines (sedating ones only, e.g., Benadryl®)</td>
<td>Polyuria, frequency, urgency, sedation, delirium, immobility</td>
</tr>
<tr>
<td>Anticholinergics</td>
<td>Thioridazine, haloperidol</td>
<td>Urinary retention, overflow incontinence, delirium, impaction</td>
</tr>
<tr>
<td>Antipsychotics</td>
<td>Amitriptyline, desipramine; not SSRIs</td>
<td>Anticholinergic actions, sedation, rigidity, immobility</td>
</tr>
<tr>
<td>Antidepressants (tricyclics)</td>
<td>Trihexyphenidyl, benztropine mesylate (not L-dopa or selegiline)</td>
<td>Anticholinergic actions, sedation</td>
</tr>
<tr>
<td>Anti-Parkinsonians</td>
<td>Opiates</td>
<td>Urinary retention, fecal impaction, sedation, delirium</td>
</tr>
<tr>
<td>α-Adrenergic antagonists</td>
<td>Prazosin, terazosin, doxazosin</td>
<td>Urethral relaxation may precipitate stress incontinence in women</td>
</tr>
<tr>
<td>α-Adrenergic agonists</td>
<td>Nasal decongestants</td>
<td>Urinary retention in men</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>All dihydropyridines*</td>
<td>Urinary retention; nocturnal diuresis due to fluid retention</td>
</tr>
<tr>
<td>Potent diuretics</td>
<td>Furosemide, bumetanide (not thiazides)</td>
<td>Polyuria, frequency, urgency</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Indomethacin, COX-2 inhibitors</td>
<td>Nocturnal diuresis due to fluid retention</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td>Rosiglitazone, pioglitazone</td>
<td>Nocturnal diuresis due to fluid retention</td>
</tr>
<tr>
<td>Anticonvulsants/analgesics</td>
<td>Gabapentin, pregabalin</td>
<td>Nocturnal diuresis due to fluid retention, sedation, delirium</td>
</tr>
<tr>
<td>Parkinson’s agents (some)</td>
<td>Pramipexole, ropinirole, amantadine</td>
<td>Nocturnal diuresis due to fluid retention</td>
</tr>
<tr>
<td>Angiotensin converting enzyme (ACE) inhibitors</td>
<td>Captopril, enalapril, lisinopril</td>
<td>Drug-induced cough can precipitate stress incontinence in women and in some men with prior prostatectomy</td>
</tr>
<tr>
<td>Vincristine</td>
<td></td>
<td>Urinary retention owing to neuropathy</td>
</tr>
</tbody>
</table>

* Examples include amlodipine (Norvasc®), nifedipine, nicardipine, isradipine, felodipine, nimodipine

<table>
<thead>
<tr>
<th>Condition</th>
<th>Clinical Type†</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detrusor overactivity with</td>
<td>Urge</td>
<td>1. Bladder retraining or prompted voiding regimens</td>
</tr>
<tr>
<td>normal contractility</td>
<td></td>
<td>2. ± Bladder relaxant medication if needed. If fails, consider Posterior tibial neurostimulation (PTNS), sacral neuromodulation, or intra-detrusor injection of onabotulinumtoxin A (not FDA-approved)</td>
</tr>
<tr>
<td>(DO)</td>
<td></td>
<td>3. Indwelling catheterization alone is often unhelpful because detrusor ‘spasms’ often increase, leading to leakage around the catheter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. In selected cases, induce urinary retention pharmacologically and add intermittent or indwelling catheterization‡</td>
</tr>
<tr>
<td>Detrusor hyperactivity with</td>
<td>Urge†</td>
<td>1. If bladder empties adequately, behavioral methods (as above) ± bladder relaxant medication (low doses; especially feasible if sphincter incompetence coexists)</td>
</tr>
<tr>
<td>impaired contractility</td>
<td></td>
<td>2. If residual urine &gt;150 ml, augmented voiding techniques◊ or intermittent catheterization (± bladder relaxant medication). If neither feasible, undergarment or indwelling catheter‡</td>
</tr>
<tr>
<td>(DHIC)</td>
<td></td>
<td>3. In selected cases, induce urinary retention pharmacologically and add intermittent or indwelling catheterization‡</td>
</tr>
<tr>
<td>Stress Incontinence</td>
<td>Stress</td>
<td>1. Conservative methods (weight loss if obese; treatment of cough or atrophic vaginitis; physical maneuvers to prevent leakage [eg., tighten pelvic muscles before cough, cross legs]; occasionally, use of tampon or pessary is useful)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. If leakage threshold ≥ 150 ml identified, adjust fluid excretion and voiding intervals appropriately</td>
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<tr>
<td></td>
<td></td>
<td>3. Pelvic muscle exercises + biofeedback/weighted intravaginal ‘cones;’ must continue indefinitely</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Surgery (sling, artificial sphincter, periurethral bulking injections)</td>
</tr>
<tr>
<td>Urethral Obstruction</td>
<td>Urge/Overflow*</td>
<td>1. Conservative methods (including adjustment of fluid excretion, bladder retraining/prompted voiding) if hydronephrosis, recurrent symptomatic UTI, and hematuria have been excluded</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Alpha adrenergic antagonist</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Consider adding a bladder relaxant if DO coexists, PVR ≤250 ml, and surgery not desired/feasible; monitor PVR!</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Finasteride, if not contraindicated and the patient either prefers it or is not a surgical candidate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Surgery (incision, prostatectomy) is an effective alternative before or after these steps</td>
</tr>
<tr>
<td>Underactive Detrusor</td>
<td>Overflow</td>
<td>1. Decompress for at least several days (the larger the PVR, the longer should be the decompression [up to a month]) and then perform a voiding trial</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Exclude urethral obstruction if this has not already been done</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. If cannot void or PVR remains large, try augmented voiding techniques◊ ± alpha adrenergic antagonist, but only if some voiding possible; bethanechol rarely useful</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. If fails, or voiding is not possible, intermittent or indwelling catheterization‡</td>
</tr>
</tbody>
</table>
TABLE 3: STEPWISE APPROACH TO TREATMENT (CONTINUED)

+ These treatments should be initiated only after adequate toilet access has been ensured, contributing conditions have been treated (e.g., atrophic vaginitis, UTI, fecal impaction, heart failure), fluid management has been optimized, and unnecessary or exacerbating medications have been addressed. For additional details, see text.

† **Urge:** Leakage in the absence of stress maneuvers and urinary retention, usually preceded by *abrupt* onset or intensification of the need to void  
**Stress:** Leakage that coincides *instantaneously* with stress maneuvers, in the absence of urinary retention or detrusor contraction  
**Overflow:** Frequent leakage of small amounts associated with urinary retention

‡ UTI prophylaxis can be used for recurrent symptomatic UTIs, but only if catheter is not indwelling.

* May also mimic stress or overflow incontinence.

# Also can cause postvoid “dribbling” alone, which is treated conservatively (e.g., by sitting to void and allowing more time, “double voiding,” and in men by gently “milking” the urethra after voiding).

◊ Augmented voiding techniques include Credé (application of suprapubic pressure) and Valsalva (straining) maneuvers, and `double' voiding. They should be performed only *after* voiding has begun.

### TABLE 4: BLADDER RELAXANT MEDICATIONS USED TO TREAT URGE INCONTINENCE *

<table>
<thead>
<tr>
<th>Medication Class, Name, and Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anticholinergic agents</strong></td>
<td>All of these anticholinergic agents have proved effective in controlled trials for older patients. Benefits of M3 selectivity remain theoretical. Concerns re: CNS side effects of oxybutynin and tolterodine emanate primarily from rare case reports and short-term, artificial experiments and are overstated; a recent study found no increased risk even in older adults with MCI (Wagg, Eur Urol, 2013). Fesoterodine is the best tested of the newer agents for older adults and has proven both safe and effective in the elderly. Immediate release oxybutynin (IR) has a rapid onset of action and can be tried prophylactically if incontinence occurs at predictable times.</td>
</tr>
<tr>
<td>Oxybutynin IR 7.5-20 mg daily (2.5-5 mg po tid-qid)*</td>
<td></td>
</tr>
<tr>
<td>Oxybutynin XL 5-30 mg once daily (given once daily)</td>
<td></td>
</tr>
<tr>
<td>Oxybutynin patch twice weekly (3.9 mg/day)</td>
<td></td>
</tr>
<tr>
<td>Oxybutynin 10% gel (1 gram topically/day)</td>
<td></td>
</tr>
<tr>
<td>Tolterodine 1-2 mg twice daily</td>
<td></td>
</tr>
<tr>
<td>Tolterodine LA 4 mg once daily</td>
<td></td>
</tr>
<tr>
<td>Darifenacin 7.5-15 mg qd†</td>
<td></td>
</tr>
<tr>
<td>Solifenacin 5-10 mg qd ‡†</td>
<td></td>
</tr>
<tr>
<td>Trosipium 20 mg daily to twice daily; XR 60 mg/day ‡◊</td>
<td></td>
</tr>
<tr>
<td>Fesoterodine 4-8 mg once daily</td>
<td></td>
</tr>
<tr>
<td><strong>Beta-3 Adrenergic Agonist</strong></td>
<td>Newer drug. Efficacy data are still limited primarily to phase 3 trials with some largely healthy elderly. Use with caution in uncontrolled hypertensive patients.</td>
</tr>
<tr>
<td>Mirabegron (begin 25 mg/day; † to 50 mg/day if needed after 8 wks)</td>
<td></td>
</tr>
<tr>
<td><strong>Smooth muscle relaxant</strong></td>
<td>Obsolete; has not proved effective in placebo-controlled trials</td>
</tr>
<tr>
<td>Flavoxate 300-800 mg daily (100-200 mg po tid-qid)</td>
<td></td>
</tr>
<tr>
<td><strong>Tricyclic Antidepressants†</strong></td>
<td>Older controlled trial data support their use, but they are not first choice. May be helpful in women with coexistent stress incontinence. Orthostatic hypotension often precludes their use, but a tricyclic antidepressant may be preferred for a depressed incontinent patient without risk for orthostatic hypotension. Doxepin has more anticholinergic side effects than imipramine and can cause confusion.</td>
</tr>
<tr>
<td>Imipramine 25-100 mg daily (10-25 mg po qd-qid)</td>
<td></td>
</tr>
<tr>
<td>[Doxepin 25-75 mg daily (10-25 mg po qd-tid)]</td>
<td></td>
</tr>
<tr>
<td><strong>IR</strong> Immediate release; XL and LA = extended release</td>
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</tr>
</tbody>
</table>

* May also be applied intravesically in patients who can use intermittent catheterization.
† May give as single daily dose of 25-100 mg after determining the optimal dose
‡ Dose reduction required for creatinine clearance ≤ 30 ml/min (e.g., in an 85 yo woman with serum creatinine of 1.2 who weighs 50 kg); avoid XR dose
◊ Levels may be affected by other drugs such as digoxin and metformin, which compete for renal tubular secretion.
○ CYP2D6 inhibitor: increases levels of metoprolol, desipramine, and likely digoxin, tramadol, thioridazine, and propafenone

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**TABLE 5**  
**CAUSES OF NOCTURIA**

I. Volume Related

- **Age-related**
  - Excess intake/alcohol
  - Diuretic, caffeine, theophylline, lithium
- **Endocrine/Metabolic**
  - Diabetes mellitus/insipidus
  - Hypercalcemia
- **Peripheral edema**
  - Congestive heart failure
  - Low albumin states
  - Peripheral vascular disease
  - Venous insufficiency
- **Drugs** (e.g., NSAIDs [indomethacin, Cox inhibitors selective/not], dihydropyridine calcium channel blockers [e.g., amlodipine])

II. Sleep Related

- **Insomnia**
- Obstructive sleep apnea (causes polyuria as well)
- Restless legs syndrome
- **Pain**
- **Dyspnea**
- **Depression**
- **Drugs**

III. Lower Urinary Tract Related

- **Small bladder capacity**
- **Detrusor overactivity**
- **Prostate related**
- **Overflow incontinence**
- **Decreased bladder compliance**
- **Sensory urgency**

TABLE 6: REMOVING AN INDWELLING URETHRAL CATHETER

Ensure that the bladder has been decompressed for at least several days, and 7-21 days if possible; the higher the residual volume, the longer the bladder should be decompressed.

Correct reversible causes of urinary retention: fecal impaction; pelvic/perineal pain; and use of anticholinergic, alpha adrenergic agonist, or calcium channel blocker medications. If an anticholinergic antidepressant/antipsychotic agent cannot be stopped, consider switching to one with fewer or no anticholinergic side effects.

Treat delirium, depression, atrophic vaginitis, impaction, or urinary tract infection, if present.

An alpha blocker (e.g., alfuzosin or tamsulosin) increases the chances of success in men but is still unproven in women. It should be initiated several days before the catheter is removed. If life expectancy is estimated to exceed a year, initiation of finasteride should be considered as well.

Record output at 6-8 hour intervals for 2 days to establish a pattern of baseline urine excretion.

Remove the catheter at a time that permits accurate recording of urine output and allows for post-voiding re-catheterization; clamping the catheter before removal is unnecessary and can be dangerous.

Reinsert the catheter only:

- After the patient voids, to determine PVR* volume; or

- After the expected bladder volume (based on records of urine output)—not the time since the catheter was removed—exceeds a preset limit (e.g., 600-800 ml); or

- If the patient is uncomfortable and unable to void despite ensured privacy and maneuvers to encourage voiding (e.g., running water, tapping suprapubic area, or stroking inner thigh).

If the patient voids and the PVR volume is:

- Greater than 400 ml -- reinsert the catheter and evaluate further, if appropriate.**

- 100-400 ml -- watch for delayed retention and evaluate further, if appropriate.**

- Less than 100 ml -- watch for delayed retention.

If the patient is unable to void, evaluate further if appropriate.** If not, resident requires permanent catheterization.

* PVR, post-voiding residual.
** Further evaluation is appropriate when the patient and physician feel that if a surgically correctable condition were found (e.g., urethral obstruction), an operation would be preferable to chronic catheterization or the other options described in the text.
TABLE 7
PRINCIPLES OF INDWELLING CATHETER CARE

Maintain sterile, closed gravity drainage system:
- Secure the catheter to upper thigh or abdomen to avoid urethral irritation and contamination. Rotate the site of attachment every few days.
- Empty the bag every 8 hours.
- Do not routinely irrigate the catheter.
- Do not clamp or kink the drainage tubing, and keep the collection bag below bladder level at all times.
- Avoid frequent cleaning of the urethral meatus; washing with soap and water once daily is sufficient; periurethral application of antimicrobial creams is ineffective.
- Adding disinfectants to the catheter bag is ineffective.

If ‘bypassing’ occurs in the absence of obstruction, it is likely due to a bladder spasm, which can be minimized by using the smallest balloon that will keep the catheter in place and by treating with a bladder relaxant medication if necessary.

Infection prophylaxis, as well as treatment of asymptomatic bacteriuria, is fruitless and usually leads to the emergence of resistant organisms.

Surveillance cultures are unnecessary and potentially misleading since bacteriuria is universal, frequently changing, and often polymicrobial.

If symptomatic UTI develops, change the catheter before obtaining a culture specimen, since cultures obtained through the old catheter may reflect organisms colonizing encrustations rather than the infecting organism. Pending culture results, antibiotic treatment should include coverage of common uropathogens, as well as uncommon ones such as Providencia stuartii and Morganella morganii.

If catheter obstruction occurs frequently, and urine cultures reveal Providencia stuartii or Proteus mirabilis, antibiotic treatment may reduce the frequency of obstruction but induces emergence of resistant organisms. In the absence of urea-splitting organisms, consider urine acidification if urine output is normal (at low output, acidification may increase blockage due to uric acid crystals). If frequent blockage persists, consider using a silicon catheter.

In the absence of obstruction and symptomatic UTI, there is no consensus on the best time to change the catheter. Some persons form material that frequently clogs the lumen; their catheter probably should be changed often enough to reduce such obstruction. Other individuals can use the same catheter for years, but it is customary to change it every 1-2 months. For patients who are difficult to catheterize, the catheter can be changed less frequently if it remains patent, and complication-free.

For long-term need, a silicone or silicone hydrogel coated catheter should be considered.