Guidelines for the Evaluation and Treatment of Recurrent Urinary Incontinence Following Pelvic Floor Surgery

Abstract

Objective: To provide general gynaecologists and urogynaecologists with clinical guidelines for the management of recurrent urinary incontinence after pelvic floor surgery.

Options: Evaluation includes history and physical examination, multichannel urodynamics, and possibly cystourethroscopy. Management includes conservative, pharmacological, and surgical interventions.

Outcomes: These guidelines provide a comprehensive approach to the complicated issue of recurrent incontinence that is based on the underlying pathophysiological mechanisms.

Evidence: Published opinions of experts, and evidence from clinical trials where available.

Values: The quality of the evidence is rated using the criteria described by the Canadian Task Force on Preventive Health Care (Table).

Recommendations

1. Thorough evaluation of each patient should be performed to determine the underlying etiology of recurrent urinary incontinence and to guide management. (II-3B)

2. Conservative management options should be used as the first line of therapy. (III-C)

3. Patients with a hypermobile urethra, without evidence of intrinsic sphincter deficiency, may be managed with a retropubic urethropexy (e.g., Burch procedure) or a sling procedure (e.g., mid-urethral sling, pubovaginal sling). (II-2B)

4. Patients with evidence of intrinsic sphincter deficiency may be managed with a sling procedure (e.g., mid-urethral sling, pubovaginal sling). (II-3B)

5. In cases of surgical treatment of intrinsic sphincter deficiency, retropubic tension-free vaginal tape should be considered rather than transobturator tape. (I-B)

6. Patients with significantly decreased urethral mobility may be managed with periurethral bulking injections, a retropubic sling procedure, use of an artificial sphincter, urinary diversion, or chronic catheterization. (III-C)

7. Overactive bladder should be treated using medical and/or behavioural therapy. (II-2B)

8. Urinary frequency with moderate elevation of post-void residual volume may be managed with conservative measures such as drugs.
to relax the urethral sphincter, timed toileting, and double voiding. Intermittent self-catheterization may also be used. (III-C)

9. Complete inability to void with or without overflow incontinence may be managed by intermittent self-catheterization or urethrolysis. (III-C)

10. Fistulae should be managed by an experienced physician. (III-C)

J Obstet Gynaecol Can 2010;32(9):893–898

INTRODUCTION

Careful investigation and patient selection before primary pelvic surgery for prolapse and/or urinary incontinence will minimize the incidence of immediate or delayed failure.1 Conservative (i.e., non-surgical) or surgical intervention may be required when prior surgery has failed, although non-surgical options should first be considered.2 Patients who complain of either de novo or recurrent urinary incontinence following reconstructive pelvic floor or incontinence surgery must undergo a thorough evaluation to identify the cause of this incontinence. The causes of incontinence for most of these patients will fall into one of the following categories.

Early Causes of Urinary Incontinence

1. Surgical correction of stress incontinence either unsuccessful or not sustained.

2. Latent (occult) stress incontinence not recognized preoperatively in a patient with pelvic organ prolapse.

3. An intraoperative or postoperative complication of surgery (e.g., fistula).

4. Surgery was inappropriate therapy or an inappropriate procedure used.

5. Pre-existing or de novo overactive bladder causing urgency incontinence.

6. Urinary tract infection causing urgency incontinence.

7. Voiding dysfunction causing urgency/frequency or overflow incontinence.

Long-term Progressive Causes of Urinary Incontinence

1. Deficiency of pelvic floor support either through genetic predisposition or other medical condition.

2. Predisposing medical conditions (e.g., chronic obstructive pulmonary disease, obesity, chronic constipation).

3. Urogenital aging and estrogen deficiency.

ASSESSMENT

Although some recurrent urinary incontinence may fit into the category outlined in the 2003 SOGC guideline, “The Evaluation of Stress Incontinence Prior to Primary Surgery,”11 specialized evaluation, including urodynamics, will permit accurate diagnosis and measurement of...
urodynamic variables that may significantly affect the management plan. Should a patient meet all the criteria on the following list, then assessment may proceed as suggested in the guideline without multichannel urodynamics.

- No more than one prior surgery for incontinence
- Symptoms of pure stress urinary incontinence
- No symptoms of overactive bladder
- No symptoms of voiding dysfunction such as urinary hesitancy, slow or interrupted stream, straining to void, or incomplete bladder emptying
- Hypermobile urethra
- Post-void residual urine volume < 100 mL
- Normal urinalysis

If any of the above requirements are not met, then a more thorough evaluation is required, using multichannel urodynamics, and referral to a subspecialist may be indicated. In all cases, the assessment must be designed to identify one or a combination of the following predisposing conditions.

1. Compromise to the urethral sphincter mechanism
2. Detrusor overactivity (overactive bladder)
3. Voiding dysfunction
4. Urogenital fistula
5. Persistent or de novo pelvic prolapse

**Recommendation**

1. Thorough evaluation of each patient should be performed to determine the underlying etiology of recurrent urinary incontinence and to guide management. (II-3B)

**Investigation of Urethral Sphincter Function**

Extrinsic support of the urethra is critical to continence, and it is provided laterally by connective tissue attachment to the pelvic side walls and posteriorly by the levator ani muscles. Urethral mobility should be assessed to evaluate extrinsic support. However, continence is maintained only if extrinsic support is complemented by normal intrinsic urethral function, which consists of the following:

1. Healthy urethral mucosa
2. A normal vascular plexus
3. Normal smooth muscle sphincter function
4. Normal external striated sphincter function

Such factors as lack of estrogen, aging tissues, devascularization, denervation, or disruption of muscular sphincters can result in marked impairment of intrinsic urethral sphincter function. These problems may also arise from prior trauma, surgery, or pelvic radiation. Some patients may have a combination of recurrent hypermobility and deficiency of intrinsic urethral function.

The majority of surgical procedures used to treat stress incontinence are designed to restore normal anatomic relationships and re-establish extrinsic urethral support. Although the exact mechanism responsible for the restoration of continence that results from successful surgery is uncertain; the only consistent postoperative urodynamic finding is enhanced pressure transmission to the urethra. This urodynamic change is probably a consequence of better transmission of intra-abdominal pressure to the newly supported urethra. It is also speculated that support of the mid-urethra results in some degree of kinking of the urethra when the proximal urethra and bladder descend during increased intra-abdominal pressure. Using this model, it is evident that in order for surgery to correct stress incontinence, it must stabilize the urethra at an appropriate anatomic level to allow pressure transmission to the urethra. If this stability is not established at the time of the operation or if it cannot be maintained over time, the surgery may fail.

Repeated surgery may cause significant trauma to the urethra resulting in a poorly vascularized, scarred, rigid “drainpipe urethra” that has no sphincteric function (intrinsic sphincter deficiency). In this condition, the urethra is a poorly coapted conduit through which urine may leak with minimal increases in intra-abdominal pressure or on a nearly continuous basis. Urethroscopy will reveal a smooth rigid tube which does not coapt. The urethrovesical junction is open and can be visualized from any point along the urethra. Videocystourethrography will confirm a urethra which is immobile and open. Intrinsic sphincter deficiency (ISD) may result after prior retropubic urethropexy (e.g., Burch procedure) or needle suspension (e.g., Pereyra procedure), or less frequently after prior anterior colpophraphy. The likelihood of ISD increases as the number of prior surgeries increases.

Urethrovessical junction mobility can be assessed by Q-tip test,2 inspection and palpation of the distal anterior vaginal wall during Valsalva manoeuvre, ultrasound examination, videocystourethrography, and urethroscopy. If recurrent hypermobility is found, it may be assumed that surgery has failed either to establish or to maintain urethral support. If the urethra is maintained in an elevated retropubic position, then it can be assumed that the goal of surgery has been achieved and that failure is the result of ISD. A patient with ISD will require subspecialized evaluation and management, including multichannel urodynamics. Urodynamic variables consistent with ISD include a maximum urethral closure pressure less than 20 cm of water or a leak point pressure less than 60 cm of water. The choice of any subsequent surgical procedure will be determined by the degree of urethral fixation by scar, the patient’s medical condition,
and the degree to which detrusor and urethral function has been compromised by denervation caused by previous surgical procedures.8

MANAGEMENT OF RECURRENT STRESS INCONTINENCE

Any decision to proceed with a specific treatment must include an assessment of the severity of the patient’s symptoms, and a trial of conservative management must be considered (as described in the SOGC guideline “Conservative Management of Urinary Incontinence”).2 Conventional retropubic urethropexy (Burch procedure)9 has a higher rate of failure in cases of suspected ISD where maximum urethral closure pressure is less than 20 cm of water. Tension-free vaginal tape has shown success rates of 74% to 82% when performed as a repeat procedure, depending on the degree of ISD present.8 Some data indicate that transobturator tape may not be as effective as tension-free vaginal tape in cases where there is an element of ISD,10 particularly if the maximum urethral closure pressure is less than 40 cm of water,11 with success rates of only approximately 50%. Patients with significant ISD, such as a fixed drainpipe urethra, may also have persistent incontinence even if undergoing a urethral sling as the repeat surgical technique. Referral to a subspecialist for a retropubic sling procedure with or without lysis of bladder neck and paraurethral scar may provide continence under these circumstances. The risk of failure and urinary retention may be increased. Periurethral bulking agents may also be injected.12 Consideration may be given to other “end-stage” options, such as placement of an artificial sphincter, urinary diversion, or chronic catheterization. Patients with recurrent stress incontinence who opt for surgical treatment should be managed according to the following recommendations.

Recommendations

2. Conservative management options should be used as the first line of therapy. (III-C)

3. Patients with a hypermobile urethra, without evidence of intrinsic sphincter deficiency, may be managed with a retropubic urethropexy (e.g., Burch procedure) or a sling procedure (e.g., mid-urethral sling, pubovaginal sling). (II-2B)

4. Patients with evidence of intrinsic sphincter deficiency may be managed with a sling procedure (e.g., mid-urethral sling, pubovaginal sling). (II-3B)

5. In cases of surgical treatment of intrinsic sphincter deficiency, retropubic tension-free vaginal tape should be considered rather than transobturator tape. (I-B)

6. Patients with significantly decreased urethral mobility may be managed with periurethral bulking injections, a retropubic sling procedure, use of an artificial sphincter, urinary diversion, or chronic catheterization. (III-C)

OVERACTIVE BLADDER

Overactive bladder, a symptom complex consisting of urgency, frequency, nocturia, and urgency incontinence, is caused by a failure of bladder inhibition and, if unrecognized prior to surgery, may cause persistent incontinence following surgery. De novo overactive bladder may develop following surgery for stress incontinence,13 particularly if extensive vaginal dissection has been performed or as a secondary result after outlet obstruction. Patients will usually present with urinary urgency, frequency, nocturia, with or without urge incontinence. Cystoscopy may identify bladder pathology responsible for the urinary symptoms, such as suture or mesh penetrating the bladder or urethra. Although a simple cystometrogram will identify most cases of overactive bladder, multichannel subtracted cystometry is indicated if the diagnosis is uncertain.

The management of overactive bladder is medical or behavioural. Medical therapy typically uses anticholinergic/antimuscarinic medications.14 Behavioural therapy includes prompted voiding, bladder training, caffeine reduction, or biofeedback, with or without electrostimulation.2 In some instances, overactive bladder is a result of outlet obstruction, (discussed in the next section), and urethrolysis may provide improvement.15

Some patients may have mixed urinary incontinence. Although surgery is not contraindicated in cases of mixed urinary incontinence,16 conservative management options for both the stress and urgency incontinence should be used, and benefits maximized before further surgery is undertaken.

Recommendation

7. Overactive bladder should be treated using medical and/or behavioural therapy. (II-2B)

VOIDING DYSFUNCTION

Voiding dysfunction as a consequence of pelvic surgery may develop for several reasons. In general, voiding dysfunction is due to either urethral obstruction or detrusor underactivity. A patient with subclinical preoperative dysfunctional voiding may not be able to empty her bladder after surgical stabilization of the urethrovesical junction.17 Excessive elevation of the urethra in patients with a normal preoperative voiding mechanism may partially obstruct urinary outflow, causing voiding dysfunction. Mild degrees of incomplete emptying will appear as urinary frequency, hesitancy, and nocturia. More severe voiding compromise is manifested in urinary retention, bladder distension and
overflow incontinence, recurrent urinary tract infections, and possible upper tract decompensation. Simple uroflowmetry including measurement of peak flow rate and post-void residual volume is sufficient to screen for voiding dysfunction. Post-void residual levels can be measured by catheterization, ultrasound examination, or contrast radiography. An intermittent voiding pattern on uroflowmetry, a decreased peak flow rate (<15 mL/second), or a high post-void residual volume (>150 mL) should prompt more sophisticated voiding studies, which may include voiding cystometry (simultaneous measurement of intravesical and abdominal pressures during voiding), urine flow rate, electromyogram, and urethral sphincter activity. Treatment should be individualized, and options include timed toileting, double voiding, intermittent catheterization, or urethrolysis. There are limited data on the use of urethral relaxants (e.g., benzodiazepines, loioresal, alpha-blockers). Detrusor stimulation using bethanecol is typically ineffective. Occasionally, voiding may be improved by use of a pessary or surgery to correct a high cystocele if present. If detrusor hypotonia is the cause, then sacral nerve stimulation can be used. Additional anti-incontinence operations for stress incontinence should be planned cautiously in patients with marked postoperative voiding dysfunction. 

**Recommendations**

8. Urinary frequency with moderate elevation of post-void residual volume may be managed with conservative measures such as drugs to relax the urethral sphincter, timed toileting, and double voiding. Intermittent self-catheterization may also be used. (III-C)

9. Complete inability to void with or without overflow incontinence may be managed by intermittent self-catheterization or urethrolysis. (III-C)

**UROGENITAL FISTULA**

Urogenital fistula following incontinence surgery is a rare complication. A fistula may exist between the vagina and the urethra, the bladder, the ureter, or a combination of these organs. Methylene blue solution may be instilled into the bladder followed by speculum examination or placement of tampons in the vagina. Direct visualization of dye or staining of the tampon will confirm the presence of a vesicovaginal fistula. Cystourethroscopy should permit the identification of a fistula in either the urethra or the bladder, as well as assessment of the precise size, location, and number of fistulae. Injection of intravenous indigo carmine followed by speculum examination or the tampon test may identify a ureterovaginal fistula if a vesicovaginal fistula has been ruled out. Intravenous pyelography or computed tomography with intravenous contrast (CT urogram) is indicated to identify possible upper tract damage.

Urogenital fistula is a surgical problem that must be corrected. The choice of procedure will depend on the severity and location of the fistula. Fistula and stress incontinence can coexist. If surgery is undertaken, it should correct all incontinence and pelvic floor prolapse disorders, either concomitantly or in stages, depending on individual circumstances.

**Recommendations**

10. Fistulas should be managed by an experienced physician. (III-C)

**REFERENCES**


