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How to Pass the FRCS(Urol)

The fourth article in this series will discuss the topics of Female Urology, Neurourology, Urethral Strictures and Reconstruction. In the next article we will cover stones and infection.

Female Urology

Q. What is urinary incontinence?

"Involuntary urine loss that is a social or hygienic problem" (ICS Definition).

Q. What is stress urinary incontinence?

Involuntary leakage on effort, exertion, sneezing, coughing. Due to hypermobility of bladder base, pelvic floor, and/or intrinsic urethral sphincter deficiency.

Q. What is urge urinary incontinence?

The involuntary leakage of urine accompanied or immediately preceded by a sudden, compelling desire to void (urgency).

Q. What is urgency?

The complaint of a sudden, compelling desire to pass urine that is difficult to defer.

Q. What is frequency?

Usually accompanies urgency with or without urge incontinence and is the complaint by a patient who considers that he/she voids too often by day.

Q. What is nocturia?

Usually accompanies urgency with or without urge incontinence and is the complaint that a individual has to wake at night one or more times to void.

Q. How would you approach the assessment of an elderly person with incontinence?

Remember – "approach the lower urinary tract first" (Resnick, US Gerontologist).

Resnick coined the term DIAPPERS to summarise the approach to use in the elderly incontinent patient. This reminds you of the non-bladder factors that may lead to or exacerbate incontinence.

- D – Delirium (ie. confusional states).
- I – Infection.
- A – Atrophic vaginitis in women.
- P – Pharmaceuticals (long acting sedatives eg. diazepam, loop diuretics etc).
- P – Parkinson's disease.
- E – Excess urine output.
- R – Restricted mobility (difficulty getting to the toilet in time).
- S – Stroke (causing detrusor hyperreflexia).

Q. Describe the anatomical basis for continence:

This is the urethral sphincter mechanism, which consists of:

- i) The intrinsic rhabdosphincter (also, confusingly, known as the external sphincter), a 'U' shaped configuration of striated muscle, being deficient posteriorly (maintenance of continence over the long-term). Innervated by somatic motoneurons from S2-4 which travel both within the pudendal nerve and also alongside parasympathetic neurons in the pelvic splanchnic nerves to the intrinsic rhabdosphincter (dual innervation). In the male the intrinsic rhabdosphincter lies just proximal to the bulbar urethra. In the female the striated urethral sphincter invests the distal two

thirds of the female urethra.

- ii) Periurethral striated muscle (pubourethral sling), which is part of levator ani. It results in short-term pressure rises during activities such as dancing (amongst others), thereby preventing leakage – hence sometimes called the 'dancing' muscle. Innervated by pudendal nerve.
- iii) In the male smooth muscle sphincter within the bladder neck – the internal sphincter (in the female, the bladder neck has little adrenergic innervation and its sphincteric function is believed to be limited - 50% of continent women have incompetent bladder necks such that urine enters the proximal urethra during a cough and yet they remain continent (Versi E, et al. *Internal urinary sphincter in maintenance of female continence*. BMJ 1986;292:166-73).
- iv) In females a correctly supported urethra, which allows transmission of intra-abdominal pressure to the urethra, resulting in compression.

Q. What and where is Onuf's nucleus?

This is the location, in the sacral cord, of the cell bodies of the *somatic* motoneurons, which innervate the intrinsic rhabdosphincter (the external sphincter).

Q. Does bilateral pudendal nerve block cause incontinence?

No. The somatic innervation of the intrinsic rhabdosphincter (the external sphincter) reaches the sphincter by travelling within the pudendal nerve, but also somatic motoneurons are believed to be transmitted in the pelvic splanchnic nerves (the *nervi erigentes*). Thus, as noted above, the intrinsic rhabdosphincter has a dual innervation. Thus, bilateral pudendal nerve block does not lead to urinary incontinence because of preservation of some somatic innervation via pelvic splanchnic nerves.

Q. Summarise the innervation of the lower urinary tract:

1. Bladder

- a) Motor innervation to detrusor is parasympathetic, cell bodies of preganglionic motoneurons lie in intermediolateral column of sacral spinal cord segments S2-4; preganglionic fibres synapse with postganglionic neurons within the pelvic plexus. Excitatory input to smooth muscle of detrusor is cholinergic.
- b) Motor innervation to trigone and lower ureters is sympathetic. Preganglionic sympathetic neuron cell bodies lie within the intermediolateral column of T10-12 and L1-2. Preganglionic neurons synapse in the sympathetic chain with postganglionic neurons which travel in the hypogastric nerves to the trigone, bladder neck (the internal sphincter – in the male) and lower ureters.
- c) Parasympathetic: sensory, stretch, fullness and pain.
- d) Sympathetic: pain, temperature, touch.

2. Urethra

- a) Intrinsic rhabdosphincter. As stated above, probably has dual motor innervation – via

somatic motoneurons, cell bodies of which lie within sacral cord segments 2-4, within Onuf's nucleus. These motoneurons travel alongside parasympathetic neurons in the pelvic splanchnic nerves (also known as nervi erigentes) and it is thought some also travel within the pudendal nerves (Tanagho EA, et al. *Urinary striated sphincter: What is its nerve supply?* Urology 1982;20:415-17). Remember, these neurons are somatic, *not autonomic*.

- b) Smooth muscle sphincter within the bladder neck in males (the internal sphincter) – sympathetic innervation, T10-L2, nerve fibres travelling to the bladder neck in the hypogastric nerves.
- c) Periurethral striated muscle (pubococcygeus) – somatic innervation, S2. Sensory innervation of urethra – afferent neurons travel via pudendal nerve, terminating within dorsal horns of S2-4, connecting to neurons which ascend to brainstem (pons) and cerebral cortex.

Q. Where is LUT neurological activity co-ordinated?

Within the pontine micturition and storage centres, which lie in the pons. Destruction of the pontine micturition centre (Barrington's nucleus) leads to inability to produce a co-ordinated bladder contraction combined with relaxation of the external sphincter (the rhabdosphincter).

Treatment of Urodynamic Stress Incontinence

A. Injectables

Q. What properties should the ideal injectable for stress urinary incontinence have?

It should:

- i) Be inert (to prevent tissue reaction).
- ii) Be non-degradable (to prevent recurrent leakage).
- iii) Become encapsulated (to prevent migration of the injected particles).
- iv) Not lose nor gain bulk (recurrent incontinence or incomplete bladder emptying).
- v) Be easily injectable (which means it should not be too viscous, otherwise you'll never get it through the needle of the syringe).

Q. What is Macroplastique®?

It is a silicone based compound (specifically polydimethylsiloxane) suspended in a povidone hydrogel. Particle size mostly > 100 microns (compare with PTFE, polytetrafluoroethylene – Teflon® – where particle size is between 40-100 microns, which allows migration of particles, in animal studies to lung, brain, kidney, spleen).

Q. How is collagen for SUI processed and what are its advantages/disadvantages as an injectable?

Bovine dermal collagen is cross-linked with glutaraldehyde. 95% is type I, 5% is type III.

Advantages:

- i) It has low viscosity (easy to inject through a small needle).
- ii) Biocompatible.
- iii) Not associated with granuloma formation and does not migrate.

Disadvantages:

- i) It is allergenic (pre-injection skin test required – 3% of patients develop hypersensitivity within three days, but hypersensitivity may be delayed as long as one month; therefore wait at least a month before injecting).
- ii) Costly.
- iii) Completely degrades within 18 months or thereabouts. Therefore repeat injections may be required.

Q. What are the advantages/disadvantages of collagen injections for SUI?

Advantages:

- i) Low viscosity (therefore small needle can be used and easy to inject).
- ii) Less tissue reaction compared with PTFE (no granuloma formation).
- iii) No risk of migration.

Disadvantages:

- i) Allergenic.
- ii) 3% of women develop a hypersensitivity reaction (therefore skin testing is required in all women 30 days before injection).
- iii) Costly; degrades within 18 months or so; 40% require > one injection to achieve continence.

Q. Describe the technique of using an injectable for SUI:

Inject into suburothelium (in theory into lamina propria rather than into muscle of sphincter or bladder neck, as this may damage sphincter). Several points (usually four quadrants) around the clock are used, raising a bleb of the injectable so that the four blebs appose each other. Avoid reinserting the cystoscope and avoid a catheter if possible, to prevent compression of the injectable.

Outcomes of injectables for SUI. Generally speaking somewhere between 50-70% of patients selected for treatment have persistently improved continence at between one to two years of follow-up.

B. Pubovaginal slings

Q. What are the indications for a pubovaginal sling?

Essentially intrinsic sphincter deficiency with urethral hypermobility.

Q. What types of materials are pubovaginal slings made from?

- i) Autologous – rectus fascia; fascia lata; vaginal wall; pyramidalis muscle.
- ii) Synthetic – Dacron (mersilene); Prolene (polypropylene).
- iii) Non-synthetic – lyophilised dura mater or porcine dermis.

Q. What are the complications?

- i) Injury of adjacent organs (bladder, vessels, bowel, nerves).
- ii) Recurrent urinary incontinence. Due to persistent urethral hypermobility – consider redo-sling. Persistent or de novo detrusor overactivity - try anticholinergics.
- iii) Retention – due to high sling tension; poor detrusor function; concurrent surgery such as cystocele or rectocele repair. May be managed by ISC. Removal of the sling may be required.

Q. What are the outcomes?

Cure rates of pubovaginal slings approach 90% at approximately three years.

Q. What is a Burch colposuspension?

As originally described, attachment of paravaginal tissue to iliopectineal (Cooper's) ligament on superior surface of superior pubic ramus (Burch, Am J Obstet Gynec, 1961). Avoid approximating the former to the latter, as retention may result and the over-tight sutures will cut out of the tissues.

Q. What are the potential complications of a Burch colposuspension?

- i) Sutures placed through bladder.
- ii) Urinary retention and voiding difficulty (3-30%).
- iii) Wound infection.
- iv) Development of de novo bladder overactivity.

C. Artificial urinary sphincter

Q. Who makes it, what model is currently in use and what is it made from?

American Medical Systems; the AMS800 (first introduced into clinical practice in 1982); silicone elastomer.

Q. For bulbar urethral placement, what is the usual cuff size and balloon pressure? What is the sphincter filled with?

- 4.5cm cuff size.
 - Balloon pressure 61-70cmH2O.
 - Filling solutions must be isosmotic.
- The silicone elastomer is semipermeable and the volume of the

pressure regulating balloon will therefore expand or contract if non-isosmotic filling solution is used. Normal saline can be used. If the surgeon wishes to use a contrast agent (so the components of the sphincter can be visualised by x-ray, a standard set of filling solutions consisting of varying proportions of water and contrast media is supplied by American Medical Systems, who make the artificial urinary sphincter).

Q. What problems or complications can occur in the short and long-term?

Short-term:

- i) Device infection usually necessitating, usually, complete removal of all components.
- ii) Urinary retention (check device is deactivated, manage by urethral catheterisation followed by catheter removal a few days later).

Long-term:

- i) Cuff erosion (presenting with perineal pain and swelling, pain in tip of penis, recurrent incontinence, UTI, bloody urethral discharge – requires cuff removal with re-implantation in six months).
- ii) Mechanical malfunction – leak of filling medium, pump failure (usually manifesting as recurrent incontinence), poor pump position (riding up of pump into groin or ‘migration’ to other relatively inaccessible sites such as posterior to or between testes, making manipulation of pump for daily activation by the patient difficult or impossible).
- iii) Tissue atrophy beneath the cuff, presenting as recurrent incontinence.
- iv) Recurrent incontinence (see below for management of).

Q. How would you investigate recurrent incontinence in a patient who has had a well functioning artificial sphincter?

- i) Activate the sphincter to see if it cycles normally (reduction in the number of ‘squeezes’ required to empty the cuff may indicate a fluid leak).
- ii) KUB X-ray (looking for contrast leak from the balloon – loss of its spherical appearance suggests a leak).
- iii) Flexible cystoscopy looking for cuff erosion.
- iv) In a neuropathic patient consider repeating urodynamics if there is a possibility that the patient’s bladder behaviour might have changed (from low pressure to high pressure secondary to poor compliance or hyperreflexia).
- v) If all these investigations are normal, the likely diagnosis is atrophy of the urethra beneath the cuff. Revision surgery will be required to reduce the cuff size.

Q. What is Botox?

Botulinum Toxin A. There are seven isoforms of the toxin. Only Types A and B have been used clinically. The toxin consists of heavy (100KD) and light chains (50KD), it is the light chain that is neurotoxic. It does not cross BBB.

Other forms:

- i) Neurobloc (B);
- ii) Dysport (A).

Q. How does Botox work?

Inhibits release of Acetylcholine causing a temporary chemodeneration.

Q. What are its reported uses within Urology?

- i) Motor and sensory urge.
- ii) Neurogenic detrusor overactivity.
- iii) Detrusor sphincter dyssynergia (DSD).
- iv) Retention due to acontractile detrusor.
- v) Chronic prostatidynia.

Q. Describe its administration:

Developed by Schurch et al, 2000. Intradetrusor injection via flexible cystoscope. 22G 4mm needle. 10 units Botox in 1ml to each of either 20 sites for idiopathic overactive bladder (OAB) or 30 sites for non-idiopathic OAB.

Q. What is the response rate?

80–90% response with mean duration of response of nine months (range 3–24). Repeat injections offer similar response (up to at least four injections).

Q. What are the complications?

- i) Generalised weakness.
- ii) Need to self catheterise (15–20%).
- iii) Paralysis.
- iv) Blurred vision.
- v) Diplopia.
- vi) Dysphagia.
- vii) Infection.
- viii) Haematuria (almost universal).
- ix) Incontinence.

Q. What are the contraindications?

- i) Myasthenia gravis.
- ii) Eaton Lambert Syndrome.
- iii) Pregnancy.
- iv) Breast feeding.
- v) Bleeding diathesis.

Neuro-urology

Spinal cord injuries

Q. What is the most likely type of bladder and sphincter behaviour (urodynamically speaking) in a T6 paraplegic man?

Detrusor hyperreflexia (DH) combined with DSD (some men with this level of SCI have a low pressure bladder and sphincter so the correlation between cord level of spinal injury and bladder/sphincter behaviour is not exact. However, the usual pattern of urodynamic behaviour in a T6 paraplegic man is DH + DSD).

Q. List the possible clinical consequences of this particular type of bladder behaviour:

- i) Hydronephrosis, leading to deterioration of renal function if unrelieved.
- ii) Recurrent UTIs (due to incomplete bladder emptying).
- iii) Incontinence (due to high pressure hyperreflexic bladder contractions).
- iv) Episodes of autonomic dysreflexia.

Q. What are the management options for a male patient with a T6 spinal cord injury and hydronephrosis due to detrusor sphincter dyssynergia combined with detrusor hyperreflexia?

The precise management options will depend on patient choice, but include:

- i) Intermittent self catheterisation (usually combined with an anticholinergic and/or regular intravesical botulinum toxin injections to lower bladder pressure).
- ii) A long-term (usually suprapubic) catheter.
- iii) External sphincterotomy (by surgical division of the sphincter or, in some case, regular intravesical botulinum toxin injections into the sphincter).
- iv) Sacral deafferentation with either (a) ISC or (b) implantation of a sacral anterior root stimulator (Brindley stimulator) onto sacral nerve roots S2-4) to ‘drive’ micturition at times that are convenient to the patient. Sacral deafferentation converts the hyperreflexic, high pressure bladder into an areflexic, low pressure one and probably also lowers external sphincter pressures.

Q. What is leak point pressure?

The detrusor pressure at which urethral leak of urine is first identified. McGuire (1981) recognised that patients with neurological voiding dysfunction and a LPP of > 40cm H₂O were at increased risk for development of upper tract deterioration (McGuire E. *Prognostic value of urodynamic testing in myelodysplastic patients.* J Urol 1981;126:205-9).

Q. What do you understand by the term 'balanced bladder' in the context of the spinal cord injury bladder?

This is a term used to describe the presence or absence of a certain relative percentage of residual urine volume after attempted voiding. An 'unbalanced' bladder in a patient with an upper motoneuron lesion is one where the residual urine volume is > 20% of the total bladder capacity. An unbalanced bladder implies lack of synergy between bladder contraction and urethral (external sphincter) relaxation i.e. it is an indication of functionally significant detrusor sphincter dyssynergia.

The term has limitations. A spinal cord injury patient might leave a residual urine volume of 30% of total bladder capacity and yet have no symptoms or other problems (i.e. they might have no urinary infection and no hydronephrosis), whereas patients with percentage residual urine volumes smaller than 20% may nonetheless develop hydronephrosis suggesting serious back pressure on the kidneys.

Reconstruction

Urethra Strictures

Q. What type of epithelium lines the penile urethra?

Pseudostratified columnar epithelium. Islands of stratified squamous epithelium are found near the external meatus.

Q. Summarise the causes of urethral strictures:

A. Anterior urethra

- i) Injury to the urethral epithelium and/or the underlying corpus spongiosum that leads to healing by fibrosis.
- ii) Straddle injury.
- iii) Iatrogenic injury (from instrumentation).
- iv) BXO.
- v) Inflammatory processes such as gonorrhoea.

B. Posterior urethra

Strictures located here are generally the result of a distraction injury either from trauma (pelvic fracture) or Radical Prostatectomy. The urethra becomes obliterated by the fibrosis that fills the space between the distracted ends of the urethra.

Q. Describe, in general terms, how you would manage an anterior urethral stricture:

Avoid the so-called 'reconstructive ladder' where several urethral dilatations are followed by several optical Urethrotomies and eventually definitive surgery in the form of an Urethroplasty. This sequential process may extend the length and depth of the stricture increasing the complexity and compromising the outcome of Urethroplasty.

Aims of treatment of urethral stricture disease – firstly define the goal of treatment, which essentially is whether the patient wishes his/her stricture to be managed (periodic dilatations or Urethrotomies) or cured (by Urethroplasty).

Q. What is the likelihood of cure (i.e. no need for any subsequent treatment) of a urethral stricture after optical Urethrotomy?

Approximately 30% to 35% (Pansadoro V, Emiliozzi P. *Internal urethrotomy in the management of anterior urethral strictures: Long term follow up.* J Urol 1996;156:73–5).

For strictures at the bulbous urethra that are less than 1.5cm in length and not associated with dense and deep spongiofibrosis, as many as 75% can be so cured.

Q. What are the principles for successful Urethroplasty?

- i) Complete excision of the area of fibrosis.
- ii) Widely spatulated urethral anastomosis.
- iii) Tension-free anastomosis.