

April Clinical Focus Topic – **URINARY FREQUENCY**

This month I am focusing on a topic that may seem a little boring... But I would like to look at it from a less common perspective.....Urinary Frequency and its relationship to voiding dysfunction.

Both the ICS and IUGA define urinary frequency as the

“complaint by a patient who considers that he/she voids too often by the day”

Abrams 2002, Haylen et al 2010

We have all obviously had numerous patients present complaining of urinary frequency. They often indicate that their bladder rules their life and that they need to void every hour (or sometimes even more). The difficult part is that whilst numerous patients present with this complaint, we know that the actual cause of urinary frequency can be markedly different from patient to patient. Unless we accurately determine the cause of the patient's urinary frequency, it will be difficult to implement the most effective treatment.

The most commonly identified causes of urinary frequency include:

1. **Excessive fluid intake** ie drinking 4-5L/day (especially all the Gen Y's carrying their water bottles!)
2. **“Habit”** ie the person is simply voiding frequently out of habit, not because they need to
3. **Secondary to Urgency** ie the person experiences abnormal sensory signalling in the LUT

When excessive fluid intake is found to be the cause of urinary frequency the management is usually quite simple. We first would discuss with the patient about possible reasons for such a high fluid intake ie *“do they always feel thirsty or is it just habit?”* If they regularly feel an unusually high thirst, we would first encourage a medical review in case there is another undiagnosed condition influencing their thirst. In contrast, if their high fluid intake appears to simply be habit, or, as I am finding more and more – a belief that 4L is good for them - then re-education about appropriate fluid intake will probably suffice.

In terms of reason number 2 above - “habit”, occasionally I find that when people complete their bladder diary for me (especially one where I ask them to specifically document how strong an urge they felt before each void) they come back for their follow up having realized for themselves that their frequency is purely habit. In this scenario, I find that they usually come back having fixed themselves saying “I realized that I was going when I really didn't need to, so I've just started holding on longer”.

Then we have frequency that occurs because the person has symptomatic “urgency”. Those of you who have attended the OAB course would remember that we spent a lot of time talking about the concept of “urgency”.

Urgency is now regarded as not only symptomatically, but also physiologically different from just having a “strong urge” to void. It is a form of sensory dysfunction that can have multiple causes, and is therefore not regarded as within the normal spectrum of bladder sensations that should occur during the bladder storage phase. It is an abnormal sensation caused by some form of anatomical/physiological dysfunction.

In people who suffer from ‘urgency’ there is no doubt they often subsequently develop a pattern of voiding frequently. This tends to be a secondary behaviour due to the urgency rather than a primary dysfunction in its own right. When a person suffers from sudden onset ‘urgency’ episodes, the only way we will realistically improve their frequency is to first normalise their bladder sensation.

NOTE: In this scenario we aim to improve the urgency which should then improve the frequency..... we don't try to improve the frequency to reduce the urgency! To do this we may give urgency suppression strategies to manage the urgency when it occurs (eg toe curling, PFC, perineal pressure, counting backwards etc etc), but we also employ a range of strategies such as reduction of bladder sensation irritants, alteration to patterns of fluid consumption, bladder retraining / bladder drills, inhibitory electrical stim, and possibly discussion with their GP/gynae/urologist regarding the prescription of anticholinergics/muscarinics or vaginal oestrogen.

But what about other causes of urinary frequency??

CLINICAL EXAMPLE:

A 55yo female presents to your clinic with urinary frequency of 12 voids per day and 3/night that she finds extremely bothersome. She has a fluid intake of 2.4L per day with an average voided volume of 200mls. She indicates that she doesn't experience “urgency” prior to voiding, but does experience a strong desire to void that would be uncomfortable to continue holding, even though when she voids she often only passes 150-250mls.

It is my experience that this is the type of clinical scenario where we as physios can very quickly jump to the urgency techniques described previously (ie urge suppression, try to hold on, electrical stim to inhibit detrusor overactivity etc). It is also the scenario where I find that doctors often jump to anticholinergics / antimuscarinics.

However..... is this the best management strategy???

I remember when I was in high school and I had a maths teacher explain that we can't assume that the relationship between two variables are always going to be bi-directional. What do I mean by this???

Well, the really nice example my teacher gave was:

“All females are human, but not all humans are female”

I think this is the theory I try to remember with urgency and frequency....

“Suffering from sensations of urgency will often cause a person to void frequently, but voiding frequently isn't always caused by a sensation of urgency”

As we all know, “Bladder Capacity” refers to the total volume of urine that the bladder can hold. It is largely determined by the compliance of the bladder wall, or in other words, the ability of the bladder wall to expand so as to hold increasing volumes of urine. Obviously, a bladder wall that can only stretch to accommodate 200mls is going to result in a much greater daily frequency than a bladder that can stretch to a capacity of 600mls. Anatomically we know that as the collagen content of the bladder increases, the compliance commonly decreases. In addition, collagen content often increases if the bladder has a denervation injury for a period of time.

When we ask our patients to perform a bladder diary, we are attempting to approximate the “capacity” of the bladder and it’s sensory patterning (if we perform a sensation related bladder diary). However, if we consider the situation where a patient may have a high post-void residual (PVR), the volumes listed on their bladder diary will obviously give a false impression of the patient’s true bladder capacity. The bladder diary output isn’t really reflective of the full “Bladder Capacity”, it is only reflective of “Functional Bladder Capacity”.

eg Functional Bladder Capacity: A bladder may be able to expand to hold a normal 500mls, but if every time the person voids they leave a 200ml residual, their functional capacity (the amount of urine production the bladder can receive from the kidneys before reaching capacity) is only going to be 300mls.

Why am I going on about this so much???

The problem is..... “What if the only reason a woman has frequency is because she has a voiding dysfunction with a residual of 150-200mls that is reducing her functional bladder capacity??” It could be that putting her on antimuscarinics or treating her with inhibitory stim may increase her residual to 300mls (due to decreased detrusor contractility from the antimuscarinic), which would then reducing her functional capacity further and worsen her urinary frequency.

Incomplete bladder emptying is a cause of urinary frequency that I find urologists working with men commonly consider due to the high post void residuals associated with an enlarged prostate. However, I personally tend to find it is commonly overlooked in females.

Research Update

Overactive Bladder Symptoms and Voiding Dysfunction in Neurologically Normal Women

Espuna-Pons M, Cardozo L, Chapple C, Sievert K, Kerrebroeck P and Kirby M.
Neurourology & Urodynamics 20120, March, Vol 31, pp 422-428

In March 2012 Espuna-Pons et al published a nice review article on post-void residuals, voiding dysfunction and urinary frequency in women. They began the research paper by summarising some evidence for voiding dysfunction in the female population. They mentioned studies such as: - the EPIC study by Irwin et al 2006, which was an international random sample of persons > 18years which found voiding dysfunction in 19.5% of women; the EpiLUTS study which was a cross-sectional study in the United States, United Kingdom and Sweden that found combined voiding and storage symptoms in 14.9% of women, and combined voiding, storage and post-micturition symptoms in

26.3% of women; and also the study by Lukacz et al 2007 which found that the overall prevalence of PVR >100mls in women presenting with pelvic floor disorders was 11%.

Therefore, whilst symptoms such as detrusor overactivity, stress incontinence and urgency are all obviously a lot more common than high post-void residuals in women, high PVRs do exist, and in those women where they occur, they can be a significant cause of urinary frequency due to reduced functional capacity.

I think that the primary issue for us as physiotherapists is working out whether incomplete bladder emptying could be a cause of our patient's urinary frequency. If it is, we don't want to suggest treatments aimed at reducing voiding ability (eg inhibitory stim or discussion with GP regarding antimuscarinics), rather we need to assist our patients to void to completion. But how do we determine whether this is the issue??

Espuna-Pons et al indicate that the two main ways high PVR is usually diagnosed is by manual palpation of the bladder post void (which they also acknowledge is quite inaccurate), bladder scan (which not all health professionals have access to) or by post void catheterisation (which involves a high UTI risk).

They then spent a large amount of time reviewing whether high PVR can be accurately identified by the subjective history.

Interesting articles they cited included:

1. Al-Shahrani et al 2005 who found that voiding symptoms alone (eg sensation of incomplete emptying, hesitancy, straining to void) were poor at identifying women with high PVR. This is probably not surprising considering that the normal bladder does not pick up bladder filling until ~150mls. Therefore, if the residual is only 120mls, it is likely that the person will not realise they are incompletely emptying.
2. Lowenstein et al 2008 who also found that voiding symptoms alone were not a good indicator.

However, whilst this doesn't look promising, Espuna-pons et al then cited a number of interesting studies that looked at whether symptom clusters could be more reliable.

The first was a study by Fitzgerald et al 2001. Fitzgerald et al found that in women with urgency/frequency or urge incontinence:

- 5% of patients with urgency and/or frequency have a PVR >100mls, and....
- 10% of patients with urgency and urge incontinence have a high PVR >100mls

What Fitzgerald also found was that in patients with urgency, frequency and UUI, using a combined symptom cluster criteria of co-existing pelvic organ prolapse \geq stage 2, symptoms of voiding difficulty and absence of stress incontinence – that you could predict 82% of patients who have an elevated post-void residual >100mls.

Interestingly, Lukacz et al 2007 who also found that anterior wall or apical prolapse, a feeling of a vaginal bulge, pelvic pressure, splitting of urinary stream and the absence of stress urinary incontinence were associated with PVR >100mls when they occurred together, but only prolapse at or beyond the hymen was a significant factor on logistical regression.

Finally, Milleman et al 2007, showed that 19% of women with urgency, frequency and urge incontinence had an elevated post-void residual >100mls, and that age >55years, prior incontinence surgery, history of multiple sclerosis and prolapse grade \geq stage 2 were also associated with high PVR.

CLINICAL APPLICATION TIP

Whilst most women with OAB (urgency and frequency symptoms) don't always have a high post void residual, there is a certain percentage that do.

There seems to be a consistent theme emerging that patients who present with urinary frequency +/- urgency, who also present with symptoms of voiding dysfunction, Prolapse \geq stage 2, and minimal stress incontinence may have elevated PVR as a contributing factor to their frequency. In these circumstances it is important that we don't suggest management options that may worsen their post-void residual.

So if we think our patient may have a high post-void residual what do we do?

This is probably a very long discussion to be had for another day. The main point I hoped to get across in this newsletter is the cluster symptoms that should alert us to the high likelihood of post-void residual being a cause of urinary frequency. However.... a few things I would suggest is.....

- In people with multiple sclerosis there is research to show that the Queen's Square suprapubic bladder stimulator is useful in reducing post-void residuals. It is about \$70 to purchase and is available from Malem Pty Ltd in Queensland.
- Personally, I have also found the Queens square bladder stimulator helpful in some of my patient who have had damage to their bladder following gynae surgery (eg resection of endometriosis from the bladder)
- Constipation is known to be a significant influence on post-void residual. Improving constipation may reduce outflow obstruction through the urethra and thereby reduce residuals.
- Both anterior vaginal wall prolapse ("classic cystocele") and apical prolapse (classic "Uterine" Prolapse) is associated with residual. Whilst we can easily get into a habit of telling patients to 'lean slightly

forward' to assist bladder emptying, in the case of uterine prolapse this may result in even greater urethral closure pressure due to the uterus compressing the urethra against the pubis. In this case, some women will void better leaning backwards.

- Finally, in the tweets this month I gave links to the various research projects that have looked at the concept of “hovering” regarding voiding function. Whilst we all have historically told our patients not to “hover” (including myself) I would encourage you to have a read over those (the links are available at the end of the newsletter).