comments

OVERACTIVE BLADDER: SYMPTOM OR SYNDROME?

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Over a quarter of a century ago, Patrick Bates coined the phrase 'the bladder is an unreliable witness', meaning that no matter what the underlying pathophysiology, the bladder has a limited means of expressing its own pathology. These lower urinary tract symptoms (LUTS) used to be categorized as irritative or obstructive; the implication of this classification is that symptoms define the underlying pathology, i.e. obstructive symptoms are caused by urethral obstruction and irritative symptoms by involuntary detrusor contraction (IDC). We now know that the relationship between symptoms and pathophysiology is, at best, imperfect, and cannot rely on symptoms to make an accurate diagnosis. Symptoms then serve two functions. First, they define what it is that is bothering the patient, and second, they alert the clinician to the possibility of a differential diagnosis. The latter is important not only because different conditions may respond better to certain treatments, but also because some conditions, such as bladder cancer, demand specific treatment lest there be severe consequences.

The ICS recently defined a new term, the overactive bladder syndrome (OAB), as 'urgency, with or without urge incontinence, usually with frequency and nocturia . . . if there is no confirmed infection or other obvious pathology' [1].

Symptom or syndrome? Does it make a difference what it is called? For both practical and theoretical reasons there is an important difference between symptoms and syndromes. A syndrome is a set of signs or a series of events occurring together that often indicate a single disease or condition as the cause. Down's syndrome is a case in point; it is characterized by a constellation of signs and symptoms (mental retardation, characteristic physical features, etc.) that are almost always a result of a trisomy 21 chromosomal abnormality. The term Down's syndrome and trisomy 21 are nearly synonymous and when either term is used, most clinicians know exactly what is meant. As the cause and natural history of Down's syndrome are known, once the diagnosis is made, one can focus on treatment and research.

This is not so when the words 'overactive bladder' are used. When the term OAB is used neither a specific condition nor a cause is obvious. Indeed, the definition of OAB implies that if a specific cause is known, e.g. prostatic obstruction, the term OAB should not be used. Yet prostatic obstruction is the cause of OAB (or at least associated with it) in the vast majority of men with LUTS. If the definition of OAB is limited to those without known pathology, we are left with no simple word or words to describe OAB symptoms in those with such known pathology.

Why does it matter what you call the symptoms? Is it not just semantics? It matters because if we think of OAB as a symptom complex of unknown aetiology and simply seek treatments that ameliorate the symptoms without determining the cause, we stifle research and creativity. The current explosion of OAB treatments are all based on the assumption that the cause of symptoms is IDC, yet IDC is only detected in about half of patients with OAB by conventional techniques [2]. So ingrained has this thinking become that most phase 3 trials of new pharmaceuticals for OAB do not even include urodynamic studies and many, if not most, clinical algorithms for treating OAB recommend empirical treatment with behavioural techniques and anticholinergic medications, and reserve urodynamic studies only for treatment failures.

What is wrong with empirical treatment? Inherently, nothing, particularly if it works well and/or if the condition being treated is self-limited. Uncomplicated UTI in young women is such an example. However, OAB is neither easy to treat nor is it usually self-limiting. Furthermore, there are no standardized outcome measures, so it is nearly impossible to define success and failure with any degree of confidence. Thus many patients who have only marginal and/or transient improvements are considered successes when they really are not and clinicians (and the patients) think that they are doing better than they really are.

Despite the perception that the current medications offer a success rate of >60% most studies show that <25% of patients remain on their medication for a year, despite persistent symptoms. Furthermore, most clinicians recognize that OAB is much more difficult to treat than the studies would suggest, and that a large percentage of patients never have good success. In brief, we do not understand the cause of OAB symptoms very well and the treatments are not very effective.

What can be done to improve this situation? One way is to refocus efforts on the basics: (i) Consider the symptoms of OAB to be the outward manifestations of an underlying condition, not the components of a syndrome with an unknown cause. (ii) Look for the underlying condition. Consider patients with OAB symptoms; some have polyuria because they drink too much (because their doctor told them to drink at least eight glasses of water a day) and thus they have urinary frequency and, if they wait too long (because of social expediency) they have urgency. For those with comorbidities like BPH it takes little more for them to develop urge incontinence. Other patients with OAB have

IDC and prostatic obstruction, and are cured of their symptoms (and IDC) by TURP.

Some patients have all the same symptoms, but there are no IDCs by conventional cystometry. Do these patients have IDC that cannot be measured, do they have IDC in their daily life that they can control when their attention is focused during a urodynamic study, or do they have something else? Some patients have IDC that they are unaware of and cannot control at all; these patients void uncontrollably. Other patients can sense the onset of IDC and then contract the sphincter, interrupt the stream and abort the detrusor contractions. These patients have urgency, but not urge incontinence. Will the latter patients fare better with behavioural techniques and the former with anticholinergics or neuromodulation? We do not know.

Some patients have neurological conditions associated with their symptoms, but the cystometric characteristics of neurogenic IDC do not appear to differ from those in patients with no neurological disorders [3]. Do they have a common cause that requires similar therapies? We do not know.

On the basis of the degree of awareness and control that a patient with OAB can demonstrate during cystometry, we proposed a new classification system for OAB that considers all of these things [4]. In Type 1 OAB, the patient has sufficient control to prevent the onset of IDC. In type 4 the patient voids uncontrollably and is unable to stop. Type 2 and 3 show varying degrees of control. Can Type 4 patients, with treatment, become Type 1 patients? We do not know. This classification may prove useful or it may not. The important point is that we need to develop clinical and research methods that allow us to understand the subtle differences between patients and why in one patient the same treatment fails that cures another. Is it in the genes, the structural anatomy, the neurophysiology, the muscles or what? Is it all of these things? To begin to answer these questions we need to (i) use existing techniques to accurately characterize the underlying pathophysiology; (ii) develop more specific outcome measures to accurately measure (relevant) outcomes; and (iii) do the research. To see how far the field has come, read the report of the Second International Consultation on Incontinence [5]; the expertise to accomplish these things has been present for over three decades, but we have not done so yet; it is time that we did.

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RADICAL PERINEAL PROSTATECTOMY

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Hugh Hampton Young, the first Professor and Chairman of Urology at Johns Hopkins, is recognized as performing the first radical perineal prostatectomy for cancer of the prostate on 7 April 1904 [1]. As detailed by W.W. Scott, Young developed the operation for removing the prostate via the perineum to relieve urinary obstruction caused by presumed benign disease in 1903, but encountered two patients with small malignancies; he wrote, 'I was struck by the fact that had the entire prostate gland been removed with its capsule, it would have been possible to cure these patients. As a study of the literature revealed that no such radical operation had ever been attempted, I made careful sketches of what I thought would be necessary and showed them to my chief, Dr William S. Halsted, whose reputation was world-wide because of a very radical operation for cancer of the breast with which he had cured a large percentage of the patients brought to him. After examining the (first) patient, Dr Halsted carefully reviewed my sketches. He appeared greatly impressed, strongly advised me to carry out the operation, and said he would like to assist.'

For many years thereafter, radical perineal prostatectomy was the accepted surgical approach for treating patients with confined prostatic malignancies and it was not until 1945 that the retropubic approach was reported by Millin [2]. Because of intraoperative bleeding and incontinence, retropubic prostatectomy had limited application and it was not until Walsh and Donker [3] introduced surgical principles reducing blood loss and preservation of the neurovascular bundles that radical retropubic prostatectomy became the most common procedure for treating patients with localized disease.

The recent introduction of laparoscopic radical prostatectomy has brought forward another approach, adding to the debate, but because of the small incision required, the short operative duration and the limited recuperative time, the perineal approach continues to offer many benefits. The indications for radical prostatectomy, preoperative assessment and anaesthesia requirement are similar for all procedures. The operative duration for radical perineal prostatectomy is short, with the mean (range) in my experience being 60 (35–100) min and the blood loss 300 (100–1200) mL. Most patients can be discharged 2 days after surgery. It has been my practice to maintain catheter drainage for 2–3 weeks, although others have removed the catheter earlier. The outcomes are similar for the three procedures. Weldon *et al.* [4] reported their experience in 220 patients undergoing radical perineal prostatectomy; continence returned in 23% of patients by 1 month, in 56% by 3 months, in 90% by 6 months and in 95% by 10 months. Potency varies and is not only related to the ability to spare nerves but also to the age of the patient and preoperative status. It was also reported that of those with good preoperative potency in whom nerve-sparing procedures were used, 70% remained potent [4]. Potency returned in half of patients at 1 year and in 70% at 2 years. There was no statistically significant difference between patients who underwent unilateral or bilateral nerve-sparing procedures (68% vs

73%, respectively). Frazier *et al.* [5] reported on patients who underwent nerve-sparing radical prostatectomy; 77% were potent at >1 year afterward.

Another area that has been closely assessed when comparing various radical prostatectomy procedures is margin status. Most studies have found no difference in the rate of positive margins in patients undergoing radical perineal prostatectomy compared with those having another approach. Weldon *et al.* [6] reported the results of 200 consecutive radical perineal prostatectomies and in those with clinical T1 and T2 disease, the overall rate of positive margins was 44%. The rate of positive margins was 7%, 16% and 25% for the apex, posterolateral and anterior prostate, respectively. These values were compared with the rates in a series of patients undergoing radical retropubic prostatectomy. The perineal group had fewer positive apical (7% vs 10–48%) and posterolateral margins (16% vs 26–44%), but there was a higher incidence of positive anterior margins in the perineal group (25% vs 2–10%). In another series, each approach had a specific high-risk location of positive margins; the apex for retropubic, the bladder neck for the perineal and posterolateral for the laparoscopic approach [7]. Other studies comparing these approaches have shown no differences in outcome [8,9].

A recent report [10] suggested that patients undergoing radical prostatectomy experience fecal incontinence more often than had been recognized. Although this observation has not been my experience, Bishoff *et al.* [10] reported that 10% of patients undergoing retropubic procedure and 15% undergoing a perineal operation experience fecal incontinence at least monthly. A more recent report [11] suggests that fecal incontinence and bowel-related symptoms are more prevalent after radical perineal prostatectomy than at baseline, yet resolve in most patients with time soon after surgery.

In summary, the continued interest in radical perineal prostatectomy for treating localized prostate cancer has been facilitated by the current emphasis on reducing medical costs, by identifying more patients with localized disease, the selected use of lymphadenectomy, and the use of laparoscopic techniques for lymph-node sampling. At times the procedure is preferred in those patients who have undergone previous inguinal hernia repair using synthetic, nonabsorbable mesh, in kidney transplant recipients, in the morbidly obese and in those who have had previous complex pelvic surgical procedures. The technique provides a cost-effective, safe and effective means of treating patients with localized prostate cancer.

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MY RADICAL PROSTATECTOMY: IT WILL NEVER HAPPEN TO ME – BUT COULD IT?

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I have long suspected that there seems to be a disproportionate incidence of illness related to the practitioners' speciality. Why are psychiatrists so odd? Why do dermatologists scratch themselves so frequently? However, I did not even contemplate that I might confirm my hypothesis. It came as a considerable shock therefore, just as I had decided to take early retirement from an increasingly frustrating NHS after 25 years as a consultant urologist, to find that my PSA level was 4.9 ng/mL. Suddenly the prospect of a few years of private practice followed by many more years of blissful retirement was shattered as I contemplated a much shorter future. Then reality took over and I reassured myself that the results from the previous 2 years had been fine, and that this was probably an anomaly, and even if it were not there was still an 80% chance that this was just a result of benign enlargement, although I had no symptoms. It would therefore be sensible to repeat the test in 3 months.

This I did and was disappointed to find that the level was then 6.1 ng/mL; clearly action was now needed. The interesting question of whom does a surgeon approach when in need did not arise. I immediately spoke to a former trainee and friend (and associate editor of this journal!) to whom I had taught Millin's

operation and who had subsequently returned the favour by teaching me nerve-sparing radical prostatectomy.

A TRUS-guided biopsy was arranged a few days later, under local anaesthetic, and was surprisingly atraumatic. With each biopsy there was a sharp sensation at the tip of the penis, but nothing to complain about, and in >15 min the procedure was complete. I was given antibiotics intravenously, along with a metronidazole suppository and supplied with more oral antibiotics for 5 days. Despite this, at the end of the course of tablets I developed rigors and felt extremely unwell. I re-started oral antibiotics and had an intravenous cannula inserted into my arm, and arranged a daily infusion of gentamicin for 5 days before starting my normal daily activity! I also had a series of TRUS scans which showed a collection and possible small abscess within the prostate, which slowly resolved over a couple of weeks.

By this time I had received the news that I had an adenocarcinoma of the prostate, Gleason 3+4, with a multifocal pattern. I took less than 30 s to decide on what treatment I should have. Watchful waiting was unacceptable as the rate of progression was too high. Radical treatment was necessary. During my consultant practice I had become increasingly disappointed by the results of radiotherapy in both prostate and bladder cancer, and formed the opinion that the reported rates of long-term survival after conventional radiotherapy for prostate cancer have been subjected to considerable 'spin' and were unreliable. The rates for brachytherapy appeared to me to indicate similar results. Also, I felt instinctively that I would be unhappy with the offending organ still in place and thus reflected what many patients have said to me over the years "Better out than in". Surgery also allowed for the possibility of subsequent radiotherapy if there was a local recurrence. Finally, I am impressed by the fact that many fewer patients have large local recurrences of tumours in the USA, where radical prostatectomy has been the most common treatment, and this contradicts the idea that surgery should only be used to achieve a cure. I believe that there is a case to be made for saying that even if there is a recurrence after surgery the quality of life issues have to be considered. Hence no choice!

Surgery was therefore arranged; I was admitted to hospital on Monday lunchtime and had a radical retropubic prostatectomy later in the afternoon. For 24 h I was slightly nauseous, but felt fine with very little discomfort. I took no analgesia at all from 48 h. By Thursday I was ready for discharge, although because I lived some way from the hospital I decided to stay until the Saturday and have the clips removed before going home, with the catheter still *in situ*.

Having the catheter in for 2 weeks was tedious, not because it was painful, but it restricted my activity by being irritating and there was a tendency to bleed slightly around it. I was mightily relieved therefore when it was time to have it removed, which I did myself at home. I was not surprised that it did not wish to fall out when the balloon was deflated (I had seen this problem with my patients) but after walking around for ≈15 min the tube painlessly delivered itself. I had no problems at all with continence apart from an occasional minuscule leak when getting up from a chair. The flow rate was excellent and with no discomfort. However, within about a week I noticed a reduction in flow and an increase in frequency that rapidly deteriorated, and I virtually developed retention of urine. This was a most unpleasant experience. However, my excellent Associate Specialist colleague responded to my call and came and passed a urethral catheter. This was not as bad an experience as I had anticipated, with only slight discomfort as the bladder neck was negotiated, but great relief immediately. It was decided to leave the catheter for a few days, but unfortunately after it was removed the symptoms of obstruction recurred. I was therefore readmitted for a day and had a bladder neck dilatation for a tight bladder neck obstruction. It was also decided at that stage that it would be a good idea to self-catheterize for some time. This I did twice a day for a month and then once a day for a few weeks. I have to say that this was very easy, after plucking up the courage to do it for the first time. I tried using local anaesthetic gel as well as the self-lubricating catheters, but I did not find that it was necessary to use the anaesthetic, as there was no significant discomfort. Unpleasant yes, and I am unclear why some should choose to do it for fun!

During this period I also tried taking an α-blocker, thinking that it would have very little effect as the bladder neck mechanism was no longer intact. To my surprise there was a significant increase in the flow rate. Not really believing it, I stopped and started several times and the effects were consistent. Gradually I phased out the catheterization and also stopped the α-blocker; I am pleased to say that voiding is now completely normal with total continence. As regards erections, things are slowly beginning to improve.

The operation brought forward my scheduled early retirement by 3 months but I was well enough to return to private surgical practice at 5 weeks after surgery. In the subsequent months I have had no problems and with the PSA remaining at <0.1 ng/mL, things look optimistic.

On reflection, what has been learnt from this experience? Clearly for someone who had not had a single day off for illness in 25 years as a consultant, a sudden realisation of my mortality concentrated the mind. It made me reassess the problems that patients have in coming to terms with similar situations, and I can more easily empathise with them. The whole process was amazingly atraumatic and I can now honestly answer the question that is frequently put when discussing the options for treating prostate cancer – 'What would you do in this situation?' It has confirmed my prejudices that surgery is the best answer, and although I never recommend a specific treatment to an individual patient, for me '*res ipse loquitur*!'

IMPROVING TREATMENT OUTCOMES IN TESTICULAR CANCER: STRATEGIES TO REDUCE TREATMENT RELATED MORBIDITY

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For most men with testicular germ cell tumours (TGCTs) current treatment strategies are highly successful. Although work continues to intensify treatment in poor-prognosis and salvage groups, a focus is developing for research on long-term treatment effects and on optimizing treatment plans. This change in focus is

reflected in several recent publications which report the long-term effects of treatment and ways in which treatment can be modified to make treatment either more efficient or less toxic. A selection of these papers is the focus of this comment.

A recent paper from the Royal Marsden Hospital illustrates the increasing concern about the possible long-term effects of cancer treatment in patients with TGCTs. In our study of 998 men with at least 5 years of follow-up we identified an increased risk of a cardiac event (sudden death, myocardial infarction or anginal chest pain). This was present after both chemotherapy (age-adjusted relative risk, ARR, 2.89, 95% CI 1.34–6.25) and radiotherapy (2.32, 1.06–5.05) compared with patients being treated by orchidectomy alone. Although the specific cause of this risk remains to be determined, taken with previously reported risks of second malignancy these reports have given impetus to strategies to minimize the exposure to therapeutic interventions [1].

The area of greatest controversy in this respect is in patients with stage I seminoma. For many years the standard management was adjuvant radiotherapy. Previous trials lead by the MRC showed that para-aortic radiotherapy is as good as 'dog-leg' radiotherapy for preventing recurrence. Recently the preliminary results of the MRC TE18 trial have been presented at several international meetings [2], showing that a dose of 20 Gy in 10 fractions (compared with a previous dose of 30 Gy in 15 fractions) is sufficient treatment. As most para-aortic radiotherapy fields include a portion of the kidney and the radiation tolerance dose of the kidney is ≈20 Gy this is potentially a very significant difference in limiting renal toxicity. Reducing the radiation dose should also deliver similar benefits in terms of bowel toxicity.

Despite these developments the continuing concern in using radiotherapy in this patient group is fuelling a re-examination of surveillance strategies. Several groups explored surveillance in the 1980s but the lack of reliable tumour markers and the need for prolonged follow-up, including cross-sectional imaging, meant that this strategy has not gained universal acceptance. One problem has been the difficulties of distinguishing between patients at high risk where adjuvant treatment would be justified, and low risk where a 'watching' policy might be more appropriate. This deficiency has been recently corrected in a pooled analysis of four surveillance studies including over 600 patients [3]. This study identified larger tumours (either as a continuous variable or defined as >4 cm), rete testis invasion and vascular invasion as univariate prognostic factors for relapse. The first two of these remained positive on multivariate analysis and form the basis of a prognostic index. Patients with both rete testis invasion and a tumour of >4 cm had a 31.5% relapse risk, whilst with no risk factors there was a 12% relapse risk.

In patients with stage I nonseminoma, avoiding additional treatment by a surveillance policy has been widely adopted in the UK. However, patients with vascular invasion have a 40–50% risk and are often offered adjuvant chemotherapy. Most of these relapses occur early and a recent paper by Lassen *et al.* [4] suggests that functional imaging with fluoro-deoxyglucose positron emission tomography (PET) could detect most patients who subsequently relapse. In their study, 40 patients with stage I nonseminoma GCTs underwent PET during staging; 10 subsequently relapsed, seven of whom had a positive PET scan at diagnosis. This suggests that ≈70% of patients who would relapse could be detected at diagnosis by PET. This has several implications; first, if PET were used it would mean that even in high-risk patients only a small proportion would subsequently relapse on surveillance. Second, for those who have a positive scan, less chemotherapy could be used, as these must be the patients who are currently cured by two cycles of adjuvant chemotherapy. These issues are being studied in more depth by the CRUK TE22 protocol, which commenced in the UK last year. This study aims to confirm the data from Lassen *et al. and* endeavour to determine whether these results can be replicated in a multicentre study. The study will recruit 135 patients with stage I nonseminoma GCT with vascular invasion, each of whom will undergo PET. Patients with a negative PET scan will be managed by surveillance, whilst those with a positive scan will be treated at the investigators' discretion. This important study will define the value of PET in this situation with a high degree of accuracy and will help to determine its usefulness in other areas of managing GCTs.

In metastatic TGCT, a major advance in reducing treatment was the finding of the MRC/EORTC TE20 study that three cycles of bleomycin/etoposide/carboplatin (BEP) is equivalent to four cycles in patients with good-prognosis metastatic GCT. A recent update of this study confirmed the long-term quality of life benefits of three rather four cycles of BEP [5]. It also suggested that fractionating the chemotherapy over 5 rather than 3 days reduced neurological toxicity. Although this was not significant when three cycles were used there was a significant difference in patients receiving four cycles. It could be argued that this is of minor importance as good-prognosis patients should be now receiving three cycles of BEP chemotherapy. However, four cycles of BEP remain standard for intermediate prognosis and more advanced patients, and we must now question whether the common UK practice of 3-day BEP is the correct one in this patient group.

In conclusion, with the continuing success of treatment for TCGT a trend is developing in the good-prognosis groups to optimize treatment, with the aim of limiting the long-term effects and burden of treatment. I fully expect efforts in this area to continue to be fuelled by further reports on the long-term toxicity of treatment. For example, there is a growing interest in reducing the extent of primary surgery, as illustrated in a recent BJU article on the role of testis-preserving surgery. Further modification of surveillance and chemotherapy can also be expected. For many years specialist care has been advocated for testis cancer to ensure the best cancer cure rates. This is likely to remain true but also, increasingly, it is likely that specialist care is required to ensure that patients are not over-treated.

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