

Association of Lower Urinary Tract Symptoms/Benign Prostatic Hyperplasia with Sexual Dysfunction

Bang-Ping Jiann, M.D.

Division of Urology, Department of Surgery, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a common condition in older men. The prevalence of histologic BPH in autopsy studies rises from approximately 20% in men 41-50 years old to 50% in men 51-60 years old, and to over 90% in men older than 80 years [1]. Enlargement of the prostate may result in subsequent bladder outlet obstruction, which can produce lower urinary tract symptoms (LUTS) or complications such as infection, vesical stones, urinary retention, and deterioration of renal function. LUTS are divided into obstructive (voiding) and irritative (storage) components. Obstructive symptoms include slow stream, intermittency, hesitancy and straining and irritative ones include increased daytime frequency of urination, nocturia, urgency and urge urinary incontinence. It is estimated that 25 to 50% of men with BPH have LUTS and 50% of men with LUTS have bladder outlet obstruction due to BPH or other urethral conditions [2].

Until recently, it was widely assumed that male sexual dysfunction was a natural consequence of ageing in men. The results of a population-based study of men 40 to 70 years old, the Massachusetts Male Aging Study (MMAS), demonstrated the prevalence of complete erectile dysfunction (ED) increased from 5% for men 40 years old to 15% for those 70 years old with a combined prevalence of 52% for minimal, moderate and complete impotence in this population [3]. The Global Study of Sexual Attitudes and Behaviors (GSSAB) is an international survey of various aspects of sex and relationships among adult men 40-80 years old from 29 countries. Based on GSSAB data, the prevalence of erectile difficulty ranged from 12.9 to 28.1%, lack of interest in sex ranged from 12.5 to 28.0% and premature ejaculation ranged from 20.7 to 30.5% in different regions [4]. The MMAS, GSSAB and other epidemiology studies all support the fact that male sexual dysfunction, including ED, ejaculatory dysfunction and hypoactive desire, are very common problems in men.

LUTS/BPH AND SEXUAL DYSFUNCTION

Because both LUS/BPH and sexual dysfunction occur more frequently in the elderly, it is difficult to ascertain whether LUTS/BPH contributes to sexual dysfunction. Many observational studies have shown a significant relationship between them. The link between LUTS and sexual dysfunction was confirmed by the Multi-National Survey of the Ageing Male-7 (MSAM-7) which included 12,815 men 50-80 years old in the United States and 6 European countries [5]. This study showed

that most men over 50 years old were sexually active despite a strong decrease in mean sexual intercourse/sexual activity with LUTS severity (ranging from with 7.5 episodes of sexual activity per month in men in their sixties to 3.2 per month in men in their eighties). ED assessed by the International Index of Erectile Function-Erectile Function domain score was strongly related to LUTS severity. After controlling for age, LUTS severity was by far the strongest predictor of ED, with an OR (odds ratio) for severe vs. mild LUTS of 8.90 (6.85-11.55) followed by diabetes, 3.01 (2.60-3.29); cardiac disease, 2.17 (1.92-2.46); hypertension, 1.83 (1.66-2.01) and dyslipidemia, 1.57 (1.41-1.73). Ejaculatory dysfunction (reduced amount of ejaculate and pain/discomfort on ejaculation) assessed by the sexual section of the Danish Prostate Symptom Score was also strongly related to LUTS severity [5].

Another recent population-based study investigated the relationship between LUTS and sexual dysfunction in men 40 to 65 years old in Denmark and also showed that LUTS was an independent predictor of ED in multivariate logistic regression analysis [6]. Analysis of baseline data from the randomized, placebo-controlled, Medical Therapy of Prostatic Symptoms (MTOPS) trial also indicated a significant relationship between LUTS and erectile function, ejaculation, overall sexual satisfaction, and sexual desire after controlling for age, hypertension, dyslipidemia, and diabetes [7]. Interestingly, sexual function domains in the MTOPS trial were also significantly related to two objective variables of BPH progression, peak flow rate and prostate size [7].

POSSIBLE LINKS BETWEEN LUTS/BPH AND SEXUAL DYSFUNCTION

Because the risk factors and mechanism of ED and LUS/BPH are multifactorial, it is unlikely that there will be a single common pathophysiology underlying the development of both conditions in an individual [8]. Some evidence for a pathophysiological link between LUTS/BPH is found in a rabbit model of bladder outlet obstruction [9]. There was an imbalance between nitric oxide and endothelin-1, as well as adverse ultrastructural changes in the corpus cavernosum, suggesting that this contributes to impairment of nitric oxide-mediated smooth muscle relaxation [9].

A recent hypothesis proposed that increased rho-kinase activity in smooth muscle leads to increased sensitivity to calcium, a heightened response to mediators of smooth muscle contraction, and tissue changes in the prostate, urinary tract and penile smooth muscle [10]. Clinically, this would translate into increased bladder neck tone causing LUTS and increased penile muscle tone leading to ED [10].

α 1-adrenergic receptors are known to play an important role in mediating the tone of smooth muscles in various tissues. It has been

suggested that α 1-adrenergic receptors are upregulated in patients with LUTS associated with BPH, resulting in increased smooth muscle tone in the prostatic capsule and bladder neck [11,12]. Penile detumescence and erection are dependent on the balance between contraction and relaxation of the corpus cavernosum smooth muscle [13]. Adrenergic-mediated contraction of smooth muscle may also be regulated by rho-kinase [14], which has been found in the prostate [15] and vas deferens [16]. Alteration in α 1-adrenergic receptor-mediated smooth muscle tone and its regulators may be a common component involved in LUTS associated with BPH, ED and ejaculatory disorder.

It is well recognized that the development of BPH requires the presence of androgens and that the marked reduction in serum testosterone caused by chemical or surgical castration reduces prostate volume. Androgen receptors, which are present in both the stroma and epithelium of the prostate as well as in most blood vessel endothelial cells, smooth muscle cells, and fibrocytes [17], may play a role in the interaction between the stroma and the epithelium of the prostate. Testosterone is required for pubertal acquisition of gender characteristics as well as adult sexual behavior and functional capacity, including libido, ejaculation, and spontaneous erections. With aging the production of testosterone declines and its bioavailability is reduced. Additional studies are needed to assess whether alterations in sex hormone levels and their receptors play a role in the pathophysiology of BPH and sexual dysfunction.

IMPACT OF 5 α -REDUCTASE INHIBITORS ON SEXUAL FUNCTION

Dihydrotestosterone (DHT) is the androgen primarily responsible for prostate growth and enlargement. Inhibition of the enzyme 5 α -reductase, which catalyzes the formation of DHT, represents another approach to treating LUTS associated with BPH. Two 5 α -reductase inhibitors, finasteride and dutasteride, are currently used in the treatment of symptomatic BPH in men with enlarged prostates.

Ejaculation disorders associated with finasteride range from decreased volume of the ejaculate to failure of ejaculation. Randomized clinical trials in men with symptomatic BPH demonstrate an incidence of ejaculatory dysfunction associated with finasteride ranging from 2.1 to 7.7% (Table 1) [18-20]. Most subjects experienced the onset of ejaculatory dysfunction, like other sexual dysfunction, within the first 9 months of treatment with finasteride. Nearly all trials involving finasteride in the treatment of LUTS resulting from BPH demonstrate a statistically significant increase in ED. In the PROSPECT trial, the incidence of ED in the finasteride and placebo groups was 15.8% and 6.3%, respectively

($P < 0.01$) [21].

Dutasteride, an inhibitor of type 1 and 2 5 α -reductase isozymes, is used for treatment of BPH, providing greater suppression of DHT than finasteride. When compared with patients receiving placebos, patients treated with dutasteride 0.5 mg once-daily (OD) in 3 randomized, double-blind, clinical trials experienced significantly higher incidence rates of ED (7% vs. 4 % for the placebo group), ejaculatory dysfunction (2% vs. 1% for the placebo group), and hypoactive desire (4% vs. 2% for the placebo group) (Table 1) [22].

IMPACT OF α 1-BLOCKERS ON SEXUAL FUNCTION

In the 1980s, Lepor and associates recognized that prostatic smooth muscle tension was mediated by α 1-adrenoreceptors which led to the development of α -blockers as a treatment for LUTS. This dynamic component of prostatic obstruction accounts for approximately 40% of outflow obstruction due to BPH [23]. There are 4 α -blockers approved by the U.S. Food and Drug Administration to treat LUTS, doxazosin, terazosin, tamsulosin and alfuzosin. At therapeutic doses, these 4 α 1-adrenergic receptor blockers have demonstrated similar effectiveness in relieving LUTS associated with BPH [24].

Terazosin appears to be associated with a relatively low incidence of ejaculatory dysfunction. In the Veterans Affairs Cooperative Study the incidence of ejaculatory dysfunction with terazosin in a study population of 610 men was 0.3% [18]. In the Hytrin Community Assessment Trial (HYCAT), it was also low, 1.4%, but significantly different statistically from a placebo ($P = 0.01$) [25]. The incidence of sexual side effects was low and similar to those in the groups taking alfuzosin 10 mg OD (ED, 1.5%; loss of ejaculation, 0.6%) and the placebo group (ED, 0.6%; loss of ejaculation, 0%) [26]. During treatment with doxazosin, the rates of ED and ejaculatory disorder were generally comparable to those observed with placebo treatment [27]. In 2 randomized, double-blind, placebo-controlled studies in men with BPH, treatment for 13 weeks with doxazosin, which was titrated to 8 mg OD, resulted in significant improvement in sexual function for those patients with sexual dysfunction at baseline [28].

Perhaps because of its higher pharmacological selectivity for α 1A-receptors in the bladder neck, seminal vesicles, and vas deferens, however, tamsulosin is associated with a significant incidence of ejaculatory dysfunction. A 3-year European follow-up study reported a cumulative rate of ejaculatory dysfunction with tamsulosin of 5.4%, but only one patient (0.3%) discontinued the medication because of abnormal ejaculation [29]. In a combined analysis from double-blind clinical trials with tamsulosin, the frequency of overall abnormal ejaculation

Table 1. The sexual side-effects of finasteride and dutasteride

| | VA Study [18] 1-yr follow-up | | PROWESS Study [19] 2-year follow-up | | PLESS Study [20] 4-year follow-up | | Dutasteride Trials [22] 2-year follow-up | |
|-----------------------|---------------------------------|------------------------|--|-------------------------|--------------------------------------|-------------------------|---|-------------------------|
| | Placebo (n=305) | Finasteride (n=310) | Placebo (n=1591) | Finasteride (n=1577) | Placebo (n=1376) | Finasteride (n=1384) | Placebo (n=1555) | Dutasteride (n=1605) |
| Decreased libido | 1.0% | 5.0% | 2.8% | 4.0% | 2.6% | 2.6% | 2.1% | 4.2%* |
| Erectile dysfunction | 5.0% | 9.0%* | 4.7% | 6.6%* | 5.1% | 5.1% | 4.0% | 7.3%* |
| Decreased ejaculation | NA | NA | NA | NA | 0.5% | 1.5% | NA | NA |
| Ejaculation disorder | 1.0% | 2.0% | 0.6% | 2.1%* | 0.1% | 0.8%* | 0.8% | 2.2%* |

* $P < 0.05$; NA: not available.

(including retrograde ejaculation, ejaculation failure, and ejaculation decrease) for a 0.4 mg dose and a 0.8 mg dose was 8.4% and 18.1%, respectively [30].

Except for the low risk of ejaculatory disorders that may be induced by treatment with α -blockers, most data suggest a beneficial role for α -blockers with regard to all aspects of sexual function. The tools actually available do not take into account clearly the ejaculation phenomenon; they are more focused on the erection dimension. The European study demonstrated improved total sexual function in patients treated with tamsulosin compared with a placebo [31], and a study of alfuzosin treatment in a general practice setting recorded a significant improvement in patients' perceived sexuality after 12 months of treatment [32]. In an observational study of men with BPH, treatment with doxazosin for 1 month resulted in a significant improvement in sexual function compared with that at baseline, especially for those with moderate-to-severe ED at baseline [33]. These studies provide evidence that treatment of LUTS with α -blockers in men with BPH may in fact improve erectile function.

Two mechanisms have been proposed to explain patients' improved sexual function following treatment for LUTS/BPH with α -blockers. First, as the symptoms become less bothersome, patients may thus be better able to enjoy other facets of life [34]. Alternatively, inhibition of the α 1- and α 1D-adrenoreceptor subtypes that predominate in cavernosal smooth muscle should facilitate penile erection [35]. The mechanism by which α -blockers affect the erectogenic response is via relaxation of the smooth muscle in the penile arteries or the corpora cavernosum, thus improving the inflow of blood [35].

SEXUAL DYSFUNCTION FOLLOWING TRANSURETHRAL PROSTATECTOMY

The gold standard for the surgical treatment of BPH remains the transurethral prostatectomy (TURP). The American Urological Association Cooperative Study enrolled 3,885 patients from 13 institutions and reported a 13% incidence of ED following TURP [36]. However, the Veterans Affairs Cooperative Group Study on TURP demonstrated that the incidence of ED in men with moderate LUTS/BPH who underwent TURP was lower than that for those in the watchful waiting group after follow-up for 3 years [37]. In a study examining 127 men's self evaluations of sexual dysfunction after TURP, 54% of the responders claimed deterioration and 50% of those blamed the surgery, but the actual rate was less, as confirmed by more objective reports [38]. The ED perceived immediately after TURP may be temporary. Tscholl et al selected 98 men with a normal Snap-Gauge test prior to TURP, and re-tested them on their 4th postoperative night. At that stage 34/98 (34.7%) were judged impotent, but on re-testing these 34 men at 3 months, only 8 remained so (8.2%) [39]. In a recent randomized, controlled trial, a significant decrease from baseline in the percentage of men with ED was demonstrated after TURP, and men who underwent TURP were significantly more likely to have an improvement in erectile function than those who had watchful waiting [40]. Neurapraxia from thermal injury or the emotional stress of surgery have been proposed as possible mechanisms [41].

Retrograde ejaculation is the most common complication of TURP, occurring in more than 50% of patients [24]. In some series, retrograde ejaculation has been reported in almost all men and needs to be discussed with all patients. Most older men are typically not both-

ered by it. Open prostatectomy also has little effect on erectile function, and, like TURP, is associated with a high risk of retrograde ejaculation.

SEXUAL DYSFUNCTION FOLLOWING OTHER SURGICAL TREATMENTS

The data on ED after minimally invasive procedures are scant and not always reported. With transurethral microwave thermotherapy (TUMT) for symptomatic BPH, ED occurred in less than 5% of participants [42,43]. Retrograde ejaculation is less common with minimally invasive procedures than with TURP. Typically the incidence of retrograde ejaculation is less than 40% after transurethral incision of the prostate and usually less than 20% with other therapies. In one study, ejaculatory disorder was reported in 11% of patients with TUMT [44].

High-power potassium-titanyl-phosphate (KTP) photoselective laser vaporization of the prostate has become a popular procedure for symptomatic BPH recently. Paick et al reported a significant improvement in the erectile function domain, increasing from 11.3 at baseline to 14.7 ($P = 0.015$), 6 months after KTP laser surgery for BPH in 45 men [45].

CONCLUSIONS

Medical and surgical therapy for LUTS associated with BPH can have a significant impact on quality of life through adverse effects on erectile and ejaculatory function. Research during the past decade has firmly established that ED and ejaculatory dysfunction are highly prevalent conditions in aging men, especially in those with LUTS associated with BPH. Sexuality is an important issue for men and should be included in discussions concerning treatments for LUTS/BPH. Physician awareness of these complications and a willingness to discuss them with the patient can minimize their impact.

REFERENCES

1. Berry SJ, Coffey DS, Walsh PC, Ewing LL: The development of human benign prostatic hyperplasia with age. *J Urol* 1984; **132**: 474-479.
2. Eckhardt MD, van Venrooij GE, Boon TA: Symptoms, prostate volume, and urodynamic findings in elderly male volunteers without and with LUTS and in patients with LUTS suggestive of benign prostatic hyperplasia. *Urology* 2001; **58**:966-971.
3. Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB: Impotence and its medical and psychosocial correlates: Results of the Massachusetts Male Aging Study. *J Urol* 1994; **151**:54-61.
4. Laumann ED, Nicolosi A, Glasser DB: Sexual problems among women and men aged 40-80 y: Prevalence and correlates identified in the Global Study of Sexual Attitudes and Behaviors. *Int J Impot Res* 2005; **17**:39-57.
5. Rosen R, Altwein J, Boyle P, et al: Lower urinary tract symptoms and male sexual dysfunction: The Multinational Survey of the Aging Male (MSAM-7). *Eur Urol* 2003; **44**:637-649.
6. Hansen BL: Lower urinary tract symptoms (LUTS) and sexual function in both sexes. *Eur Urol* 2004; **46**:229-234.
7. McConnell JD, Roehrborn CG, Bautista OM, et al: The long-term effect of doxazosin, finasteride, and combination therapy on the clinical progression of benign prostatic hyperplasia. *N Eng J Med* 2003; **349**:2387-2398.

8. Costabile RA, Steers WD: How can we best characterize the relationship between erectile dysfunction and benign prostatic hyperplasia? *J Sex Med* 2006; **3**:676-681.
9. Khan MA, Thompson CS, Dashwood MR, Mumtaz FH, Morgan RJ, Mikhailidis DP: Endothelin-1 and nitric oxide in the pathogenesis of urinary tract disorders secondary to bladder outlet obstruction. *Curr Vasc Pharmacol* 2003; **1**:27-31.
10. Brannigan RE: Ejaculatory disorders and lower urinary tract symptoms. *Curr Urol Rep* 2004; **5**:280-286.
11. Price DT, Schwinn DA, Lomasney JW, Allen LF, Caron MG, Lefkowitz RJ: Identification, quantification, and localization of mRNA for three distinct alpha 1 adrenergic receptor subtypes in human prostate. *J Urol* 1993; **150**:546-551.
12. Walden PD, Gerardi C, Lepor H: Localization and expression of the alpha 1A-1, alpha 1B and alpha 1D-adrenoreceptors in hyperplastic and non-hyperplastic human prostate. *J Urol* 1999; **161**:635-640.
13. Lue TF: Erectile dysfunction. *N Engl J Med* 2000; **342**:1802-1813.
14. Wettschureck N, Offermanns S: Rho/Rho-kinase mediated signaling in physiology and pathophysiology. *J Mol Med* 2002; **80**:629-638.
15. Rees RW, Foxwell NA, Ralph DJ, Kell PD, Moncada S, Celtek S: Y-27632, a Rho-kinase inhibitor, inhibit proliferation and adrenergic contraction of prostatic smooth muscle cells. *J Urol* 2003; **170**:2517-2522.
16. Buyukafsar K, Levent A, Art M: Expression of Rho-kinase and its functional role in the contractile activity of the mouse vas deferens. *Br J Pharmacol* 2003; **140**:743-749.
17. El-Alfy M, Luu-The V, Huang XF, Berger L, Labrie F, Pelletier G: Localization of type 5 17beta-hydroxysteroid dehydrogenase, 3beta-hydroxysteroid dehydrogenase, and androgen receptor in the human prostate by in situ hybridization and immunocytochemistry. *Endocrinology* 1999; **140**:1481-1491.
18. Lepor H, Wilford WO, Barry MJ, et al: The efficacy of terazosin, finasteride, or both in benign prostatic hyperplasia: Veterans Affairs Cooperative Studies Benign Prostatic Hyperplasia Study Group. *N Engl J Med* 1996; **335**:533-539.
19. Marberger MJ: Long-term effects of finasteride in patients with benign prostatic hyperplasia: A double-blind, placebo-controlled, multicenter study. PROWESS Study Group. *Urology* 1998; **51**:677-686.
20. Andriole GL, Guess HA, Epstein JI, et al: Treatment with finasteride preserves usefulness of prostate-specific antigen in the detection of prostate cancer: Results of a randomized, double-blind, placebo-controlled clinical trial. PLESS Study Group: Proscar Long-term Efficacy and Safety Study. *Urology* 1998; **52**:195-202.
21. Nickel JC, Fradet Y, Boake RC, et al: Efficacy and safety of finasteride therapy for benign prostatic hyperplasia: Results of a 2-year randomized controlled trial (the PROSPECT study). PROscar Safety Plus Efficacy Canadian Two year Study. *CMAJ* 1996; **155**:1251-1259.
22. Roehrborn CG, Boyle P, Nickel JC, Hoefner K, Andriole G, ARIA3001 ARIA3002 and ARIA3003 Study Investigators: Efficacy and safety of a dual inhibitor of 5-alpha-reductase types 1 and 2 (dutasteride) in men with benign prostatic hyperplasia. *Urology* 2002; **60**:434-441.
23. Lepor H: Nonoperative management of benign prostatic hyperplasia. *J Urol* 1989; **141**:1283-1289.
24. AUA Practice Guidelines Committee: AUA guideline on management of benign prostatic hyperplasia (2003). Chapter 1: diagnosis and treatment recommendations. *J Urol* 2003; **170**:530-547.
25. Roehrborn CG, Oesterling JE, Auerbach S, et al: The Hytrin Community Assessment Trial study: A one-year study of terazosin versus placebo in the treatment of men with symptomatic benign prostatic hyperplasia. HYCAT Investigator Group. *Urology* 1996; **47**:159-168.
26. Roehrborn CG, Van kerrebroeck P, Nordling J: Safety and efficacy of alfuzosin 10 mg once-daily in the treatment of lower urinary tract symptoms and clinical benign prostatic hyperplasia: A pooled analysis of three double-blind, placebo-controlled studies. *BJU Int* 2003; **92**:257-261.
27. Proscar (finasteride) [prescribing information]. Whitehouse Station, NJ: Merck & Co., Int.; 2004.
28. Kirby RS, Andersen M, Gratzke P, Dahlstrand C, Hoye K: A combined analysis of double-blind trials of the efficacy and tolerability of doxazosin-gastrointestinal therapeutic system, doxazosin standard and placebo in patients with benign prostatic hyperplasia. *BJU* 2001; **87**:192-200.
29. Schulman CC, Cortvriend J, Jonas U, Lock TM, Vaage S, Speakman MJ: Tamsulosin: 3-year long-term efficacy and safety in patients with lower urinary tract symptoms suggestive of benign prostatic obstruction: Analysis of a European, multinational, multicenter, open-label study. European Tamsulosin Study Group. *Eur Urol* 1999; **36**:609-620.
30. Prescribing information: Flomax® (tamsulosin hydrochloride) capsules. Physicians' Desk Reference, 55th edn. Medical Economics Company: Montvale, NJ, 2002, pp 974-977.
31. Hofner K, Claes H, De Reijke TM, Folkestad B, Speakman MJ: Tamsulosin Study Group. Tamsulosin 0.4 mg once daily: Effect on sexual function in patients with lower urinary tract symptoms suggestive of benign prostatic obstruction. *Eur Urol* 1999; **36**:335-341.
32. Lukacs B, Leplege A, Thibault P, Jardin A: Prospective study of men with clinical benign prostatic hyperplasia treated with alfuzosin by general practitioners: 1-year results. *Urology* 1996; **48**:731-740.
33. De Rose AF, Carmignani G, Corbu C, et al: Observational multicentric trial performed with doxazosin: Evaluation of sexual effects on patients with diagnosed benign prostatic hyperplasia. *Urol Int* 2002; **68**:95-98.
34. Francisc EA, d'Ancona FC, Meuleman EJ, Debruyne FM, de la Rosette JJ: Sexual function following high energy microwave thermotherapy: Results of a randomized controlled study comparing transurethral microwave thermotherapy to transurethral prostatic resection. *J Urol* 1999; **161**:486-490.
35. Carbone DJ Jr, Hodges S: Medical therapy for benign prostatic hyperplasia: Sexual dysfunction and impact on quality of life. *Int J Impot Res* 2003; **15**:299-306.
36. Mebust WK, Holtgrewe HL, Cockett ATK, Peters PC: Transurethral prostatectomy: Immediate and postoperative complications. A cooperative study of 143 participating institutions evaluation 3,885 patients. *J Urol* 1989; **141**:243-247.
37. Wasson JH, Reda DJ, Bruskewitz RC, Elinson J, Keller AM, Henderson WG: A comparison of tansurethral surgery with watchful waiting for moderate symptoms of benign prostatic hyperplasia. The Veterans Affairs Cooperative Study Group on Transurethral Resection of the Prostate. *N Engl J Med* 1995; **332**:75-79.
38. Kinn AC, Helmy-Dhejne C, Larsson J: Sexual function one year after transurethral prostate resection. Patient's own assessments. *Scand J Urol Nephrol* 1998; **32**:33-35.
39. Tscholl R, Largo M, Poppinghaus E, Recker F, Subotic B: Incidence of erectile impotence secondary to transurethral resection of benign prostatic hyperplasia, assessed by preoperative and postoperative Snap Gauge tests. *J Urol* 1995; **153**:1492-1493.
40. Brookes ST, Donovan JL, Peters TJ, Abrams P, Neal DE: Sexual dysfunction in men after treatment for lower urinary tract symptoms: Evidence from randomised controlled trial. *BMJ* 2002; **324**:1059-1061.
41. Parsons CI: Impotence following transurethral resection of the prostate. In: common Problems in Infertility and Impotence, Rajfer J, ed., Year Book medical, Chicago pp 352-355.
42. Tsai YS, Lin JS, Tong JC, et al: Transurethral microwave thermotherapy for symptomatic benign prostatic hyperplasia: Long-term

- durability with Prostate. Eur Urol 2001; **39**:688-694.
43. Roehrborn CG, Issa MM, Bruskewitz RC et al: Transurethral needle ablation for benign prostatic hyperplasia: 12-month results of a prospective, multicenter US study. Urology 1998; **51**:415-421.
44. Rodrigues Netto N Jr, Claro Jde A, Cortado PL: Ejaculatory dysfunction after transurethral microwave thermotherapy for treatment of benign prostatic hyperplasia. J Endourol 1994; **8**:217-219.
45. Paick JS, Um JM, Kim SW, Ku JH: Influence of high-power potassium-titanyl-phosphate photoselective vaporization of the prostate on erectile function: A short-term follow-up study. J Sex Med 2007; **4**:1701-1707.

Apply Now!
www.tcs.org.tw

加入台灣尿失禁防治協會，您便可以立刻擁有本雜誌及下列各項書籍

Join TCS now, you can have this journal and the following books!



台灣尿失禁防治協會：台北縣三峽鎮復興路 399 號 E-mail: msuuf@ms15.hinet.net Tel: 02-86719336 Fax: 02-86716801