Published in final edited form as:
Int J Impot Res. 2009 ; 21(5): 267–274. doi:10.1038/ijir.2009.26.

Measurement of benign prostatic hyperplasia treatment effects on male sexual function

TA Skolarus[1] and **JT Wei**[1]

[1] Dow Division of Health Services Research, Department of Urology, University of Michigan, Ann Arbor, MI, USA

Abstract

Benign prostatic hyperplasia (BPH) is the leading cause of lower urinary tract symptoms among the aging male population. Epidemiological, pathophysiological and clinical studies indicate that many of these men also suffer from declining sexual function, especially those undergoing treatment for their BPH-related urinary symptoms. Although urinary symptoms and quality of life may improve with BPH therapy, the resulting effects on sexual function vary by medical, surgical and minimally invasive approaches and have not been consistently reported. As comprehensive, validated instruments to measure male sexual function are now available for routine use in the clinical setting, urologists and primary care providers caring for patients with BPH have the opportunity to monitor both urinary and sexual function before, during and after BPH therapy. Herein, we describe the relationship between BPH and its treatments on male sexual function, the role of new measures for sexual functioning and opportunities for future work to improve the care of men suffering from both maladies.

Keywords

BPH; erectile dysfunction; medical therapy; surgery

Introduction

Lower urinary symptoms, secondary to benign prostatic hyperplasia (BPH), and declines in male sexual function are common manifestations of aging, and have been associated with each other in a number of clinical and epidemiological studies.[1–9] In this regard, biological mechanisms have been forwarded[10] and efforts to discern a relationship independent of common risk factors such as age and comorbidity have been made.[8,11,12]

Causality notwithstanding, deterioration in both urinary and sexual function are significant issues contributing to the quality of life in the aging male population.[11,13–15] Moreover, the evolution of instruments to measure lower urinary tract symptoms (LUTS) and sexual function alongside the implementation of medical therapy for BPH has allowed us to better understand how male sexual functioning is altered by therapies for BPH. For example, comprehensive measures of ejaculatory function, quality of life and overall satisfaction have been developed and are useful for comparing various therapies.

Correspondence: Dr JT Wei, Department of Urology, University of Michigan, 2301 Commonwealth Blvd, Rm 1021, Ann Arbor, MI 48105-2967, USA. jtwei@umich.edu.

Conflict of interest

Dr Wei has funded research by and is a paid consultant for Sanofi-Aventis Pharmaceuticals. He is a consultant and proctor for American Medical Systems. Dr Skolarus declares no potential conflict of interest.

In light of the multiple treatments for BPH, that is, medical, surgical and minimally invasive therapies, a better understanding of their individual effects on male sexual function will allow both urologists and the increasing number of primary care providers treating BPH to select the optimal treatment for their patients.

BPH symptoms and therapy

Benign prostatic hyperplasia is the leading cause of LUTS among the aging male population.[9,16] LUTS range from nocturia, urinary frequency and urgency to a decreased and intermittent stream with incomplete bladder emptying, and commonly result in a decreased quality of life.[13,14,16–18] This decreased quality of life and increased bother are often the primary reasons men seek treatment.[18]

For men with mild to moderate urinary symptoms without bother, watchful waiting and behavior modification are recommended as the side effects of medical therapy outweigh potential benefits in quality of life.[16,19] As urinary symptoms worsen and become more bothersome, medical therapy with α-adrenergic blockade alone, or in combination with 5-α-reductase inhibitors (for men with larger prostates), are the options.[16] For moderate to severe symptoms that are bothersome, minimally invasive therapy (that is, microwave therapy, transurethral needle ablation) and surgery (transurethral resection, laser ablation or open prostatectomy) are also valid options.[16] However, the latter more aggressive treatment approaches harbor greater potential for impaired sexual functioning and discussion of the potential risks and benefits with the patient are paramount.

The current international standard for measuring the severity and frequency of urinary symptoms secondary to BPH is the International Prostate Symptom Score (IPSS)—a combination of the seven-question American Urological Association BPH Symptom Index[20] and a disease-specific quality of life question.[21,22] This validated instrument consists of seven questions scored from 0 to 5 (0–35) in an increasing order of symptom severity (for example, urinary frequency, nocturia, bladder emptying) and an assessment of bother, that is, living the rest of your life with your current symptoms. The IPSS addresses BPH-related urinary symptoms and quality of life due to these symptoms, however it does not speak of any of the commonly coexisting male sexual function deficits related to BPH and its treatments.

Epidemiological, pathophysiological and clinical support of BPH and male sexual dysfunction relationship

Although studies conflict regarding a causal relationship between BPH-related urinary symptoms and declines in male sexual function, associations independent of age and comorbidity have been reported.[8,11,12] In fact, a dose-response relationship between increasing LUTS and worsening sexual function has been found. After accounting for age, a relationship of worsening sexual function with increasing LUTS was revealed in a cross-sectional evaluation of the Olmsted County Study where ejaculatory function and overall problems with sexual function were affected the most (partial correlation coefficients −0.22 and −0.23, respectively).[12] Likewise, in the Cologne Male Survey, worsening erectile function was not only associated with age, but also with worsening LUTS independent of the age-related effects (odds ratio (OR) 2.11, 95% confidence interval CI (1.75–2.55)).[8] In a broader population, the Multinational Survey of the Aging Male (MSAM-7) clearly demonstrated that although both LUTS and sexual dysfunction share the similar risk factors of age and comorbidity, the severity of sexual dysfunction increased with worsening urinary symptoms in an independent manner.[11] For example, the odds of erectile and ejaculatory dysfunction on multivariate analysis increased across all categories of worsening urinary function as measured using the IPSS (mild symptoms, OR 1.98, 95% CI (1.67–2.34), OR 1.64, 95% CI (1.40–1.93), moderate symptoms OR 3.76, 95% CI (3.14–4.50), OR 3.19, 95% CI (2.68–3.78) and severe symptoms OR 7.67,

95% CI (5.87–10.02), OR 6.25, 95% CI (4.78–8.17), respectively). Consistent with prior studies,[1–7] common risk factors for both BPH and sexual dysfunction, that is, increasing age, diabetes, hypertension and so on, were identified in these analyses.

Regarding the declines in sexual function with worsening urinary tract symptoms, these and other studies approach the functional assessment inconsistently. Although the MSAM-7[11] used the gold standard at the time, the International Index of Erectile Function,[23] the Cologne Male Survey[8] used the validated Kölner Erfassungsbogen der Erektilen Dysfunktion[7] instrument, which focuses more specifically on erectile function than on measures of satisfaction and ejaculatory function as in the International Index of Erectile Function (IIEF). In the earlier Olmsted County Study, the five-question sexual function instrument assessed concern about sexual function, ability to achieve erection, frequency of sexual drive, level of satisfaction, and whether sexual function was changing over time.[24] Taken together, these studies support the contention that LUTS and sexual function are related somehow but the lack of consistent, comprehensive measures of male sexual function has hampered its assessment.

How is BPH treatment associated with male sexual function?

Attention to the side effects of medical therapy for BPH symptoms on men's sexual health was heightened after a series of randomized trials showed improvements in urinary function with α-adrenergic receptor blockade (terazosin, doxazosin, alfuzosin and tamsulosin)[25–29] and 5-α-reductase inhibitor therapy[30–32] in the 1990s. Although these treatments delayed or avoided surgery through improvements in urinary symptoms because of prostatic urethral and bladder neck relaxation (α-blockade) and prostate shrinkage from decreased local dihydrotesterone production (5-α-reductase inhibitors), decreasing erectile and ejaculatory function as well as libido were recognized as common side effects. For example, decreasing libido, erectile and ejaculatory function were noted with finasteride and combination therapy (finasteride and doxazosin) in the Medical Therapy of Prostate Symptoms Study.[33] This was an important finding that confirmed explanations regarding the effects of decreased local dihydrotesterone production in the prostate as well as the more global effects on libido.[34,35] Finasteride and its relative, dutasteride, have both been associated with decreased libido and erectile dysfunction from decreased dihydrotesterone, as well as ejaculatory dysfunction from decreased seminal fluid production.[34–37]

α-adrenergic blockade therapies rarely decrease libido and may even improve overall sexual function while treating urinary symptoms,[38–40] however their effects on ejaculation vary and have been inconsistently measured. Non-selective α-blockade (that is, targeting α_1-adrenergic receptor subtypes in the prostate and bladder neck, as well as vascular smooth muscle[41]) using terazosin has shown marginal worsening of ejaculatory function,[42] although doxazosin may also mildly affect ejaculatory function.[33] To limit the vascular smooth muscle effects of non-selective α-blockade (for example, orthostatic hypotension), selective α-blockade using drugs with increased α_{1A}- to α_{1B}-adrenergic receptor selectivity to preferentially affect the prostate and urethra (for example, tamsulosin),[41] or those with clinical selectivity for the genitourinary tract (for example, alfuzosin),[41] are now commonly used, and likewise may affect ejaculatory function. For example, a dose-dependent effect of tamsulosin exists such that at the most commonly used dose of 0.4 mg, 6% of men may experience ejaculatory dysfunction, whereas at the maximum dose of 0.8 mg, 18% of men may be affected.[43] To compare the effects of tamsulosin (0.4 mg) to alfuzosin (2.5 mg three times daily), one study demonstrated no differences in abnormal ejaculation among the drugs as did another evaluating 0.2 mg tamsulosin versus 10 mg alfuzosin.[44,45] However, in healthy young volunteers, alfuzosin has been shown to maintain ejaculatory function better than maximum-dose tamsulosin, consistent with animal studies.[46,47] Most recently, a pooled analysis of silodosin, another selective α-blocker,[48] showed that 28% of patients experienced ejaculatory dysfunction compared with

~1% for placebo.[49] Despite this and similar to other studies, discontinuation of therapy because of these effects was very low (2.8%). Silodosin's effects seem primarily mediated by anejaculation and failure of emission.[50,51] Hence, these and other studies reveal that α-blockade mechanically affects the expulsion of the ejaculate, unlike the decreased production encountered in 5-α-reductase inhibitor therapy.[52]

The above findings indicate the varied capacities of medical therapy to affect male sexual function during medical treatment for BPH-related LUTS. Recently, the prospective BPH Registry confirmed the varying side effects of BPH medical therapies in a broad-based clinical cohort.[53] As shown in Table 1, the physiologic mechanisms and sexual function deficits associated with each BPH medical therapy vary and need to be considered when selecting a regimen. However, in order to best monitor for potential side effects, clinicians need to use consistent, validated instruments before and during therapy, adjusting the regimen accordingly.

As surgical treatments for BPH evolved toward minimally invasive approaches and the instruments to measure sexual function improved, efforts to understand their effects on male sexual function were furthered.[54] Even early studies measuring treatment outcomes after transurethral resection of the prostate reported on impotence and the obvious ejaculatory deficits after surgery.[55] As electrocautery disrupts the integrity of the bladder neck and potentially scars the ejaculatory ducts, declines in ejaculatory function are extremely common with this approach (>50%),[56] whereas subsequent erectile dysfunction is now rare (<5%).[57] With increasing knowledge of other potential sexual function deficits after surgical treatment, decreased libido and worsened satisfaction with sexuality 1 year after transurethral resection have also been demonstrated.[58]

In this literature, minimally invasive therapies seem to be less frequently associated with erectile or ejaculatory dysfunction.[56,59] The effects of surgical and minimally invasive therapies vary for different aspects of male sexual function (see Table 1). Given that the patient population for surgical therapy is likely to differ from medical therapy (that is, more severe urinary symptoms), minimizing surgical treatment effects on sexual function is necessary while attempting to improve poor urinary function. In addition, better understanding of the manner by which the success of surgical therapy and the irreversible ejaculatory deficits affect the patient's perceptions of their sexual function are areas where prospective research is needed. Specifically, this literature fails to use consistent measures of sexual function before and after surgical therapy.

Evolution of male sexual function symptom and treatment measurement in common clinical settings

Evolving measurement aims have broadened the scope of instruments assessing male sexual function in the studies cited earlier. Indeed, it is now recognized that erectile function is only part of picture, with focus increasing on ejaculatory dysfunction, libido, satisfaction, quality of life and bother.[11,24] Before comprehensive measurement instruments such as the Male Sexual Health Questionnaire (MSHQ),[60] the manifestations of male sexual dysfunction were addressed more in terms of binary outcomes for erectile and ejaculatory function. For example, early studies after transurethral resection of the prostate simply evaluated the presence of impotence (for example, 3–10% following surgery) and the ability to ejaculate (for example, 1/3 of patients) after prostatectomy;[61] however, we now know much more about the degree to which erectile and ejaculatory function contribute more broadly to overall male sexual function and are affected after BPH treatment.

As erectile dysfunction therapy advanced, the IIEF, a 15-question instrument addressing domains of erectile function, orgasmic function, sexual desire, intercourse satisfaction and overall satisfaction, was developed and validated by Ray Rosen.[23] To improve its application

in the clinical setting, a shorter version of the IIEF, the IIEF-5, was examined and found to be an excellent diagnostic tool to determine the presence and severity of erectile dysfunction.[62] The IIEF-5 focuses on erectile function and satisfaction with intercourse, however does little to quantify other aspects of male sexual function, such as libido, overall satisfaction and relationship issues, that are addressed in the longer form.

Realizing ejaculatory dysfunction and declines in satisfaction were increasingly common, especially in men with BPH-related urinary symptoms, Dr Rosen further sought to develop a more comprehensive state of the art sexual measure. To this end, a multidimensional measure of sexual function and satisfaction, the MSHQ was developed and validated for older men with both sexual dysfunction and LUTS.[60] The two domains of sexual function contain three questions to assess erectile function and seven questions to more broadly gauge ejaculatory function than had been previously done. This measure seeks to better understand ejaculatory function in terms of timing, anejaculation, volume, force, and function relative to 5 years earlier. Importantly, it now appears that ejaculatory function is perturbed not only by surgical but also by medical therapies for BPH. A sexual satisfaction domain rounds out the instrument with six questions assessing communication, affection, quality, frequency, sexual and overall satisfaction with the individual's partner. The complete, validated instrument consists of 25 items and better qualifies the ejaculatory and satisfaction aspects of male sexual dysfunction relative to the IIEF.

Recently, a brief four-question assessment of ejaculatory function and bother, the Male Sexual Health Questionnaire-Ejaculatory Dysfunction Short Form (MSHQ-EjD) was created. Capturing the most bothersome characteristics of ejaculatory dysfunction for older men with urinary symptoms, three functional (frequency, force and volume) and one bother question were included in the instrument.[63] Assessing this instrument relative to the unabridged, seven-item ejaculatory function domain of the MSHQ in multiple clinical populations, including men in the BPH Registry, the MSHQ-EjD correlated well with the parent instrument and was deemed an excellent tool for measuring ejaculatory dysfunction.[63] Consequently, this more-brief measure increases its utility in the clinical setting akin to the IIEF-5. In terms of BPH and its treatments, clinician's now have these two straightforward, validated instruments to readily assess both erectile and ejaculatory function, in addition to the IPSS, for men seeking care.

Future investigation

Now that easily administered, validated instruments are available to measure the prevalence and effects of ejaculatory dysfunction on male sexual function, more research is needed for the prevention of ejaculatory dysfunction, as well as the declines in libido, satisfaction and erectile function when treating men with BPH-related urinary symptoms. To take advantage of potentially common pathophysiological mechanisms,[64] PDE-5 inhibitors have been investigated to improve erectile function as well as urinary symptoms due to BPH.[65,66] Similarly, are there medical therapies to actually improve ejaculatory function[67] and likewise BPH-related LUTS? Investigating the mechanisms by which BPH therapies may actually cause sexual dysfunction could generate opportunities for these prevention strategies to be developed.

In terms of surgical treatments, anecdotal reports suggest that there are subtle techniques for preserving erectile and ejaculatory function but these have not been systematically studied. As transurethral resection of the prostate has evolved, rates of associated erectile dysfunction have decreased, whereas ejaculatory dysfunction remains common.[52,68–70] Addressing this through a better understanding of surgical anatomy and physiology is likely to provide insights into how to better preserve function.

Disseminating the body of knowledge related to the effects of BPH and its treatments on male sexual function to the primary care community will also improve patient care. As primary care

providers increasingly care for patients with BPH,[71] better addressing the knowledge discrepancies regarding the underlying sexual side effects of treatment will improve care outside the urology office.[72,73] Moreover, in light of the infrequent discussions of sexual health in older Americans, understanding the hidden BPH treatment effects is even more important for providers as those less likely to discuss their status typically have worse sexual function.[74]

Lastly, investigating a composite measure of male urinary and sexual function, including disease-specific quality of life, that is easy to administer, validated, and shorter than the IPSS, IIEF-5 and MSHQ-EjD Short Form will provide a simple means by which one can assess BPH treatment-related sexual effects. Although some may argue disease-specific measures are best, we now know that lower urinary tract symptoms and declines in male sexual function are interrelated, underdiagnosed, and may perhaps even share a common pathophysiology. Offering patients a straightforward, comprehensive measure may improve BPH and sexual function care under both the urologist and primary care physician.

Acknowledgments

This study was supported by an NIH T32 training grant (NIH 2 T32 DK007782-06).

References

1. 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. [PubMed: 8254833]
2. Blanker MH, Bohnen AM, Groeneveld FP, Bernsen RM, Prins A, Thomas S, et al. Correlates for erectile and ejaculatory dysfunction in older Dutch men: a community-based study. J Am Geriatr Soc 2001;49:436–442. [PubMed: 11347788]
3. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. JAMA 1999;281:537–544. [PubMed: 10022110]
4. Melman A, Gingell JC. The epidemiology and pathophysiology of erectile dysfunction. J Urol 1999;161:5–11. [PubMed: 10037356]
5. Boyle P, Robertson C, Mazzetta C, Keech M, Hobbs FD, Fourcade R, et al. The prevalence of male urinary incontinence in four centres: the UREPIK study. BJU Int 2003;92:943–947. [PubMed: 14632852]
6. Namasivayam S, Minhas S, Brooke J, Joyce AD, Prescott S, Eardley I. The evaluation of sexual function in men presenting with symptomatic benign prostatic hyperplasia. Br J Urol 1998;82:842–846. [PubMed: 9883222]
7. Braun M, Wassmer G, Klotz T, Reifenrath B, Mathers M, Engelmann U. Epidemiology of erectile dysfunction: results of the 'Cologne Male Survey'. Int J Impot Res 2000;12:305–311. [PubMed: 11416833]
8. Braun MH, Sommer F, Haupt G, Mathers MJ, Reifenrath B, Engelmann UH. Lower urinary tract symptoms and erectile dysfunction: co-morbidity or typical 'Aging Male' symptoms? Results of the 'Cologne Male Survey'. Eur Urol 2003;44:588–594. [PubMed: 14572759]
9. Li MK, Garcia L, Patron N, Moh LC, Sundram M, Leungwattanakij S, et al. An Asian multinational prospective observational registry of patients with benign prostatic hyperplasia, with a focus on comorbidities, lower urinary tract symptoms and sexual function. BJU Int 2008;101:197–202. [PubMed: 18005205]
10. McVary KT. Erectile dysfunction and lower urinary tract symptoms secondary to BPH. Eur Urol 2005;47:838–845. [PubMed: 15925081]
11. Rosen R, Altwein J, Boyle P, Kirby RS, Lukacs B, Meuleman E, 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. [PubMed: 14644114]

12. Chung WS, Nehra A, Jacobson DJ, Roberts RO, Rhodes T, Girman CJ, et al. Lower urinary tract symptoms and sexual dysfunction in community-dwelling men. Mayo Clin Proc 2004;79:745–749. [PubMed: 15182088]
13. Girman CJ, Jacobsen SJ, Rhodes T, Guess HA, Roberts RO, Lieber MM. Association of health-related quality of life and benign prostatic enlargement. Eur Urol 1999;35:277–284. [PubMed: 10087388]
14. Welch G, Weinger K, Barry MJ. Quality-of-life impact of lower urinary tract symptom severity: results from the Health Professionals Follow-up Study. Urology 2002;59:245–250. [PubMed: 11834396]
15. 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. [PubMed: 12934061]
16. AUA guideline on management of benign prostatic hyperplasia (2003). Chapter 1: Diagnosis and treatment recommendations. J Urol 2003;170 (2 Pt 1):530–547. [PubMed: 12853821]
17. Boyle P, Robertson C, Mazzetta C, Keech M, Hobbs R, Fourcade R, et al. The relationship between lower urinary tract symptoms and health status: the UREPIK study. BJU Int 2003;92:575–580. [PubMed: 14511037]
18. O'Leary MP, Wei JT, Roehrborn CG, Miner M. Correlation of the International Prostate Symptom Score bother question with the Benign Prostatic Hyperplasia Impact Index in a clinical practice setting. BJU Int 2008;101:1531–1535. [PubMed: 18445080]
19. Barry MJ. Evaluation of symptoms and quality of life in men with benign prostatic hyperplasia. Urology 2001;58(6 Suppl 1):25–32. discussion 32. [PubMed: 11750246]
20. Barry MJ, Fowler FJ Jr, O'Leary MP, Bruskewitz RC, Holtgrewe HL, Mebust WK, et al. The American Urological Association symptom index for benign prostatic hyperplasia. The Measurement Committee of the American Urological Association. J Urol 1992;148:1549–1557. discussion 1564. [PubMed: 1279218]
21. Batista-Miranda JE, Diez MD, Bertran PA, Villavicencio H. Quality-of-life assessment in patients with benign prostatic hyperplasia: effects of various interventions. Pharmacoeconomics 2001;19:1079–1090. [PubMed: 11735675]
22. Cockett, A.; Aso, Y.; Denis, L. Prostate symptom score and quality of life assessment. In: Cockett, ATK.; Khoury, S.; Aso, Y.; Chatelain, C.; Denis, L.; Griffiths, K.; Murphy, G., editors. Proceedings of the Second International Consultation on Benign Prostatic Hyperplasia (BPH); 27–30 June 1993; Paris; Channel Island, Jersey: Scientific Communication International; 1994. p. 553-555.
23. Rosen RC, Riley A, Wagner G, Osterloh IH, Kirkpatrick J, Mishra A. The international index of erectile function (IIEF): a multidimensional scale for assessment of erectile dysfunction. Urology 1997;49:822–830. [PubMed: 9187685]
24. Panser LA, Rhodes T, Girman CJ, Guess HA, Chute CG, Oesterling JE, et al. Sexual function of men ages 40–79 years: the Olmsted County Study of Urinary Symptoms and Health Status Among Men. J Am Geriatr Soc 1995;43:1107–1111. [PubMed: 7560700]
25. Jardin A, Bensadoun H, Delauche-Cavallier MC, Attali P. Alfuzosin for treatment of benign prostatic hypertrophy. The BPH-ALF Group. Lancet 1991;337:1457–1461. [PubMed: 1710750]
26. Lepor H, Auerbach S, Puras-Baez A, Narayan P, Soloway M, Lowe F, et al. A randomized, placebo-controlled multicenter study of the efficacy and safety of terazosin in the treatment of benign prostatic hyperplasia. J Urol 1992;148:1467–1474. [PubMed: 1279214]
27. Chapple CR, Wyndaele JJ, Nordling J, Boeminghaus F, Ypma AF, Abrams P. Tamsulosin, the first prostate-selective alpha 1A-adrenoceptor antagonist. A meta-analysis of two randomized, placebo-controlled, multicentre studies in patients with benign prostatic obstruction (symptomatic BPH). European Tamsulosin Study Group. Eur Urol 1996;29:155–167. [PubMed: 8647141]
28. Abrams P, Schulman CC, Vaage S. Tamsulosin, a selective alpha 1c-adrenoceptor antagonist: a randomized, controlled trial in patients with benign prostatic 'obstruction' (symptomatic BPH). The European Tamsulosin Study Group. Br J Urol 1995;76:325–336. [PubMed: 7551841]
29. Roehrborn CG, Siegel RL. Safety and efficacy of doxazosin in benign prostatic hyperplasia: a pooled analysis of three double-blind, placebo-controlled studies. Urology 1996;48:406–415. [PubMed: 8804494]

30. Gormley GJ, Stoner E, Bruskewitz RC, Imperato-McGinley J, Walsh PC, McConnell JD, et al. The effect of finasteride in men with benign prostatic hyperplasia. The Finasteride Study Group. N Engl J Med 1992;327:1185–1191. [PubMed: 1383816]

31. Lepor H, Williford WO, Barry MJ, Brawer MK, Dixon CM, Gormley G, 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. [PubMed: 8684407]

32. Debruyne FM, Jardin A, Colloi D, Resel L, Witjes WP, Delauche-Cavallier MC, et al. Sustained-release alfuzosin, finasteride and the combination of both in the treatment of benign prostatic hyperplasia. European ALFIN Study Group. Eur Urol 1998;34:169–175. [PubMed: 9732187]

33. McConnell JD, Roehrborn CG, Bautista OM, Andriole GL Jr, Dixon CM, Kusek JW, et al. The long-term effect of doxazosin, finasteride, and combination therapy on the clinical progression of benign prostatic hyperplasia. N Engl J Med 2003;349:2387–2398. [PubMed: 14681504]

34. Byrnes CA, Morton AS, Liss CL, Lippert MC, Gillenwater JY. Efficacy, tolerability, and effect on health-related quality of life of finasteride versus placebo in men with symptomatic benign prostatic hyperplasia: a community based study. CUSP Investigators. Community based study of Proscar. Clin Ther 1995;17:956–969. [PubMed: 8595647]

35. Amory JK, Anawalt BD, Matsumoto AM, Page ST, Bremner WJ, Wang C, et al. The effect of 5alpha-reductase inhibition with dutasteride and finasteride on bone mineral density, serum lipoproteins, hemoglobin, prostate specific antigen and sexual function in healthy young men. J Urol 2008;179:2333–2338. [PubMed: 18423697]

36. Rosen RC, Giuliano F, Carson CC. Sexual dysfunction and lower urinary tract symptoms (LUTS) associated with benign prostatic hyperplasia (BPH). Eur Urol 2005;47:824–837. [PubMed: 15925080]

37. Roehrborn CG, Boyle P, Nickel JC, Hoefner K, Andriole G. 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. [PubMed: 12350480]

38. Roehrborn CG, Rosen RC. Medical therapy options for aging men with benign prostatic hyperplasia: focus on alfuzosin 10 mg once daily. Clin Interv Aging 2008;3:511–524. [PubMed: 18982921]

39. Chung BH, Lee JY, Kim CI, Kim CS, Oh CY, Lee SW, et al. Sexuality and the management of BPH with alfuzosin (SAMBA) trial. Int J Impot Res 2009;21:68–73. [PubMed: 19078970]

40. Kaplan SA, AFDER, Kirby RS, O'Leary MP, McVary KT. Beneficial effects of extended-release doxazosin and doxazosin standard on sexual health. BJU Int 2006;97:559–566. [PubMed: 16469026]

41. Lowe FC. Role of the newer alpha-adrenergic-receptor antagonists in the treatment of benign prostatic hyperplasia-related lower urinary tract symptoms. Clin Ther 2004;26:1701–1713. [PubMed: 15639685]

42. Roehrborn CG, Oesterling JE, Auerbach S, Kaplan SA, Lloyd LK, Milam DE, 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. [PubMed: 8607227]

43. Lepor H. Phase III multicenter placebo-controlled study of tamsulosin in benign prostatic hyperplasia. Tamsulosin Investigator Group. Urology 1998;51:892–900. [PubMed: 9609623]

44. Hofner K, Claes H, De Reijke TM, Folkestad B, Speakman MJ. 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. [PubMed: 10473995]

45. Lapitan MC, Acepcion V, Mangubat J. A comparative study on the safety and efficacy of tamsulosin and alfuzosin in the management of symptomatic benign prostatic hyperplasia: a randomized controlled clinical trial. J IntMed Res 2005;33:562–573.

46. Hellstrom WJ, Sikka SC. Effects of acute treatment with tamsulosin versus alfuzosin on ejaculatory function in normal volunteers. J Urol 2006;176(4 Pt 1):1529–1533. [PubMed: 16952675]

47. Giuliano F, Bernabe J, Droupy S, Alexandre L, Allard J. A comparison of the effects of tamsulosin and alfuzosin on neurally evoked increases in bladder neck and seminal vesicle pressure in rats. BJU Int 2004;93:605–608. [PubMed: 15008740]

48. Shibata K, Foglar R, Horie K, Obika K, Sakamoto A, Ogawa S, et al. KMD-3213, a novel, potent, alpha 1a-adrenoceptor-selective antagonist: characterization using recombinant human alpha 1–adrenoceptors and native tissues. Mol Pharmacol 1995;48:250–258. [PubMed: 7651358]
49. Marks LS, Gittelman MC, Hill LA, Volinn W, Hoel G. Rapid efficacy of the highly selective alpha1A-adrenoceptor antagonist silodosin in men with signs and symptoms of benign prostatic hyperplasia: pooled results of 2 phase 3 studies. J Urol 2009;181:2634–2640. [PubMed: 19371887]
50. Yono M, Yamamoto Y, Imanishi A, Fukagawa A, Latifpour J, Yoshida M. Short- and long-term effects of silodosin, a selective alpha-adrenoceptor antagonist, on ejaculatory function in rats. BJU Int 2009;103:1680–1685. [PubMed: 19220259]
51. Kobayashi K, Masumori N, Hisasue S, Kato R, Hashimoto K, Itoh N, et al. Inhibition of Seminal emission is the main cause of anejaculation induced by a new highly selective alpha1A-blocker in normal volunteers. J Sex Med 2008;5:2185–2190. [PubMed: 18399947]
52. AUA Practice Guidelines Committee. AUA Guideline on Management of Benign Prostatic Hyperplasia, Chapter 3: Results of the Treatment Outcomes Analyses. American Urological Association; Linthincum, MD: 2003. p. 56
53. Rosen RC, Wei JT, Althof SE, Seftel AD, Miner M, Perelman MA. Association of sexual dysfunction with lower urinary tract symptoms of BPH and BPH medical therapies: results from the BPH Registry. Urology 2009;73:562–566. [PubMed: 19167031]
54. Arai Y, Aoki Y, Okubo K, Maeda H, Terada N, Matsuta Y, et al. Impact of interventional therapy for benign prostatic hyperplasia on quality of life and sexual function: a prospective study. J Urol 2000;164:1206–1211. [PubMed: 10992367]
55. Finkle AL, Moyers TG. Sexual potency in aging males. IV. Status of private patients before and after prostatectomy. J Urol 1960;84:152–157. [PubMed: 13822915]
56. Schulman C. Impact of treatment of BPH on sexuality. Prostate Cancer Prostatic Dis 2001;4(S1):S12–S16. [PubMed: 12497054]
57. Fowler FJ Jr, Wennberg JE, Timothy RP, Barry MJ, Mulley AG Jr, Hanley D. Symptom status and quality of life following prostatectomy. JAMA 1988;259:3018–3022. [PubMed: 2452905]
58. Kinn AC, Helmy-Dhejne C, Larsson J. Sexual function one year after transurethral prostatic resection. Patients' own assessments. Scand J Urol Nephrol 1998;32:33–35. [PubMed: 9561571]
59. Cimentepe E, Unsal A, Saglam R. Randomized clinical trial comparing transurethral needle ablation with transurethral resection of the prostate for the treatment of benign prostatic hyperplasia: results at 18 months. J Endourol 2003;17:103–107. [PubMed: 12689404]
60. Rosen RC, Catania J, Pollack L, Althof S, O'Leary M, Seftel AD. Male Sexual Health Questionnaire (MSHQ): scale development and psychometric validation. Urology 2004;64:777–782. [PubMed: 15491719]
61. Hargreave TB, Stephenson TP. Potency and prostatectomy. Br J Urol 1977;49:683–688. [PubMed: 597709]
62. Rosen RC, Cappelleri JC, Smith MD, Lipsky J, Pena BM. Development and evaluation of an abridged, 5–item version of the International Index of Erectile Function (IIEF-5) as a diagnostic tool for erectile dysfunction. Int J Impot Res 1999;11:319–326. [PubMed: 10637462]
63. Rosen RC, Catania JA, Althof SE, Pollack LM, O'Leary M, Seftel AD, et al. Development and validation of four-item version of Male Sexual Health Questionnaire to assess ejaculatory dysfunction. Urology 2007;69:805–809. [PubMed: 17482908]
64. McVary K. Lower urinary tract symptoms and sexual dysfunction: epidemiology and pathophysiology. BJU Int 2006;97(Suppl 2):23–28. discussion 44–25. [PubMed: 16507050]
65. Roehrborn CG. Lower urinary tract symptoms, benign prostatic hyperplasia, erectile dysfunction, and phosphodiesterase-5 inhibitors. Rev Urol 2004;6:121–127. [PubMed: 16985592]
66. Kaplan SA, Gonzalez RR. Phosphodiesterase type 5 inhibitors for the treatment of male lower urinary tract symptoms. Rev Urol 2007;9:73–77. [PubMed: 17592540]
67. Master VA, Turek PJ. Ejaculatory physiology and dysfunction. Urol Clin North Am 2001;28:363–375. x. [PubMed: 11402588]
68. Lynch M, Anson K. Time to rebrand transurethral resection of the prostate? Curr Opin Urol 2006;16:20–24. [PubMed: 16385196]

69. Doll HA, Black NA, McPherson K, Williams GB, Smith JC. Differences in outcome of transurethral resection of the prostate for benign prostatic hypertrophy between three diagnostic categories. Br J Urol 1993;72:322–330. [PubMed: 7693294]
70. Wasson JH, Reda DJ, Bruskewitz RC, Elinson J, Keller AM, Henderson WG. A comparison of transurethral 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. [PubMed: 7527493]
71. Tenover JL, Pagano GA, Morton AS, Liss CL, Byrnes CA. Efficacy and tolerability of finasteride in symptomatic benign prostatic hyperplasia: a primary care study. Primary Care Investigator Study Group. Clin Ther 1997;19:243–258. [PubMed: 9152564]
72. Seftel A, Rosen R, Kuritzky L. Physician perceptions of sexual dysfunction related to benign prostatic hyperplasia (BPH) symptoms and sexual side effects related to BPH medications. Int J Impot Res 2007;19:386–392. [PubMed: 17377613]
73. Roehrborn CG, Nuckolls JG, Wei JT, Steers W. The benign prostatic hyperplasia registry and patient survey: study design, methods and patient baseline characteristics. BJU Int 2007;100:813–819. [PubMed: 17822462]
74. Lindau ST, Schumm LP, Laumann EO, Levinson W, O'Muircheartaigh CA, Waite LJ. A study of sexuality and health among older adults in the United States. N Engl J Med 2007;357:762–774. [PubMed: 17715410]
75. De Rose AF, Carmignani G, Corbu C, Giglio M, Traverso P, Naselli A, 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. [PubMed: 11834898]
76. Kirby RS, Roehrborn C, Boyle P, Bartsch G, Jardin A, Cary MM, et al. Efficacy and tolerability of doxazosin and finasteride, alone or in combination, in treatment of symptomatic benign prostatic hyperplasia: the Prospective European Doxazosin and Combination Therapy (PREDICT) trial. Urology 2003;61:119–126. [PubMed: 12559281]
77. Chapple CR. A Comparison of Varying alpha-Blockers and Other Pharmacotherapy Options for Lower Urinary Tract Symptoms. Rev Urol 2005;7(Suppl 4):S22–S30. [PubMed: 16986051]
78. 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. [PubMed: 12887479]
79. Narayan P, Lepor H. Long-term, open-label, phase III multicenter study of tamsulosin in benign prostatic hyperplasia. Urology 2001;57:466–470. [PubMed: 11248621]
80. 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. [PubMed: 10559616]
81. Physicians' Desk Reference. 55. Medical Economics Company; Montvale, NJ: 2002. Prescribing information. Flomax (tamsulosin hydrochloride) capsules; p. 974-977.
82. Nagai A, Hara R, Yokoyama T, Jo Y, Fujii T, Miyaji Y. Ejaculatory dysfunction caused by the new alpha1-blocker silodosin: A preliminary study to analyze human ejaculation using color Doppler ultrasonography. Int J Urol 2008;15:915–918. [PubMed: 18721206]
83. Kawabe K, Yoshida M, Homma Y. Silodosin, a new alpha1A-adrenoceptor-selective antagonist for treating benign prostatic hyperplasia: results of a phase III randomized, placebo-controlled, double-blind study in Japanese men. BJU Int 2006;98:1019–1024. [PubMed: 16945121]
84. Kaminetsky J. Comorbid LUTS and erectile dysfunction: optimizing their management. Curr Med Res Opin 2006;22:2497–2506. [PubMed: 17265598]
85. McConnell JD, Bruskewitz R, Walsh P, Andriole G, Lieber M, Holtgrewe HL, et al. The effect of finasteride on the risk of acute urinary retention and the need for surgical treatment among men with benign prostatic hyperplasia. Finasteride Long-Term Efficacy and Safety Study Group. N Engl J Med 1998;338:557–563. [PubMed: 9475762]

86. 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. [PubMed: 9610579]

87. Nickel JC, Fradet Y, Boake RC, Pommerville PJ, Perreault JP, Afridi SK, 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. [PubMed: 8911291]

88. Marberger M, Roehrborn CG, Marks LS, Wilson T, Rittmaster RS. Relationship among serum testosterone, sexual function, and response to treatment in men receiving dutasteride for benign prostatic hyperplasia. J Clin Endocrinol Metab 2006;91:1323–1328. [PubMed: 16434455]

89. O'Leary MP, Roehrborn C, Andriole G, Nickel C, Boyle P, Hofner K. Improvements in benign prostatic hyperplasia-specific quality of life with dutasteride, the novel dual 5alpha-reductase inhibitor. BJU Int 2003;92:262–266. [PubMed: 12887480]

90. Marihart S, Harik M, Djavan B. Dutasteride: a review of current data on a novel dual inhibitor of 5alpha reductase. Rev Urol 2005;7:203–210. [PubMed: 16985831]

91. Rosario DJ, Woo H, Potts KL, Cutinha PE, Hastie KJ, Chapple CR. Safety and efficacy of transurethral needle ablation of the prostate for symptomatic outlet obstruction. Br J Urol 1997;80:579–586. [PubMed: 9352697]

92. Bruskewitz R, Issa MM, Roehrborn CG, Naslund MJ, Perez-Marrero R, Shumaker BP, et al. A prospective, randomized 1-year clinical trial comparing transurethral needle ablation to transurethral resection of the prostate for the treatment of symptomatic benign prostatic hyperplasia. J Urol 1998;159:1588–1593. discussion 1593–1584. [PubMed: 9554360]

93. Steele GS, Sleep DJ. Transurethral needle ablation of the prostate: a urodynamic based study with 2-year followup. J Urol 1997;158:1834–1838. [PubMed: 9334612]

94. Zlotta AR, Giannakopoulos X, Maehlum O, Ostrem T, Schulman CC. Long-term evaluation of transurethral needle ablation of the prostate (TUNA) for treatment of symptomatic benign prostatic hyperplasia: clinical outcome up to five years from three centers. Eur Urol 2003;44:89–93. [PubMed: 12814680]

95. Miner M, Rosenberg MT, Perelman MA. Treatment of lower urinary tract symptoms in benign prostatic hyperplasia and its impact on sexual function. Clin Ther 2006;28:13–25. [PubMed: 16490576]

96. Guazzoni G, Montorsi F, Coulange C, Milroy E, Pansadoro V, Rubben H, et al. A modified prostatic UroLume Wallstent for healthy patients with symptomatic benign prostatic hyperplasia: a European Multicenter Study. Urology 1994;44:364–370. [PubMed: 7521092]

97. Roehrborn CG. The Agency for Health Care Policy and Research. Clinical guidelines for the diagnosis and treatment of benign prostatic hyperplasia. Urol Clin North Am 1995;22:445–453. [PubMed: 7539190]

98. 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. Br Med J 2002;324:1059–1061. [PubMed: 11991908]

99. Riehmann M, Knes JM, Heisey D, Madsen PO, Bruskewitz RC. Transurethral resection versus incision of the prostate: a randomized, prospective study. Urology 1995;45:768–775. [PubMed: 7538238]

100. Hoffman RM, MacDonald R, Wilt TJ. Laser prostatectomy for benign prostatic obstruction. Cochrane Database Syst Rev 2004:CD001987. [PubMed: 14973978]

Table 1

The effects of medical, surgical and minimally invasive BPH therapy on male sexual function and lower urinary tract symptoms[a]

BPH Treatment	Male sexual function					Urinary function	
	Ejaculatory function		Erectile function				
	Decreased production	Anejaculation/retrograde ejaculation	Quality of erection	Libido/drive	Satisfaction	IPSS	QOL
Medical therapy							
α-adrenergic blockers							
Doxazosin[b,33,36,40,75,76]	↔	↔↓	↑	↔	↔	↑	↑
Terazosin[b,26,31,42]	↔	↔↓	↔	↔	↔	↑	↑
Alfuzosin[b,32,36,39,44,77,78]	↔	↔↓	↑	↔	↑	↑	↑
Tamsulosin[b,27,36,43,44,77,79–81]	↔	↓	↔	↔	↑	↑	↑
Silodosin[49,51,82,83]	↔	↓↓	n/a	n/a	n/a	↑	↑
5-α-reductase inhibitors							
Finasteride[b,30,32,34,36,71,84–87]	↓	↔	↓	↓	↓	↑	↑
Dutasteride[36,37,88–90]	↓	↔	↓	↓	↓	↑	↑
Combination therapy[b,32,33,36,84]	↓	↔↓	↓	↓	↓	↑	↑
Minimally invasive therapy							
Transurethral needle ablation[b,54,59,91–95]	↔	↔↓	↔	↔	↔	↑↑	↑↑↑
Transurethral microwave heat treatments[b,54,95]	↔	↔↓	↔↓	↔	↔	↑↑	↑↑
Urolume stent (American Medical Systems, Minnetonka, Minnesota) [b,96]	↔	↓	↔	↓	↔	↑↑	↔
Surgical therapy							
Transurethral resection of the prostate[b,54,58,69,70,95,97,98]	↔	↓↓↓	↓↓	↓↓	↓↓	↑↑↑	↑↑↑
Transurethral electrovaporization[b]	↔	↓↓↓	↓↓	↔	↓↓	↑↑↑	↑↑↑
Transurethral incision of the prostate[b,97,99]	↔	↓↓	↓↓	↔	↔	↑↑	↑↑↑
Transurethral holmium laser resection/enucleation[b,54,98,100]	↔	↓↓↓	↓	↓	↓↓	↑↑↑	↑↑↑
Transurethral laser vaporization[b]	↔	↓↓↓	↓↓	↓	↓↓	↑↑	↑↑↑

Int J Impot Res. Author manuscript; available in PMC 2010 May 3.

BPH Treatment	Male sexual function					Urinary function	
	Ejaculatory function		Erectile function				
	Decreased production	Anejaculation/retrograde ejaculation	Quality of erection	Libido/drive	Satisfaction	IPSS	QOL
Transurethral laser coagulation[b],[54]	↔	↓↓	↓↓	↓	↓↓	↑↑↑	↑↑↑
Open prostatectomy[b],[97]	↔	↓↓↓	↓	↓↓	↓↓	↑↑	↑

Abbreviations: AUA, American Urological Association; BPH, benign prostatic hyperplasia; IPSS, International Prostate Symptom Score; QOL, quality of life.

[a]The therapeutic effects in this table are based on a review of the cited literature. They do not represent a systematic review nor meta-analysis of the effects of BPH therapy on sexual function.

[b]Estimated effect sizes are based on the AUA Guideline treatment outcomes meta-analysis 2003.[52]

↑↑↑ vast improvement, ↑↑ moderate improvement, ↑ slight improvement, ↔ no/minimal change, ↓ slight worsening, ↓↓ moderate worsening, ↓↓↓ severe worsening.

IPSS/AUA Symptoms Index Score: ↑↑↑ vast improvement (−15), ↑↑ moderate improvement (−10), ↑ slight improvement (−5), ↔ no change (0).

Disease-specific Quality of Life Score: ↑↑↑ vast improvement (≤ −3), ↑↑ moderate improvement (−2), ↑ slight improvement (−1), ↔ no change (0).

Erectile dysfunction: ↔ no change (<5%), ↓ slight worsening (5%), ↓↓ moderate worsening (10%), ↓↓↓ severe worsening (15%).