Clinical Considerations for Intravesical Prostatic Protrusion in the Evaluation and Management of Bladder Outlet Obstruction Secondary to Benign Prostatic Hyperplasia

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Key Words
Benign prostatic hyperplasia • Median lobe hypertrophy • Intravesical prostatic protrusion • Transrectal ultrasonography • Lower urinary tract symptoms

Abstract
Background: Intravesical prostatic protrusion (IPP) is a manifestation of benign prostatic hyperplasia marked by overgrowth of the prostatic median lobe into the bladder, producing bladder outlet obstruction and related storage and voiding symptoms. Methods: A MEDLINE® database search of the current literature was guided using combination of "prostate" with the following terms: intravesical prostatic protrusion, bladder trabeculation, bladder outlet obstruction, lower urinary tract symptoms, alpha blockers, transrectal ultrasonography, and prostatectomy. Results: Although IPP can be identified via a variety of imaging modalities, it is easily detected via transrectal ultrasonography (TRUS). Failing to detect IPP promptly by TRUS may result in refractory symptoms of benign prostatic hyperplasia, as the condition may not respond to typical α1-adrenoceptor antagonist therapy. In addition, depending on grade, IPP can influence outcomes and complications of prostatectomies. Conclusion: Upon report of lower urinary tract symptoms, initial performance of TRUS along with digital rectal examination prevents delay in the appropriate evaluation and management of prostatic diseases.

Introduction
Benign prostatic hyperplasia (BPH) is one of the most widespread diseases amongst men, but there is no consensus or clear practical guideline to define the presence and severity of obstruction, other than pressure-flow studies. Intravesical prostatic protrusion (IPP) is a phenomenon in which the prostate adenoma enlarges into the bladder along the plane of least resistance [1]. Previous studies in men with IPP have demonstrated an increased rate of bladder outlet obstruction (BOO) or progression of clinical BPH [2]. However, IPP is a manifestation of BPH that cannot be accurately diagnosed by traditional digital rectal examination (DRE) and noninvasive studies. Interestingly, though its usage is ubiquitous in clinics, customary DRE examination fails to detect the configuration of IPP.
A sampling of population data has denoted that 10% of males between 40 and 79 years of age had an IPP classified as severe [3] and unfortunately, even in these cases, bimanual examination of the bladder is ambiguous in the detection of IPP. As IPP can affect lower urinary tract symptoms (LUTS) and function in men, understanding the evaluation and management of IPP is important. We review how the pathogenesis of IPP deviates from that of other forms of BPH, leading to the presentation of distinctive clinical signs and sequelae. We also explore how the approach to the diagnosis and management of IPP as traditional BPH may lead to treatment inefficacy. Moreover, we identify how the outcomes of robot-assisted laparoscopic prostatectomy may be influenced by varying severity levels of IPP.

**Methods**

The inclusion criteria for the literature search using the MEDLINE® search engine was established using a combination of “prostate” and the following terms: intravesical prostatic protrusion, bladder trabeculation, bladder outlet obstruction, lower urinary tract symptoms, alpha blockers, transrectal ultrasonography, and prostatectomy. Although, there was no date restriction on the search, we placed an emphasis on the past 5 years. No specific exclusion criteria were set. Publication quality was assessed using the relative citation ratio derived from iCite bibliometrics. We selected papers that revealed artifacts for the pathophysiology, clinical manifestations, clinical evaluation, and management of IPP.

**Pathophysiology and Clinical Manifestations**

The pathophysiology and clinical manifestations of IPP are distinct from routine BPH. In BPH, all lobes of the prostate may routinely be affected and many types of patterns are recognized by transrectal ultrasonography (TRUS). Initially described by Alexander Randall in the early 20th century [1], these patterns include lateral lobe, trilobular, median lobe, subtrigonal lobe, and substernal hypertrophy, as well as a median bar. Frequently, hypertrophy may assume any combination of these configurations. However, the morphological change defined by IPP is distinct from any of these types. The types implicated in IPP are median lobe, trilobular, and median bar because they can occupy bladder space at a measurable capacity. The median lobe, which arises from the periurethral zone [4], is situated between the urethra and ejaculatory ducts. Its upper surface is bound by the bladder trigone and projects into the bladder producing the uvula vesicae, *i.e.*, the elevation behind the internal urethral orifice. While lateral lobe BPH would cause compression of the prostatic urethra, median lobe IPP may trigger a “ball-valve” type of obstruction, disrupting laminar flow at the bladder neck and distorting the funnelling effect of the normal prostatic-urethral angle [4], and leading to dyskinetic movement of the bladder during micturition [5]. This would be responsible for more obstruction than if there were no protrusion and just bilateral hypertrophied lateral lobes, as the strong bladder contraction could force open a channel between the lobes but tend to aggravate the “ball-valve” effect in IPP [6].

Urethral resistance ensues due to IPP and impedes the hydraulic energy that normally drives micturition [7]. A 2015 fluid structural interaction analysis study demonstrated that IPP predisposed the prostate to deformation caused by intravesical pressure. The authors found that the compression of the prostatic urethra and increased variation of cross-sectional area around bladder neck would diminish urine flow efficiency, as well as compromise the effect of obstruction alleviation treatment [7]. Bladder neck and urethral constraint may be due to the nature of the fascia “capsule” surrounding the prostate. The prostate is adjoined anteriorly by pubo-prostatic ligaments, posteriorly by Denonvilliers’ fascia, and laterally by endopelvic fascia [8]. Superior to these connections, there is merging with other fascia that leaves the IPP susceptible to the radial component of intravesical pressure, thus leading to prostatic deformation. This study provided further evidence suggesting that IPP influences BOO independently, and that flow deterioration is more resistant to alleviation of obstruction during treatment as the IPP grade increases [7].

The intravesical pressure fundamental to opening the bladder neck is increased when detrusor pressures increase in response to voiding through resistance (*i.e.*, BOO). While the bladder can yield a higher transitory voiding pressure, the constant pressure needed will force the detrusor muscle to gradually wane. In response to the need for a higher intravesical pressure to overcome BOO, hypertrophy of isolated muscle bundles occurs followed by bladder trabeculation or increased detrusor wall thickness [9]. The area between hypertrophied bundles becomes narrower, resulting in less viable detrusor muscle with eventual progression to total lack of function. Moreover, the resultant BOO leads to the presentation of LUTS. A 2015 clinical study found that the International Prostate Symptom Score (IPSS) voiding subscore had a strong correlation with terminal dribbling and an underrated LUTS, while IPP was the only significant risk factor for uroflowmetry-confirmed terminal dribbling [10].
The prostate’s conformation, shape, and IPP may interfere with the voiding process. Clinical studies have demonstrated that IPP can identify BOO. IPP has shown a positive predictive value of 72% for BOO [11]. In fact, the positive predictive value of the combined parameters IPP and resistive index of the prostatic capsular artery increases to 83.8% [12]. Because this resistive index is a signal of vascular resistance [13], it is a good indicator for BPH-related BOO (12). IPP ≥ 10 mm has been reported to have a good sensitivity in defining BOO and even better at defining the response to the α-adrenoceptor antagonists in patients with a trial without catheter after acute urinary retention [14, 15]. Additionally, a study on 200 patients with BOO showed that a higher degree of IPP was associated with a higher postvoid residual urine volume (PVR). IPP also affects the value of the IPSS and uroflowmetry volumes. Several other studies have shown the association between the IPP and BOO index, in which moderate to severe degree IPP has a significant influence on BOO and impact on the efficacy of α-adrenoceptor antagonists in the treatment of LUTS in BPH patients. A 2015 study discovered that the degree of IPP has a strong correlation with the total and voiding IPSS, but no correlation with maximum flow rate ($Q_{\text{max}}$), $Q_{\text{ave}}$, voided volume, and PVR [16].

The degree of BOO increases with the severity of IPP. This is encountered with a significant elongation of the prostatic urethra and trigonal stimulation, thus contributing to storage symptoms (e.g., acute urinary retention) caused by stimulation of the bladder [17–19]. Storage symptoms in patients with IPP have three potential explanations. First, there can be a coexistent state of primary storage of bladder dysfunction. Second, IPP itself also represents an aggravated BPH which may be combined with secondary storage bladder dysfunction due to BOO. Third, there may be a fixed effect of the bladder neck; an enlarged bladder neck with a collagen tissue component could compromise the effect of the internal bladder neck sphincter. Normally, during the storage phase of the bladder, the bladder neck demarcates entry of urine into the prostatic urethra. However, in the presence of IPP, the bladder neck may not be tightly closed during the storage phase, allowing small amounts of urine to pass into the prostatic urethra, thus prematurely activating the micturition reflexes typically observed with urinary incontinence [20] and clinically presenting as irritative and obstructive symptoms of bilobar BPH. Extroversion of the prostatic urethral mucous membrane at the bladder neck and the associated urethral distension may trigger a urethrovessical stimulating reflex to enable bladder contractions [21, 22] responsible for storage symptoms and overactive bladder. Dilatation of the non-sphincteric urethra during bladder contractions (i.e., vesicourethral inhibitory reflex) [23] may be compromised in prostatic obstruction. Additionally, as explained previously, the added connective tissue between detrusor muscle fascicles due to prolonged BPH may lead to the myogenic changes responsible for overactive bladder with or without urge incontinence [24]. Due to the bearing of IPP on bladder neck funneling, incomplete voiding and the subsequent urine stasis have been linked in the pathogenesis of bladder stone formation [25]. Kim et al. [26] hypothesized that severe IPP and low $Q_{\text{max}}$ value may disturb the excretion of the stone nidus and result in the development of bladder stone. Moreover, because IPP can affect the integrity of the internal urethral sphincter via mechanical distension, there may be concerns for retrograde ejaculation because due to the failure of the internal urethral sphincter to close during ejaculation. However, there is presently a dearth of physiological and clinical studies on these potential secondary complications.

Prostate-specific antigen (PSA) is speculated to be elevated in IPP patients because they usually already have, save for a few clinical exceptions [27], a large prostatic volume [28]. Since a more forceful vesical contraction is required to open a channel between the lobes in those with significant IPP, it may contribute to more PSA leakage from the prostate into the serum. Additionally, it has been long known that PSA leakage from the prostate into the serum originates at the transition zone, where hypertrophy indicates BPH [29]. Unearthed in a modeling study by Xu et al. [30], the predictive accuracy of IPP removed prostate cancer predicting score (IRPPS), which consists of total PSA, transition zone volume, and IPP, was determined to be higher than that of PSA density, %PSA, and total PSA, suggesting that IRPPS could predict prostate cancer when the patient has a PSA of 4.0–10.0 ng/ml and a measurable IPP grade. The authors concluded that total PSA had positive correlation with IPP. For IPP patients with a PSA of 4.0–10.0 ng/ml, IRPPS could help validate the presence of prostate cancer at prostate biopsy.

Clinical Evaluation and Management

Modern day clinics and hospitals have been relying on DRE and bimanual examination of the bladder to evaluate BOO. IPP may not be readily detected by these methods, as evidenced by the diagnostic roadblocks depicted in Table 1. Because of this, misdiagnosing IPP as
traditional BPH will generally allow the physician to consider α-adrenoceptor antagonist therapy for an indeterminate length of time. α-adrenoceptor antagonists (e.g., tamsulosin) are generally ineffective for treating IPP because the target receptors, which are absent inferior to the bladder neck, are distal to the area of protrusion [11]. A 2013 study showed that men with IPP ≥ 10 mm had poor response to tamsulosin treatment among patients with LUTS due to benign prostatic obstruction, prostatic volume < 40 ml, and PSA < 1.5 ng/ml [14, 31]. BPH patients with IPP have exhibited less improvement of storage symptoms after 12 weeks of medication, suggesting that IPP may be a possible cause of intractable storage symptoms in early treatment [32]. A 2016 study found that increased IPP values are associated with lower response to α-adrenoceptor-specific management [33]. Tamsulosin therapy may be more effective in improving IPSS and Qmax in patients with mild IPP than in those with moderate or severe IPP [34]. Moreover, Hirayama et al. [35] deduced that combination therapy (i.e., α1-blockers and dutasteride) has insufficient efficacy towards LUTS and/or BPH with severe IPP due to a low proportion of stromal components in the prostate. For these reasons, well-designed studies are needed to assess the effect of IPP on the scope of treatment. IPP may have role in predicting the changes in postoperative IPSS, quality of life (QoL), Qmax, and PVR [6]. In a 2015 study, postoperative changes in IPSS and QoL score were higher in the significant IPP group (IPP ≥ 5 mm) than in the group with no significant IPP (IPP < 5mm) [36]. Another study demonstrated that IPP ≥ 5.5 mm was significantly associated with BOO [37]. Early detection of IPP by TRUS will obviate the delay encountered by refractory pharmacological response.

The extent of IPP is measured as the vertical distance from the tip of the intravesical protrusion to the circumference of the bladder at the bladder neck. With respect to this measurement, the grading system for IPP is: Grade I (< 5 mm); Grade II (5–10 mm); and Grade III (> 10 mm) [34]. Grade III is considered high-grade, or severe. The grade of IPP is strongly correlated to the clinical progression of BPH [38] as well as the urodynamic evidence of BOO. For patients with Grade I or II IPP, 21% are obstructed, whereas for patients with Grade III IPP, 96% are obstructed [4, 39]. IPP is a predictive marker of failure of trial without catheter in patients with acute urinary retention, with a 6-fold higher risk of failure in those with Grade II or III IPP [40]. Patients with Grade III have the greatest likelihood of developing acute urinary retention or requiring BPH-related prostatectomy, and thus may benefit from early surgical treatment [41]. Calcifications of the prostate will likely exacerbate LUTS. Furthermore, IPP may be classified as free-floating [42]. Though complete prostatic infarction is very rare, free-floating IPP presents as a mass of mummified BPH upon a probable spontaneous demarcation of the enlarged medial lobe. A potential rationalization for this may be torsion of the enlarged lobe with partial infarction of the base due to vasculopathic phenomena such as diabetes mellitus [42].

It may be difficult to clinically differentiate low flow caused by obstruction or detrusor underactivity. Hence, it has been suggested that pressure-flow or urodynamic studies be used as the gold standard for the evaluation of LUTS in men. However, it is invasive, time-consuming, and costly. Therefore, it is generally indicated for patients who are not responsive to initial conservative treatment or pharmacotherapy, patients with previous intervention, and those of extreme age range and equivocal cases [11]. That being said, we find it imperative that diagnosis of IPP be confirmed by transabdominal ultrasonography (TAUS) or TRUS of the prostate before proceeding with any sort of medical or surgical management. Measurement of IPP through TAUS in the sagittal view is noninvasive, readily available, cost-effective, and free of radiation hazards [6]. Despite this, TRUS may be preferable to TAUS due to the minimal effect of urine volume during the measurement of IPP [15]. TRUS can also more accurately predict prostatic volume when compared to TAUS [43]. With the cutoff at IPP ≥ 10 mm for the diagnosis of benign prostatic obstruction through TAUS, the sensitivity, specificity, and accuracy of the diagnosis have been calculated at 89.9, 97.5 and 92.7%, respectively [44]. Though uroflowmetry and measurement of PVR are essential and noninvasive means for evaluating BOO, they are inadequate to diagnose IPP. A simplified algorithm for the evaluation and management of IPP or non-IPP BOO is depicted in figure 1. More recently, suprapubic sonography of detrusor wall thickness and IPP has found to be a simple, noninvasive, and accurate system to assess prostatic obstruction in patients with LUTS due to BPH [45]. Flexible cystoscopy would need to be utilized to assess free-floating IPP [42]. Depending on the severity of BOO, cystoscopy may also be used to analyze bladder trabeculation and the formation of a post-prostatic pouch.

The significance of diagnosing IPP by TRUS is further noted by recent evidence that IPP can have an impact on the outcomes of robotic-assisted laparoscopic pros-
tatectomy (RALP) [46–48]. A 2016 study accounts that the presence and grade of IPP, as measured via preoperative TRUS, are significantly related to early recovery of urinary continence after RALP [46]. In addition, the grade of IPP has an impact on continence recovery at 12 months after surgery. Initially, the authors of this study concluded that a higher grade of IPP would have a higher degree of subclinical bladder dysfunction pre-RALP, resulting in a lower rate of urinary continence post-RALP. Secondly, a higher grade of IPP would lead to a higher chance to surgical damage at the smooth muscular internal urethral sphincter during bladder neck dissection. In considering the pentafecta (i.e., an achievement of the ideal outcome of RALP by the concurrent attainment of continence and potency with no evidence of biochemical recurrence, as well as early complications and positive surgical margins) [49], the authors suggest that both IPP plus its grade should be considered and that early TRUS is the best approach for this. The same study explained that if the patient had high-grade IPP, bidirectional bladder neck reconstruction using V-Loc sutures could be performed to effectively preserve the bladder neck [46]. The presence of IPP additionally has a positive outcome on the amelioration of obstructive symptoms in patients

Fig. 1. Evaluation algorithm for intravesical prostatic protrusion in bladder outlet obstruction. BMP = Basal metabolic panel; CBC = complete blood count; US, ultrasonography.
undergoing photoselective vaporization prostatectomy [50]. For such a procedure, postoperative outcome is determined by a preoperative cystoscopy to assess the IPP morphology [50].

**Conclusion**

IPP is correlated with the IPSS, prostatic volume, maximum flow rate, and PVR volume and hence can be used to assess the severity of BPH. It is important for physicians to perform TRUS, due to its superior practicality over TAUS, for the early detection of IPP as soon as they have suspicion of BOO. Diagnosing IPP by TRUS will expedite the necessary surgery and spare the patient pharmacological medications more fit for other forms of BPH. Findings of IPP and grade of IPP via TRUS can also aid urologists performing RALP in proper surgical planning and patient counseling. A higher grade of IPP might favor more surgical damage at the level of the internal urethral sphincter during bladder neck dissection. Patients with low-grade IPP have significantly higher chances of recovering full continence. Initial performance of TRUS along with DRE may abrogate delay in the appropriate evaluation and management of prostatic diseases. More prospective studies are essential to confirm the importance of IPP by early TRUS in the management of prostatic diseases.

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**References**

1. Randall A: Surgical pathology of prostatic obstructions. Baltimore, MD, Williams & Wilkins, 1931.
2. Luo GC, Foo KT, Kuo T, Tan G: Diagnosis of prostate adenoma and the relationship between the site of prostate adenoma and bladder outlet obstruction. Singapore Med J 2013;54:482–486.
3. Lieber MM, Jacobson DJ, McGree ME, St Sauver JL, Girman CJ, Jacobsen SJ: Intravesical prostatic protrusion in men in Olmsted County, Minnesota. J Urol 2009;182:2819–2824.
4. Foo KT: Solving the benign prostatic hyperplasia puzzle. Asian J Urol 2016;3:6–9.
5. Lee A, Lee HJ, Lim KB, Huang HH, Ho H, Foo KT: Can intravesical prostatic protrusion predict bladder outlet obstruction even in men with good flow? Asian J Urol 2016;3:39–43.
6. Sigdel GB, Belokar WK: Clinical significance of intravesical prostatic protrusion in patients with benign prostatic hyperplasia. J Univ Coll Med Sci 2015;3:6–10.
7. Zheng J, Pan J, Qin Y, Huang J, Luo Y, Gao X, Zhou X: Role for intravesical prostatic protrusion in lower urinary tract symptom: a fluid structural interaction analysis study. BMC Urol 2015;15:86.
8. Raychaudhuri B, Cahill D: Pelvic fasciae in urology. Ann R Coll Surg Engl 2008;90:633–637.
9. Park JS, Lee HW, Lee SW, Moon HS, Park HY, Kim YT: Bladder wall thickness is associated with responsiveness of storage symptoms to alpha-blockers in men with lower urinary tract symptoms. Korean J Urol 2012;53:487–491.
10. Kim JH, Shim JS, Choi H, Moon du G, Lee JG, Kim JJ, Bae HJ, Park JY: Terminal dribbling in male patients with lower urinary tract symptoms: relationship with International Prostate Symptom Score and with intravesical prostatic protrusion. BMC Urol 2015;15:89.
11. Kuo TL, Teo JS, Foo KT: The role of intravesical prostatic protrusion (IPP) in the evaluation and treatment of bladder outlet obstruction (BOO), Neurourol Urodyn 2016;35:535–537.
12. Suzuki T, Otsuka A, Ozono S: Combination of intravesical prostatic protrusion and resistive index is useful to predict bladder outlet obstruction in patients with lower urinary tract symptoms suggestive of benign prostatic hyperplasia. Int J Urol 2016;23:929–933.
13. Bude RO, Rubin JM: Relationship between the resistive index and vascular compliance and resistance. Radiology 1999;211:411–417.
14. Cumpanas AA, Botoca M, Minciu R, Bucuras V: Intravesical prostatic protrusion can be a predicting factor for the treatment outcome in patients with lower urinary tract symptoms due to benign prostatic obstruction treated with tamsulosin. Urology 2013;81:859–863.
15. Mariappan P, Brown DJ, McNeil AS: Intravesical prostatic protrusion is better than prostate volume in predicting the outcome of trial without catheter in white men presenting with acute urinary retention: a prospective clinical study. J Urol 2007;178:573–577.
16. Tjahjodjati I, Santosjo J: Relationship between prostate-urethral angle, intravesical prostatic protrusion, International Prostatic Symptom Score, and uroflowmetry in benign prostatic hyperplasia patients. Int J Integr Health Sci 2015;3:50–54.
17. Lu SY, Yang CM, Fan YH, Lin AT, Chen KK: Intravesical prostatic protrusion correlates well with storage symptoms in elderly male patients with non-neurogenic overactive bladder. Urol Sci 2016;27:17–20.
18. Keqin Z, Zhishun X, Jing Z, Haixin W, Dongqing Z, Benkang S: Clinical significance of intravesical prostatic protrusion in patients with benign prostatic enlargement. Urology 2007;70:1096–1099.
21. Shaﬁk A, Shaﬁk IA, el-Sibai O, Shaﬁk AA: Effect of uterine stimulation on vesical con- tractile activity. Am J Med Sci 2007;334: 240–243.

22. Shaﬁk A, el-Sibai O, Ahmed I: Effect of uter- nal dilation on vesical motor activity: iden- tiﬁcation of the urethralvesical reﬂex and its role in voiding. J Urol 2003;169:1017–1019.

23. Shaﬁk A, Shaﬁk AA, el-Sibai O, Ahmed I: Effect of vesical contraction on the non-sphincteric part of the urethra: recognition of vesicourethral inhibitory reﬂex. Int J Urol 2004;11:213–217.

24. Steers WD: Pathophysiology of overactive bladder and urge urinary incontinence. Rev Urol 2002;4 (Suppl 4):S7–S18.

25. Philippou P, Moraitis K, Masood J, Junaid I, Buchholz N: The management of bladder lidiathiss in the modern era of endourology. Urology 2012;79:980–986.

26. Kim JW, Oh MM, Park HS, Cheon J, Lee JG, Kim JJ, Moon du G: Intravesical prostatic protrusion is a risk factor for bladder stone in patients with benign prostatic hyperplasia. Urology 2014;84:1026–1029.

27. Wang D, Huang H, Law YM, Foo KT: Re- lationships between prostatic volume and intravesical prostatic protrusion on transab- dominal ultrasound and benign prostatic ob- struction in patients with lower urinary tract symptoms. Ann Acad Med Singapore 2015; 44:60–65.

28. Bantis A, Zissimopoulos A, Kalaytis C, Gi- annakopoulos S, Sountoulides P, Ageloniidou E, Voudalikakis C, Touloupidis S: Correla- tion of serum prostate speciﬁc antigen, the volume and the intravesical prostatic protrusion for diagnosing bladder outlet obstruction in patients with benign prostatic hyperplasia. Hell J Nucl Med 2007;10:138–143.

29. Hammerer PG, McNeal JE, Stamey TA: Cor- relation between serum prostate speciﬁc anti- gen levels and the volume of the individual glandular zones of the human prostate. J Urol 1995;153:111–114.

30. Xu D, Yu Y, Zhu Y, Huang T, Chen Y, Qi J: A new model consists of intravesical pros- tatic protrusion, prostate volume and serum prostate speciﬁc antigen in the evaluation of prostate cancer. Pathol Oncol Res 2014;20: 439–443.

31. Kaplan SA: Re: intravesical prostatic pro- trusion can be a predicting factor for the treatment outcome in patients with lower uri- nary tract symptoms due to benign prostatic obstruction treated with tamsulosin. J Urol 2014;191:157.

32. Park HK, Choo GY, Chung H, Paick SH, Kim HG, Kim HS, Lho YS, Hong WS, Yang SK: Effect of intravesical prostatic protrusion on the characteristics of lower urinary tract symptom change after 12-week drug medica- tion: a prospective analysis. Low Urin Tract Symptoms 2013;15:1–4.

33. Kalkani A, Tandogdu Z, Aydin M, Karaca AS, Hazar Al, Balci MB, Aydin M, Nuhoglu B: Intravesical prostatic protrusion: a po- tential marker of alpha-blocker treatment suc- cess in patients with benign prostatic enlarge- ment. Urology 2016;88:161–165.

34. Park HY, Lee JY, Park SY, Lee SW, Kim YT, Choi HY, Moon HS: Efficacy of alpha blocker treatment according to the degree of intravesical prostatic protrusion detected by transrectal ultrasonography in patients with benign prostatic hyperplasia. Korean J Urol 2012;53:92–97.

35. Hirayama K, Masui K, Hamada A, Shichiri Y, Masuzawa N, Hamada S: Evaluation of intravesical prostatic protrusion as a predic- tor of dutasteride-resistant lower urinary tract symptoms/benign prostatic enlargement with a high likelihood of surgical intervention. Urology 2015;86:565–569.

36. Lee JW, Ryu JH, Yoo TK, Byun SS, Jeong YJ, Jung TY: Relationship between intraves- ical prostatic protrusion and postoperative outcomes in patients with benign prostatic hyperplasia. Korean J Urol 2012;53:478–482.

37. Shin SH, Kim JW, Kim JW, Oh MM, Moon du G: Deﬁning the degree of intravesical prostatic protrusion in association with bladder outlet obstruction. Korean J Urol 2013; 54:369–372.

38. Lee LS, Sim HG, Lim KB, Wang D, Foo KT: Intravesical prostatic protrusion predicts clinical progression of benign prostatic enlarge- ment in patients receiving medical treatment. Int J Urol 2010;17:69–74.

39. Chia SJ, Heng CT, Chan SP, Foo KT: Cor- relation of intravesical prostatic protrusion with bladder outlet obstruction. BJU Int 2003;91:371–374.

40. Lebdai S, Ammi M, Bigot P, Cornu JN, Mat- thieu R, Descazeaud A, Azzouzi AR: Clinical impact of the intravesical prostatic protru- sion: a review by the LUTS committee of the French Urological Association. Prog Urol 2014;24:313–318.

41. Yoshida T, Kinoshita H, Yoshida K, Mishima T, Taniguchi H, Yanishi M, Komai Y, Yasuda K, Sugi M, Matsuda T: Intravesical prostatic protrusion as a predicting factor for the ad- verse clinical outcome in patients with symp- tomatic benign prostatic enlargement treated with dutasteride. Urology 2016;91:154–157.

42. Spranger R, Steiner H, Berger A, Horninger W, Bartsch G: Intravesical free-floating pro- state. J Urol 2002;168:2543–2544.

43. Stravodimos KG, Petrokas A, Kapetanakis T, Vourekas S, Kortisiadis G, Adamakis I, Mi- tropoulos D, Constantinides C: TRUS versus transabdominal ultrasound as a predictor of enucleated adenoma weight in patients with BPH: a tool for standard preoperative work- up? Int J Urol Nephrol 2009;41:767–771.

44. Yu HF, He YH, Yu KY, Wang Q, Huang PT, Yang Y, Wu DZ, Chen YH: Transabdominal ultrasound measurement of intravesical prostatic protrusion helps diagnosis of benign prostatic obstruction. Zhonghua Nan Ke Xue 2008;14:628–630.

45. Franco G, De Nunzio C, Leonardo C, Tubaro A, Cicciariello M, De Dominicis C, Miano L, Laurenti C: Ultrasound assessment of intra- vesical prostatic protrusion and detrusor wall thickness–new standards for noninvasive bladder outlet obstruction diagnosis? J Urol 2010;183:2270–2274.

46. Jo JK, Hong SK, Byun SS, Zargar H, Au- torino R, Lee SE: Urinary continence after robot-assisted laparoscopic radical prostatec- tomy: the impact of intravesical prostatic protrusion. Yonsei Med J 2016;57:1145–1151.

47. Kaplan SA: Re: Intravesical prostatic protru- sion as a predictor of early urinary continence recovery after laparoscopic radical prostatectomy. J Urol 2016;195:137–138.

48. Lee CH, Ha HK: Intravesical prostatic protrusion as a predictor of early urinary continence recovery after laparoscopic radical prostatectomy. Int J Urol 2014;21:653–656.

49. Patel VR, Sirvarama A, Coelho RF, Chau- han S, Palmer KJ, Orvieto MA, Camacho I, Coughlin G, Rocco B: Pentafecta: a new con- cept for reporting outcomes of robot-assisted laparoscopic radical prostatectomy. Eur Urol 2011;59:702–707.

50. Kim MS, Park KK, Chung BH, Lee SH: Ef- fect of photoselective vaporization prostata- tomy on lower urinary tract symptoms due to benign prostatic hyperplasia with or without intravesical prostatic protrusion. Korean J Urol 2013;54:36–41.