REVIEW ARTICLE

Benign Prostatic Hyperplasia: Terminology and Assessment

Part 1 in a Series on Benign Prostatic Hyperplasia

Matthias Oelke, Klaus Höfner, Udo Jonas, Karl-Ulrich Laval, Ulf Tunn

SUMMARY

Introduction: Benign prostatic hyperplasia (BPH) is one of the most common benign tumors in the aging man. The exact cause of BPH remains unknown. Methods: Systematic literature review from 1986 to 2006. Results: Clinical BPH is a disease that can develop as a result of hyperplasia and adenoma of the transition zone of the prostate. Clinical BPH is characterized by the presence of prostate enlargement, lower urinary tract symptoms, and bladder outlet obstruction, to varying degrees. No clear correlation between these three components has been found so far. The aim of assessment in clinical BPH is to clarify the interrelationships between prostate size, lower urinary tract symptoms and bladder outlet obstruction. In addition, the urinary tract should be investigated for functional or anatomical changes. Evaluation of clinical BPH should be performed according to the guidelines of the German Urological Association (DGU), which recommended a patient history, symptom and quality-of-life questionnaires, physical examination, laboratory tests, uroflowmetry, measurement of postvoid residual urine, and ultrasound of the urinary tract. Optional tests, for use where the diagnosis is still unclear or a special treatment is being considered, include transrectal ultrasound of the prostate, voiding diary, urodynamic measurement, excretory urography, urethro-cystoscopy and urethro-cystography.

Dtsch Arztebl 2007; 104(33): A 2261–7

Key words: prostatic hyperplasia, dysuria, diagnosis, ultrasonic diagnosis, guideline

This article is based on a systematic search of the literature published between 1986 and 2006. In addition, key articles on the epidemiology and pathophysiology of clinical benign prostatic hyperplasia (BPH) published before 1986 were also taken into account. The diagnostic recommendations made here are equivalent to those set forth in the German Urological Association Guidelines for the Diagnosis of Benign Prostatic Hyperplasia (1).

Terminology

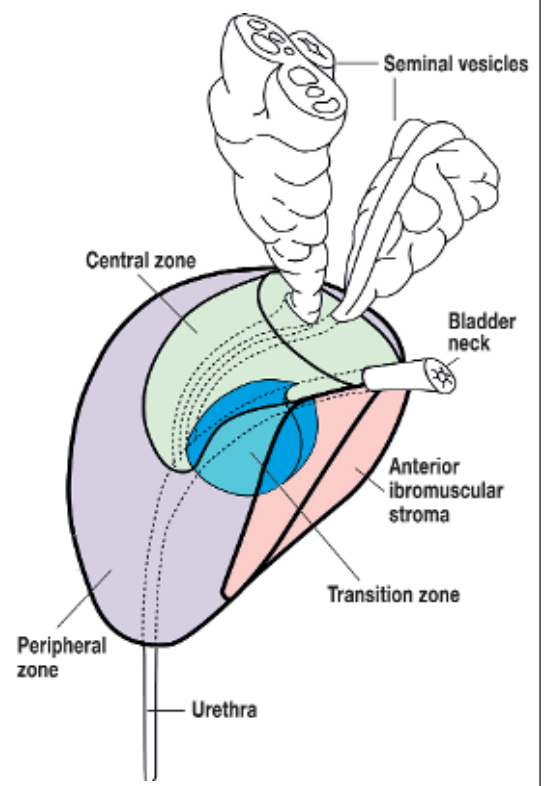
In healthy men, the prostate is approximately 20 to 25 ml in volume and consists of 30 to 50 tubuloalveolar glands. Based on the configuration of these glands and the morphology of the glandular cells, the prostate can be divided into four zones: the anterior, central, peripheral, and transition zones *(diagram 1)* (2, 3). Healthy men have a transition zone volume of approximately 5 ml.

Benign prostatic hyperplasia

BPH is characterized pathologically by the abnormal proliferation of cells (hyperplasia) in the transition zone, leading to structural changes accompanied by the formation of nodules, which can consist of stromal tissue (i.e. involving undifferentiated mesenchymal cells [mesenchymal hyperplasia], fibroblasts [fibroblastic hyperplasia], smooth muscle cells [leiomyomatous hyperplasia], and fibromuscular stroma [fibromuscular hyperplasia]) or glandular tissue (glandular hyperplasia). In most cases, however, simultaneous hyperplasia of both tissue types can be observed. Nodular hyperplasia can develop in the lateral (lateral lobe hyperplasia) and/or dorsal (median lobe hyperplasia) region of the transition zone. The proliferation of cells leads to an increase in the size of the transition zone and, thus, of the entire prostate gland. Although a number of contributing factors have been identified, the precise etiology of the hyperplastic changes observed in BPH remains unknown.

Urologische Klinik, Academisch Medisch Centrum, Universität von Amsterdam: Dr. med. Oelke; Urologische Klinik, Evangelisches Krankenhaus Oberhausen: Prof. Dr. med. Höfner; Urologische Klinik, Medizinische Hochschule Hannover: Prof. Dr. med. Jonas; Urologische Praxis, Münsterstraße, Düsseldorf: Dr. med. Laval; Urologische Klinik, Klinikum Offenbach am Main: Prof. Dr. med. Tunn

DIAGRAM 1



Zonal anatomy of the prostate after McNeil, adapted from (3)

BPH is a widespread disease and its prevalence increases steadily with advancing age. Meta-analyses of autopsy studies have shown that approximately 50% of men in the sixth decade of life and 90% of men in the ninth decade of life demonstrate histologic BPH *(diagram 2)* (4).

Symptoms of benign prostatic hyperplasia

Clinical BPH is characterized by different degrees of prostate enlargement, lower urinary tract symptoms (LUTS), and bladder outlet obstruction (BOO) *(box 1)* (1). A large number of studies have shown that there is no clear correlation between these individual measures of disease severity (5–7).

In any single patient, a change in one of these measures can thus occur alone or in combination with a change in one or two of the other measures. As a result, prostate size, LUTS, and BOO need to be assessed separately, and inferences about one measure cannot be drawn based on changes observed in another. The pathophysiological concept can best be described using a Venn diagram *(diagram 3)* (8).

Benign prostate enlargement

It has been estimated that in approximately 50% of men with histologic BPH, prostate enlargement can be detected by digital rectal examination (DRE); just as the prevalence of histologic BPH increases with advancing age, so too does the likelihood of detecting prostate enlargement by DRE. In one study, epidemiological data on clinical BPH were collected from men between 50 and 80 years of age living in the German city of Herne (9). Nationwide projections based on these data show that more than 3.2 million out of 11.6 million men over the age of 50 in Germany have an enlarged prostate.

Lower urinary tract symptoms

The symptoms caused by BPH prompt many men to seek medical care and can be divided into two categories: obstructive and irritative *(table 1)*. In an international study, men were

asked about the frequency of individual symptoms and the resulting changes in quality of life (10). Although obstructive symptoms occurred more frequently among respondents, irritative symptoms caused greater impact on well-being and quality of life. The study also showed that men with mild symptoms experienced only a minimal decrease in quality of life and thus did not seek medical care. In such cases, bladder outlet obstruction in the absence of LUTS may remain unrecognized ('silent obstruction'). In the German epidemiological study of clinical BPH, 29.3% of men between 50 and 80 years of age indicated that they experience moderate to severe symptoms, which translates to approximately 5 million men in Germany in need of treatment (9). In another study, changes in symptom severity were assessed over a period of 4 years in untreated men (11). During the first year, symptoms improved in 20% of men, whereas only 3% of men reported improvements during the fourth year. In contrast, a worsening of symptoms was reported by 14% of men after 1 year and by 31% of men after 4 years.

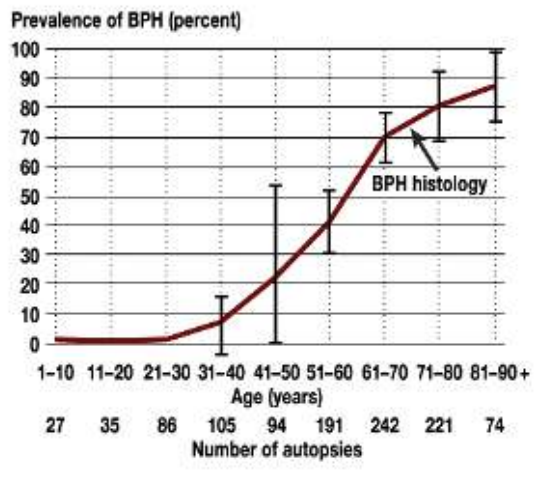
The symptoms and their degree of severity are not helpful in identifying the underlying disease (12). The prevalence of LUTS is similar in men and women within the same age group, and the causes of LUTS include, in addition to BPH itself, age-related changes in detrusor muscle function and degenerative, metabolic, and neurogenic processes in the human body. In particular, lesions of the central nervous system (e.g. cerebral vascular disease or Parkinson's disease) often lead to detrusor overactivity, which manifests clinically as increased micturition frequency (pollakisuria, nocturia), as urinary urgency, or as urinary incontinence. The LUTS caused by these diseases cannot be distinguished from those caused by BPH in men. Symptoms typically associated with BPH, including poor urinary stream, delayed micturition, and postvoid residual urine, can also be caused by impaired detrusor contractility, which in one study was shown to be the true source of symptoms in 25 to 31% of men with suspected bladder obstruction (13). Because LUTS are neither gender nor disease specific, the term "prostatism" should no longer be used to describe them.

Bladder outlet obstruction

The narrowing of the prostatic urethra due to the enlarged transitional zone is the pathophysiological basis of bladder outlet obstruction. Urodynamic studies show that only about 60% of men with symptoms actually have bladder outlet obstruction (14). In asymptomatic men, on the other hand, bladder outlet obstruction was found in 52%, so that symptomatic and asymptomatic men with BPH have almost the same chance of bladder outlet obstruction. Studies in Germany have shown that about 2.1 million men older than 50 have a bladder outlet obstruction (9).

Infravesical mechanical obstruction can cause changes of the bladder wall and function. Subsequent to the obstruction, the detrusor muscle thickens and its micostructure undergoes changes. Microscopic investigations of the bladder wall in men with bladder

DIAGRAM 2



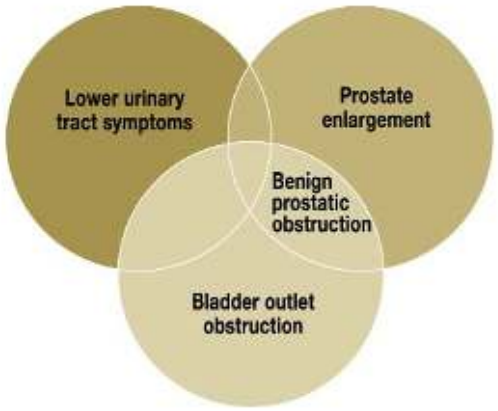
Prevalence of benign hyperplasia (BPH) in the different decades of life, adapted from (4)

BOX 1

Terminology of BHP associated changes to the lower urinary tract

- BPH benign prostatic hyperplasia (histological BPH)
- BPE benign prostatic enlargement
- BOO bladder outlet obstruction
- BPO benign prostatic obstruction (BOO caused by BPE)
- LUTS lower urinary tract symptoms

DIAGRAM 3



Schematic representation of the associations between benign prostate enlargement (BPE), lower urinary tract symptoms (LUTS), and bladder outlet obstruction (BOO) in patients with benign prostatic hyperplasia (BPH), adapted from (8)

outlet obstruction show muscle cell hypertrophy and infiltration of connective tissue between muscle cells and muscle cell bundles (15). In principle, all changes to the bladder wall are reversible if the obstruction is treated in the early stages. After transurethral resection of the prostate, bladder wall thickening is not detectable anymore in up to 88% of patients some 4 to 12 weeks after treatment (16).

Many morphological and functional changes of the urinary tract are probably directly or indirectly related to bladder outlet obstruction (17). The following changes are found disproportionally often in patients with BPH (data reported as a percentage of all BPH patients) and are reversible with adequate urological treatment:

- Bladder trabeculation (33%) due to deposits of collagen and elastic fibrils in the bladder wall
- Vesicoureteral reflux (14%)
- Increased serum creatinine concentrations (14% of symptomatic and 2% of asymptomatic patients with BHP). In patients with impaired renal function, perioperative morbidity is twofold higher and mortality sixfold higher (18).
- Bilateral hydronephrosis (7%) *(figure)*. It remains unknown whether renal failure requiring dialysis develops as a result of upper urinary tract dilatation because data from untreated patients are lacking.
- Macrohematuria (5%)
- Bladder diverticula (3.3%)
- Bladder calculi (3%)
- Urinary retention (incidence 1 to 2% per year).

The exact genesis is unknown, but urinary retention depends on the severity and form of the symptoms in the lower urinary tract, bladder outlet obstruction, age, prostatic enlargement,

TABLE 1

Obstructive and irritative symptoms

Obstructive symptoms (micturition symptoms)	Irritative symptoms (symptoms of urine storage)
Hesitancy	Urgency
Decreased urinary stream	Pollakisuria
Abdominal straining	Nocturia
Prolonged micturition	Urge incontinence
Intermittency	
Small volumes	
Postvoid dribbling	
Feeling of incomplete voiding/urinary retention	
Overflow incontinence	

and serum concentration of prostate specific antigen (PSA) (11, 19). Since only few patients with bladder outlet obstruction develop urinary retention, additional factors are assumed to have a role in its development.

Postvoid residual urine is common, but there is no definite association with bladder outlet obstruction and amount of postvoid residual urine. Some 30% of men with postvoid residual urine do not have bladder outlet obstruction, and 24% of men with bladder outlet obstruction have no residual urine after voiding (20). Urinary tract infections occur in 20% of patients, but no direct correlation with postvoid residual urine or the amount of residual urine has been found (21). The clinical aspects of benign prostate syndrome are summarized in *box 2*.

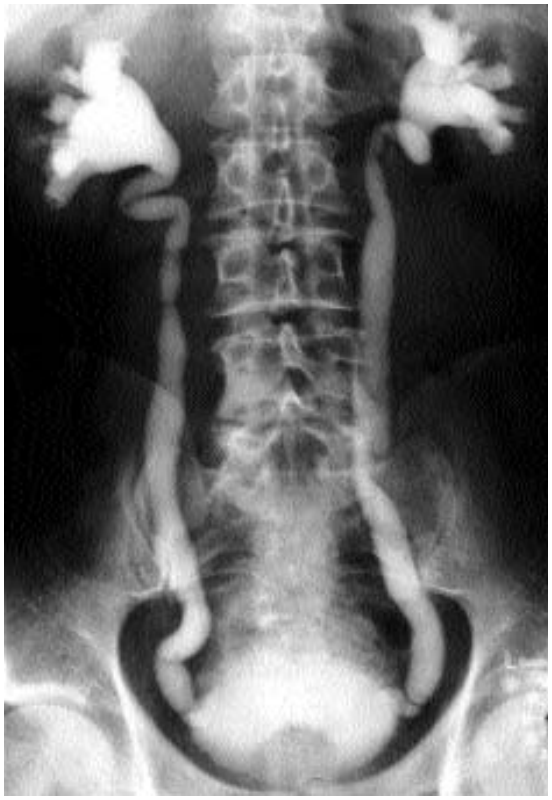


Figure:
Bilateral hydronephrosis in prostate enlargement and urodynamically proved bladder outlet obstruction. Voiding urogram of a patient with compensated renal failure (serum creatinine 155 µmol/l), but without lower urinary tract symptoms

BOX 2

Clinical aspects of BPH

- In men with benign prostatic hyperplasia, the enlarged prostate can occur in isolation or accompanied by lower urinary tract symptoms and/or bladder outlet obstruction
- No clear associations exist between prostate enlargement, symptoms, and bladder outlet obstruction
- Irritative and obstructive bladder symptoms prompt the patient to consult his doctor
- Irritative bladder symptoms compromise wellbeing and quality of life more than obstructive bladder symptoms
- Bladder outlet obstruction can result in irreversible damage to the lower or upper urinary tract

Diagnosis of clinical BPH

Most men with BPH complain of symptoms of the lower urinary tract. Doctors should ascertain whether the symptoms are really caused by BPH or by another pathology. The relation between symptoms, prostate enlargement, and bladder outlet obstruction also needs to be ascertained. The diagnosis of the benign prostatic syndrome should follow the guidelines of the German Urological Association (1). In the assessment, distinction needs to be made between obligatory and optional investigations. Optional investigations follow in cases where diagnostic uncertainties persist after the basic examination or additional exams are needed to choose a certain therapeutic approach.

Obligatory examinations

Medical history: In addition to the general and neurourological history, a detailed history with regard to micturition should be taken. In the micturition history, obstructive and irritative symptoms are elicited and possibly quantified *(table 1)*. The history should also clarify whether drugs are being taken that might affect the functioning of the lower urinary tract. Anticholinergics, antidepressants, antiparkinson drugs, and calcium channel antagonists can cause hypocontractility of the detrusor muscle, leading to postvoid residual urine or urinary retention. Parasympathomimetic drugs, on the other hand, increase detrusor contractility and can cause or increase urge incontinence. Alpha receptor blockers, muscle relaxants, or hydrated ergot alkaloids can cause urinary incontinence by lowering urethral resistance, and alpha sympathomimetics can cause postvoid residual urine or urinary retention by increasing urethral resistance. Diuretics increase diuresis and can increase nocturia or urge incontinence. Alcohol can cause urinary retention subsequent to detrusor relaxation.

Symptom and quality of life questionnaires: The frequency and extent of symptoms can be quantified by using a questionnaire, and changes during therapy can thus be documented. The International Prostate Symptom Score (IPSS) is the most commonly used questionnaire *(table 2)*. The first 7 questions capture the frequency of symptoms of the lower urinary tract within the preceding 4 weeks, the 8th question the extent to which the patient's quality of life is compromised. The symptom score is obtained by adding up the answers to questions 1 to 7 and will be a number between 0 and 35. On the basis of this score, the symptoms can be classed as mild (IPSS score 0–7), moderate (IPSS score 8–19), or severe (IPSS score 20–35).

Physical examination: In addition to a general physical examination, the patient's neurourological status should be assessed as a means of orientation; this provides information on the anal sphincter muscle tone and the sensorimotor state of the lower extremities, the perineum, and the genitals. During the basic neurological examination, the reflex pathways of the lower extremities should also be assessed *(table 3)*, to enable conclusions about the functional fitness of the neural pathways in the bladder and the bladder sphincter (sympathetic innervation Th11 to L2, parasympathetic and somatic innervation S2 to S4).

TABLE 2

International prostate symptom score

IPSS questions						
All responses relate to the past 4 weeks **Please tick**	**Not at all**	**Less than 1 time in 5 (< 20%)**	**Less than half the time**	**About half the time (ca. 50%)**	**More than half the time**	**Almost always**
1. How often have you had a sensation of not emptying your bladder completely after you finished urinating?	0	1	2	3	4	5
2. How often have you had to urinate again less than two hours after you finished urinating?	0	1	2	3	4	5
3. How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5
4. How often have you found it difficult to postpone urination?	0	1	2	3	4	5
5. How often have you had a weak urinary stream?	0	1	2	3	4	5
6. How often have you had to push or strain to begin urination?	0	1	2	3	4	5
7. How many times did you most typically get up to urinate from the time you went to bed at night until the time you got up in the morning?	Never (0)	Once (1)	Twice (2)	Three times (3)	Four times (4)	Five or more times (5)
Symptom score =						

Quality of life index							
If you were to spend the rest of your life with your urinary condition the way it is now, how would you feel about that?	Delighted (0)	Pleased (1)	Mostly satisfied (2)	Mixed – about equally satisfied and dissatisfied (3)	Mostly dissatisfied (4)	Unhappy (5)	Terrible (6)
Quality of life =							

The digitorectal examination provides information on the size, pain, and consistency of the prostate. Compared with transrectal ultrasonography, the prostate volume is usually underestimated by 10 to 20% on digital rectal examination. The probability of having prostatic cancer in patients with suspected BPH is 4.5 to 16% (22).

Laboratory tests: Serum creatinine and PSA concentrations should be measured in all patients, and a urinary analysis should be performed. If creatinine concentrations are raised further diagnostic tests of the upper urinary tract are advised to exclude a postrenal cause. The PSA measurement helps to assess the likelihood of a prostatic carcinoma and judge the risk of progression (increased likelihood of a symptomatic progression or urinary retention when PSA > 1.6 µg/l) (23). Raised PSA values (> 4 µg/l) can also be caused by acute urinary retention, acute prostatitis, prostatic infarctions, or manipulations to the prostate. On the other hand, 5 alpha reductase inhibitors halve serum PSA concentrations after 6 to 12 months' treatment. Some 20% of patients with prostate cancer do not have raised PSA values (< 4 µg/l), however – in such patients, only transrectal palpation of the prostate or repeated PSA measurements (PSA velocity) can provide indications about the cancer. Urinalysis is done to rule out urinary tract infections and hematuria. Such urinary changes can occur in BPH, but can also indicate other pathologies affecting the urinary tract, such as a bladder tumor or bladder calculi.

Uroflowmetry: Measuring urinary flow in patients with bladder outlet obstruction can document a decrease in maximum and mean urinary flow rates as well as an increase in the

TABLE 3

Basic neurological examination

Reflex	Reflex arc over the segments of the spinal cord	Performing the test
Cremasteric reflex	L1 – L2	Stroking the inner thigh results in contraction of the equilateral cremasteric muscle, and the equilateral scrotal sac is lifted
Adductor reflex	L2 – L4	The reflex hammer is used closely below the medial condyle of femur (Pes anserinus) and an adductor movement is observed in the same leg
Bulbocavernosus reflex	S3 – S4	Pinching the penile glans leads to contraction of the anal sphincter
Anal reflex	S3 – S5	Stroking the perianal region leads to contraction of the anal sphincter

time to maximum flow rate and flow time. For uroflowmetry assessment, a micturition volume of ≥ 150 ml is recommended, and in case of a reduced stream, at least one repeat examination should be done. In addition to the maximum urinary flow rate as the most important individual variable, the curve provides important insights into the underlying bladder voiding disorder and the effect of treatment. A maximum urinary flow of < 15 ml/s indicates a urinary voiding disorder, which may also be caused by decreased detrusor contractility. Only a maximum urinary flow ≥ 15 ml/s indicates unimpaired bladder voiding, so that uroflowmetry can be used to screen patients with LUTS.

Postvoid residual urine: This is usually measured by ultrasonography, but it can also be done with a catheter. In routine practice, volumes of residual urine > 50 ml are regarded as significant. Postvoid residual urine can occur in bladder outlet obstruction in BHP, but also in other forms of bladder outlet obstruction (for example, urethral stricture, meatus stenosis) or detrusor underactivity. Global bladder function can be assessed by measurement of postvoid residual urine. No data exists to support the assumption that patients with postvoid residual urine will develop urinary retention later on.

Urosonography: The bladder and kidneys should be examined in every patient. Since a raised serum creatinine concentration occurs only after about 50% of nephrons have failed, an ultrasound investigation of the kidneys can diagnose upper urinary tract dilatation even in the absence of raised creatinine. Sonography of the kidneys and bladder can also detect calculi, diverticles, or neoplasms in the urinary tract. Since bladder outlet obstruction results in compensatory hypertrophy of the detrusor muscle, ultrasound measurements of the detrusor thickness when the bladder contains at least 250 ml urine can be used as an additional variable in assessing the degree of obstruction. A detrusor wall thickness of ≥ 2 mm indicates a bladder outlet obstruction with 95% certainty (24).

Optional examinations

TRUS: Transrectal ultrasonography (TRUS) can determine the volume of the prostate more precisely than transabdominal volumetry; the methods differ by about 10%. Only TRUS can visualize and assess the zonal anatomy of the prostate.

Micturition protocol: Patients record the exact time and volume of each micturition over 2 to 3 days. The protocol records objectively the number of micturitions during the day and at night, the urinary volume, and the ingested amount of fluids. This enables objective judgment of pollakisuria, nocturia, polydipsia, or nocturnal polyuria, and their control during treatment.

Urodynamic investigations: These should be used only if standard diagnostic tests have not been able to assess the degree of obstruction. By simultaneously measuring urinary stream and vesical pressure, it is possible to differentiate patients with weakened stream subsequent to bladder outlet obstruction or detrusor underactivity (25). The ability to distinguish between a weak detrusor and/or obstruction makes this the investigation of choice particularly in patients with a concomitant neurological disorder (for example, stroke, Parkinson's disease, or herniated disk) or diabetes or diabetic neuropathy.

Intravenous urography: This advanced diagnostic test is indicated in hematuria, recurrent urinary tract infection, urolithiasis, previous operations on the urinary tract, or another pathological finding on ultrasonography. Urethro-cystoscopy and urethro-cystography should also be limited to specific questions – for example, in bladder tumors or urethral stricture.

Conflict of Interest Statement
The authors declare that no conflict of interest exists according to the guidelines of the International Committee of Medical Journal Editors.

Manuscript received on 22 June 2006, final version accepted on 18 October 2006.

Translated from the original German by Matthew D. Gaskins and Dr Birte Twisselmann.

REFERENCES

1. Berges R, Dreikorn K, Höfner K et al.: Leitlinien der Deutschen Urologen zur Diagnostik des benignen Prostatasyndroms (BPS). Urologe [A] 2003; 42: 584–90.
2. McNeal JE: Regional morphology and pathology of the prostate. Am J Clin Pathol 1968; 49: 347–57.
3. Roehrborn CG, McConnell JD: Etiology, pathophysilogy, epidemiology, and natural history of benign prostatic hyperplasia. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ (eds.): Campbell´s Urology. Philadelphia, London, New York: Saunders 2002; 1297–336.
4. Berry SJ, Coffey DS, Walsh PC, Ewing LL: The development of human benign prostatic hyperplasia with age. J Urol 1984; 132: 474–9.
5. Barry MJ, Cockett AT, Holtgrewe HL, McConnell JD, Sihelnik SA, Winfield HN: Relationship of symptoms of prostatism to commonly used physiological and anatomical measures of the severity of benign prostatic hyperplasia. J Urol 1993; 150: 351–8.
6. Rosier PF, de la Rosette JJ: Is there a correlation between prostate size and bladder outlet obstruction? World J Urol 1995; 13: 9–13.
7. van Venrooij GE, Boon TA, de Gier RP: International prostate symptom score and quality of life assessment versus urodynamic parameters in men with benign prostatic hyperplasia symptoms. J Urol 1995; 153: 1516–9.
8. Hald T: Urodynamics in benign prostatic hyperplasia. A survey. Prostate 1989; 2 (Suppl.): 69–77.
9. Berges RR, Pientka L, Höfner K, Senge T, Jonas U: Male lower urinary tract symptoms and related health care seeking in Germany. Eur Urol 2001; 39: 682–7.
10. Peters TJ, Donovan JL, Kay HE et al.: The International Continence Society „Benign Prostatic Hyperplasia“ Study: the bothersomeness of urinary symptoms. J Urol 1997; 157: 885–9.
11. Jimenez-Cruz F: Identifying the patients at risk for disease progression. Eur Urol 2003; Suppl 2: 6–12.
12. Chancellor MB, Rivas DA, Keeley FX, Lotfi MA, Gomella LG: Similarity of the American Urological Association Symptom Index among men with benign prostatic hyperplasia (BPH), urethral obstruction not due to BPH and detrusor hyperreflexia without outlet obstruction. Br J Urol 1994; 74: 200–3.
13. Ameda K, Sullivan MP, Bae RJ, Yalla SV: Urodynamic characterization of nonobstructive voiding dysfunction in symptomatic elderly men. J Urol 1999; 162: 142–6.
14. Reynard JM, Yang Q, Donovan JL et al.: The ICS-“BPH“ Study: uroflowmetry, lower urinary tract symptoms and bladder outlet obstruction. Br J Urol 1998; 82: 619–23.
15. Gilpin SA, Gosling JA, Barnard RJ: Morphological and morphometric studies of the human obstructed, trabeculated urinary bladder. Br J Urol 1985; 57: 525–9.
16. Kojima M, Inui E, Ochiai A et al.: Reversible change of bladder hypertrophy due to benign prostatic hyperplasia after surgical relief of obstruction. J Urol 1997; 158: 89–93.
17. McConnell JD, Barry MJ, Bruskewitz RC et al.: Benign prostatic hyperplasia: diagnosis and treatment. Clinical practice guideline No.8, AHCPR Publication No. 94-0582, U.S. Department of Health and Human Services, Agency for Health Care Policy and Research, Rockville Maryland 1994.
18. Mebust WK, Holtgrewe HL, Cockett AT, Peters PC: Transurethral prostatectomy: immediate and postoperative complications. A cooperative study of 13 participating institutions evaluating 3,885 patients. J Urol 1989; 141: 243–7.
19. Jacobsen SJ, Jacobsen DJ, Girman CJ et al.: Natural history of prostatism: risk factors for acute urinary retention. J Urol 1997; 158: 481–7.
20. Leblanc G, Tessier J, Schick E: The importance and significance of post-micturitional bladder residue in the evaluation of prostism. Prog Urol 1995; 5: 511–4.
21. Hampson SJ, Noble JG, Rickards D, Milroy EJ: Does residual urine redispose to urinary tract infection? Br J Urol 1992; 70: 506–8.

22. Akdas A, Tarcan T, Turkeri L, Cevik I, Biren T, Ilker Y: The impact of prostate-specific antigen density in predicting prostate cancer when serum prostate-specific levels are less than 10 ng/ml. Eur Urol 1996; 29: 189–92.
23. 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 Engl J Med 2003; 349: 2387–98.
24. Oelke M, Höfner K, Wiese B, Grünewald V, Jonas U: Increase in detrusor wall thickness indicates bladder outlet obstruction (BOO) in men. World J Urol 2002; 19: 443–52.
25. Höfner K, Jonas U: Urodynamics in benign prostatic hyperplasia. Curr Opion Urol 1996; 6: 184–8.

Corresponding author
Dr. med. Matthias Oelke
Urologische Klinik, Academisch Medisch Centrum
Universität von Amsterdam, Meibergdreef 9
1105 AZ Amsterdam, Netherlands
m.oelke@amc.uva.nl