LOWER URINARY TRACT SYMPTOMS/BENIGN PROSTATIC HYPERPLASIA: MINIMIZING MORBIDITY CAUSED BY TREATMENT

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ABSTRACT

The beneficial effects of treatment for lower urinary tract symptoms (LUTS) suggestive of benign prostatic hyperplasia (BPH), or LUTS/BPH, have to be balanced against the morbidity associated with treatment. Invasive surgery, such as transurethral resection of the prostate, has been associated with irreversible complications (eg, impotence and retrograde ejaculation). α1-Adrenoceptor antagonists provide effective and fast relief of LUTS/BPH. In contrast to finasteride, they are not associated with sexual dysfunction (eg, decreased libido or impotence). α1-Adrenoceptor antagonists induce adverse events associated with interference with blood pressure regulation. The α1A/α1D-adrenoceptor antagonist tamsulosin has the lowest potential to interfere with blood pressure regulation and induce related adverse events. In addition, tamsulosin seems to be as well tolerated as phytotherapy, except for a higher incidence of abnormal ejaculation. Abnormal ejaculation occurs in 4% to 11% of patients receiving an α1-adrenoceptor antagonist, which is, however, well tolerated; <1% of patients discontinue because of this adverse event. In placebo-controlled trials, abnormal ejaculation has been predominantly reported for tamsulosin, but in most direct comparative studies, the incidence was comparable to that of other α1-adrenoceptor antagonists. Men with LUTS/BPH have an increased risk of impaired sexual function. However, α1-adrenoceptor antagonists, such as tamsulosin, may slightly improve sexual dysfunction together with LUTS problems. Combination therapy of an α1-adrenoceptor antagonist and finasteride has a similar adverse-event profile as each monotherapy, except for an increased risk of abnormal ejaculation. The discontinuation rate because of adverse events does not seem to be higher than with monotherapy. Medical therapies, and particularly α1-adrenoceptor antagonists such as tamsulosin, can be considered a first-line treatment option for LUTS/BPH because they provide effective relief of bothersome LUTS with excellent tolerability.


The main objectives of treatment for lower urinary tract symptoms (LUTS) suggestive of benign prostatic hyperplasia (BPH), or LUTS/BPH, are to provide fast and sustained relief of bothersome symptoms, to improve quality of life (QOL), and to control disease progression. However, the beneficial effects of treatment should be balanced against the eventual risks and bother resulting from complications or adverse events associated with treatment. A treatment with a good efficacy/tolerability or benefit/risk ratio will be more acceptable to patients, and it will have a more positive effect on QOL. Of course, this should also be balanced against the extent to which the patient is bothered by the urinary symptoms and how these affect the activities of daily living of the patient and the patient’s partner. For example, a patient with severe symptoms and significant bother will accept considerably more morbidity after a surgical procedure or will complain less about adverse events of medical therapy than a patient with minimal symptoms and bother. The rate of adverse events or complications associated with treatment for LUTS/BPH should therefore be examined very carefully. This article aims to evaluate morbidity associated with the various medical treatment options for LUTS/BPH.

MEDICAL THERAPY

Compared with surgery, medical therapy has the advantage because it is a noninvasive therapy, with reversible adverse events on drug discontinuation.
α₁-Adrenoceptor antagonists are the most frequently prescribed medical therapy, compared with the 5α-reductase inhibitor finasteride or phytotherapy. This is probably because α₁-adrenoceptor antagonists provide the most rapid and effective relief of voiding and bothersome storage symptoms of all currently available medical therapies, thereby rapidly improving QOL. However, the effect on QOL is also dependent on the side-effect profile of treatment.

5α-REDUCTASE INHIBITORS

5α-Reductase inhibitors, such as finasteride, induce adverse events related to sexual dysfunction, such as impotence (reported by 3% to 16% of treated patients [treatment emergent]) and decreased libido (reported by 2% to 10% of treated patients [treatment emergent]). In addition, abnormal ejaculation (eg, ejaculation failure) is reported by 0% to 8% of patients. Adverse effects related to sexual function (eg, decreased libido and impotence). However, none–subtype-selective α₁-adrenoceptor antagonists, such as doxazosin and terazosin, may interfere with blood pressure regulation, which induces adverse events, such as dizziness, asthenia, somnolence, first-dose symptomatic orthostatic hypotension, and potentially syncope. Adverse events associated with blood pressure regulation, such as symptomatic orthostatic hypotension or syncope, are important risk factors for falling, which may cause major injuries, such as fractures. These cardiovascular adverse events will be discussed first. In addition, sexual function has been identified by LUTS/BPH patients to be a particularly important factor influencing QOL. Because fast improvement of QOL is a very important goal in the treatment of LUTS/BPH, the effect of α₁-adrenoceptor antagonists on sexual function, particularly on abnormal ejaculation, is also discussed.

ADVERSE EVENTS ASSOCIATED WITH INTERFERENCE WITH BLOOD PRESSURE REGULATION

When the currently available α₁-adrenoceptor antagonists are compared, their efficacy in relieving LUTS/BPH is similar when they are administered at therapeutic doses. α₁-Adrenoceptor antagonists, however, can be differentiated from each other by their side-effect profile. The subtype-selective α₁A/α₁D-adrenoceptor antagonist tamsulosin displays lower selectivity for α₁B-adrenoceptors—which are presumed to be involved in blood pressure regulation, especially in the elderly population—than for α₁-adrenoceptor subtypes believed to be responsible for the desired effect on the lower urinary tract (α₁A and α₁D-adrenoceptors). In contrast, none of the other currently available α₁-adrenoceptor antagonists demonstrate clear selectivity for any of the α₁-adrenoceptor subtypes. Compared with the non–subtype-selective α₁-adrenoceptor antagonists, tamsulosin has been shown to have the lowest potential to interfere with blood pressure regulation and induce related adverse events. A meta-analysis, which indirectly compared the tolerability of alfuzosin, doxazosin, tamsulosin, and terazosin reported in randomized, placebo-controlled trials, showed that alfuzosin (especially the sustained-release formulation) and tamsulosin 0.4 mg once daily were associated with the lowest discontinuation rate from the adverse events dizziness and orthostatic hypotension (Figures 1–3). Results from direct comparative studies are discussed in more detail here. A 12-week, double-blind, direct comparative study between tamsulosin 0.4 mg once daily (n = 132) and alfuzosin 2.5 mg 3 times daily (n = 124) showed that alfuzosin statistically significantly reduced blood pressure compared with baseline, whereas this was not the case with tamsulosin. The difference in blood pressure reduction between tamsulosin and alfuzosin was most pronounced in elderly patients. Another double-blind, randomized, controlled trial studied the orthostatic effects in 73 elderly subjects (≥60 years of age) receiving tamsulosin 0.4 mg once daily (n = 37) or alfuzosin twice daily (n = 36) during 1 week. Tamsulosin tended to cause less symptomatic orthostatic hypotension (defined as a decrease in systolic blood pressure >20 mm Hg and the occurrence of feeling faint, dizzy, lightheaded, or nauseous) during orthostatic stress testing than alfuzosin (0.4% vs 2.8% of patients, respectively). Furthermore, compared with alfuzosin, tamsulosin caused fewer adverse events, such as headache (5.4% vs 11.1%, respectively), faintness (5.4% vs 13.9%, respectively), and digestive symptoms (5.4% vs 19.4%, respectively), and interfered less with overall well-being. A double-blind, randomized study in 50 normotensive elderly subjects has further shown that the non–subtype-selective α₁-adrenoceptor antagonist terazosin (uptitrated from 1 mg to 5 mg once daily) caused statistically more symptomatic orthostatic hypotension than tamsulosin (0.4 mg once daily) during orthostatic stress testing (36% vs 4% of patients, respectively; P = 0.011). A recent open-label, randomized, multicenter study randomized 1789 patients with moderate-to-severe LUTS/BPH to either tamsulosin 0.4 mg once daily (n = 1002) or terazosin uptitrated within 2 weeks to 5 mg once daily for 8 weeks (n = 981). Terazosin reduced
blood pressure to a larger extent than tamsulosin, particularly at day 19 when terazosin was fully titrated to 5 mg. In addition, tamsulosin caused statistically significantly (P < 0.001) fewer adverse events associated with blood pressure regulation, such as dizziness and fatigue (Figure 4).

Thus, among the currently available \( \alpha_1 \)-adrenoceptor antagonists, the subtype-selective \( \alpha_{1A}/\alpha_{1D} \)-adrenoceptor antagonist tamsulosin seems to least modify blood pressure and induce cardiovascular-related adverse events. This favorable tolerability profile of tamsulosin will probably also result in an increased treatment compliance; cardiovascular-related adverse events, such as asthenia and dizziness, which developed during treatment with the non–subtype-selective \( \alpha_1 \)-adrenoceptor antagonists doxazosin\(^{20} \) and terazosin,\(^{21} \) were the major reasons for patients to discontinue these treatments. The very good tolerability of tamsulosin was also demonstrated in a double-blind, randomized direct comparative study with \( S \) repens \( 320 \) mg once daily (Figure 5).\(^{22} \) The only adverse event that occurred with a higher incidence with tamsulosin than with \( S \) repens was abnormal ejaculation.

**Abnormal Ejaculation**

As mentioned previously, \( \alpha_1 \)-adrenoceptor antagonists are not associated with sexual dysfunction (decreased libido and impotence). Abnormal ejaculation (e.g., retrograde ejaculation, reduced ejaculate volume, or absence of ejaculate volume) has, however, been attributed to \( \alpha_1 \)-adrenoceptor antagonists treatment. It is probably related to the mode of action of \( \alpha_1 \)-adrenoceptor antagonists. Functional studies and binding assays have shown that the \( \alpha_{1A} \)-adrenoceptor subtype is the predominant subtype in the bladder neck and vas deferens.\(^{23–25} \) In addition, \( \alpha_1 \)-adrenoceptors seem to be present in the seminal vesicles. Antagonism of these \( \alpha_1 \)-adrenoceptors will relax smooth muscle and may induce abnormal ejaculation.\(^{26} \) Abnormal ejaculation has been reported to occur in 4% to 11% of patients receiving an \( \alpha_1 \)-adrenoceptor an-
Several randomized, placebo-controlled trials with tamsulosin 0.4 mg once daily in patients with LUTS/BPH showed that abnormal ejaculation occurred in 4.5% (European studies) to 11% (US studies) of patients receiving tamsulosin versus 1% of patients receiving placebo (Table I). In addition, during open-label treatment with tamsulosin for 4 years, 4.9% of 516 patients with LUTS/BPH had abnormal ejaculation. In real-life practice, retrograde ejaculation has been reported by only 0.3% of 12,484 patients with LUTS/BPH treated with tamsulosin 0.4 mg once daily. During the 26th Congress of the Société Internationale D’Urologie (SIU) 2002, Debruyne et al. presented the results of a pooled analysis of 2 randomized, double-blind, placebo-controlled, 3-month European trials, a randomized double-blind, direct comparative 3-month European trial, and their open label extensions, which evaluated the impact of tamsulosin 0.4 mg once daily on sexual function. The incidence of abnormal ejaculation was 4.8% (34 of 702 patients with LUTS/BPH).

Direct comparative trials between tamsulosin and other $\alpha_1$-adrenoceptor antagonists have generally shown comparable rates of abnormal ejaculation. During a double-blind, randomized, direct comparative 14-week study of tamsulosin 0.4 mg once daily ($n = 132$) and alfuzosin 2.5 mg 3 times daily ($n = 124$), abnormal ejaculation occurred in only 1 patient in the tamsulosin group compared with 0 patients in the alfuzosin group ($n = 124$). A single-blind, randomized study compared tamsulosin 0.2 mg once daily with terazosin titrated from 1 to 5 mg once daily for 8 weeks in 98 Korean patients with LUTS/BPH. The efficacy of both treatments was comparable, and none of the patients in either treatment group had abnormal ejaculation. This was confirmed by another single-blind, direct comparative 4-week trial of tamsulosin 0.2 mg once daily and terazosin uptitrated from 1 to 2 mg in 212 Chinese patients with LUTS/BPH.
BPH, which showed comparable efficacy and no reports of abnormal ejaculation in either treatment group. However, in a US, open-label 8-week trial with tamsulosin 0.4 mg once daily (n = 1002) or terazosin uptitrated to 5 mg once daily (n = 981), ejaculation failure and ejaculation disorder were reported statistically significantly more often with tamsulosin than with terazosin.19

It should be noted that abnormal ejaculation is a very well-tolerated adverse event of #1-adrenoceptor antagonist treatment. In general, patients do not seem to consider retrograde ejaculation or a reduced ejaculate volume to be bothersome, which is confirmed by the fact that only a few patients discontinue treatment because of abnormal ejaculation. Placebo-controlled and open-label studies with tamsulosin show that 1% of patients treated with tamsulosin discontinued because of abnormal ejaculation versus 0% on placebo (Table I).26–29 The pooled analysis of several European trials showed only a 0.6% discontinuation rate because of abnormal ejaculation.31 Moreover, this adverse event is reversible upon drug withdrawal.

The data show that the incidence of abnormal ejaculation with #1-adrenoceptor antagonist treatment is low. It has been suggested that tamsulosin is associated with a higher incidence than other #1-adrenoceptor antagonists. However, most direct comparative trials report a comparable incidence.

**OVERALL SEXUAL FUNCTION**

Problems with sexual function, such as diminished libido and impotence, may become more prominent with aging. Consequently, many men with LUTS/BPH have concomitant sexual dysfunction. Furthermore, it seems that men with LUTS/BPH have an increased risk of reduced sexual function (Figure 6).26,38,39 A large study involving 2011 randomly selected French men showed that men...
with LUTS/BPH were more likely to be dissatisfied with their sex life than those without symptoms, and that the probability of sexual dissatisfaction increased with the severity of LUTS. Figure 6 demonstrates the percentage of men with sexual dysfunction by International Prostate Symptom Score (I-PSS). It shows that with increasing symptom severity, a larger percentage of men develop erectile dysfunction, low sexual drive, or sexual problems. Patients with LUTS/BPH considered sexual activities and satisfaction with sexual relationships a very important aspect of QOL, and sexual dysfunction, such as erectile dysfunction, is experienced as very bothersome. Therefore, LUTS/BPH can have a substantial negative impact on sexual function, reducing the patient’s QOL.

When looking at the effect of \( \alpha_1 \)-adrenoceptor antagonists on overall sexual function, it appears that they may have some beneficial effect. Höfner et al. assessed the effect of the \( \alpha_{1A}/\alpha_{1D}\)-adrenoceptor antagonist tamsulosin 0.4 mg once daily on sexual function. Sexual function was measured by means of a lifestyle questionnaire containing 3 questions on sexual function: “Has the condition affected your sex life with respect to (1) interest in sex, (2) morning erection/quality of erection, and (3) achieving orgasm/ejaculation?” Each of these 3 questions was rated on a scale from 0 to 4. The total sexual function score ranged from 0 to 12 points, with a decrease in score indicating an improvement in sexual function. A total of 575 patients with LUTS/BPH were randomized to tamsulosin 0.4 mg once daily treatment (n = 381) or placebo (n = 193) for 12 weeks. At 12 weeks, the total sexual function score had decreased by 0.31 points (baseline, 3.35 points) in the patients treated with tamsulosin. However, the placebo group showed no significant change in sexual function.

### Table I. Patients with abnormal ejaculation or who discontinued due to abnormal ejaculation in placebo-controlled and open-label trials with tamsulosin

<table>
<thead>
<tr>
<th>Study</th>
<th>Incidence of Abnormal Ejaculation</th>
<th>Discontinued Due to Abnormal Ejaculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Höfner et al. (1999)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (n = 193)</td>
<td>2 (1.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Tamsulosin 0.4 mg (n = 381)</td>
<td>17 (4.5)</td>
<td>3 (0.8)</td>
</tr>
<tr>
<td>Lepor (1998)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (n = 254)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Tamsulosin 0.4 mg (n = 254)</td>
<td>15 (6.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Narayan et al. (1998)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo (n = 239)</td>
<td>1 (0.4)</td>
<td>NA</td>
</tr>
<tr>
<td>Tamsulosin 0.4 mg (n = 248)</td>
<td>27 (11.0)</td>
<td>NA</td>
</tr>
<tr>
<td>Schulman et al. (2001)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tamsulosin (n = 515)</td>
<td>25 (4.9)</td>
<td>1 (0.2)</td>
</tr>
</tbody>
</table>

NA = not available.
tamsulosin (n = 236 responders at study completion), implying an overall improvement in sexual function. However, the score had increased by 0.49 points (baseline, 2.28 points) in the placebo group (n = 10 responders), suggesting an overall deterioration of sexual function. The positive change in sexual function score in the tamsulosin group was statistically significantly different from the negative change in score in the placebo group (P = 0.042). It should, however, be noted that the mean sexual function score at baseline was somewhat higher in the tamsulosin group, leaving more room for improvement.

The effect of tamsulosin 0.4 mg once daily (n = 105) versus alfuzosin 2.5 mg 3 times daily (n = 106) on sexual function was assessed in a 12-week, direct comparative study. Changes in the total sexual function score were comparable for patients receiving tamsulosin and alfuzosin (~0.14 points from a baseline of 2.87 points vs +0.15 points from a baseline of 2.60 points, respectively; P = 0.485). A real-life practice-based study in approximately 3000 patients with LUTS/BPH who visited a urologist’s office and who received tamsulosin for approximately 6 months also suggested that tamsulosin may improve sexual function.

In the opinion of the patients, tamsulosin tended to improve libido, erection, ejaculation, and overall sexual satisfaction. Similar positive effects on overall sexual function have been reported with other α1-adrenoceptor antagonists. The positive effect of α1-adrenoceptor antagonists, such as tamsulosin, on sexual function may be a result of the overall improvement in QOL associated with relief in LUTS/BPH because of treatment. It is likely that as LUTS become less bothersome, patients become less “disabled” by their urinary symptoms and are better able to enjoy other aspects of life without feeling inhibited or limited by their LUTS. In addition, it may also be a direct pharmacologic effect of α1-adrenoceptor antagonists in the penile arteries and/or the corpus cavernosum, resulting in relaxation of smooth muscle and subsequent increase in blood flow into the lacunar spaces of the corpores cavernosae.

In summary, patients with LUTS/BPH have an increased risk of impaired sexual function. α1-Adrenoceptor antagonists, such as tamsulosin 0.4 mg once daily, may improve overall sexual function and QOL.

COMBINATION THERAPY

Not only is monotherapy used in clinical practice (eg, α1-adrenoceptor antagonist or a 5α-reductase inhibitor, such as finasteride), but combination therapy of both agents is also used. Studies with a duration of ≤1 year have not demonstrated any additional benefit of combination therapy over the α1-adrenoceptor antagonist alone. The results of the recent long-term (≥6 years) Medical Therapy of Prostatic Symptoms (MTOPS) study, however, demonstrated additional benefit of the combination therapy in the longer term, particularly in high-risk patients (eg, patients with a high I-PSS, high prostate-specific antigen values, large prostate volume, high postvoid residual amounts, and/or low flow rate). Furthermore, it showed that combination therapy of an α1-adrenoceptor antagonist and finasteride is generally well tolerated (Table II). The combination results in a similar overall side-effect profile as monotherapy, except for the risk of abnormal ejaculation, which is increased compared with monotherapy. This corresponds to the results of other direct comparative 6- to 12-month studies. The results of these trials also show that the discontinuation rate from adverse events is comparable for α1-adrenoceptor antagonists and finasteride (Table III). Combination therapy seems to be associated with
an almost similar discontinuation rate from adverse events as is associated with monotherapy.

**CONCLUSIONS**

The objectives of treatment for LUTS/BPH are (1) to provide a rapid and sustained reduction of bothersome symptoms, (2) to improve QOL, and (3) to control disease progression. However, these beneficial effects should be balanced against the bother and risks resulting from adverse events or complications associated with treatment. Many patients with LUTS/BPH are reluctant to undergo surgical therapy because it is invasive and associated with irreversible complications, such as incontinence, impotence, and retrograde ejaculation. Since the 1990s, the rate of surgical therapies has decreased with a parallel increase in the number of medical prescriptions. α1-Adrenoceptor antagonists are the most frequently prescribed medical therapy, probably because they most effectively and rapidly relieve bothersome LUTS and improve QOL. In contrast to finasteride, they are not associated with impotence or decreased libido. Non-subtype-selective α1-adrenoceptor antagonists are, however, associated with blood pressure reductions and associated cardiovascular adverse events, such as dizziness, asthenia, somnolence, symptomatic orthostatic hypotension, and, potentially, syncope. In contrast, the subtype-selective α1A/α1D-adrenoceptor antagonist tamsulosin interferes only marginally with blood pressure regulation and has the lowest potential for inducing adverse events related to interference with blood pressure regulation. In addition, tamsulosin 0.4 g once daily appears to be as well tolerated as the plant extract *S. repens*. The only adverse event that was reported statistically significantly more often with tamsulosin was ejaculation disorder.

**TABLE II.** Most common adverse event reported with finasteride or a nonsubtype-selective α1-adrenoceptor (AR) antagonist in a direct comparative study

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Placebo (N = 737)</th>
<th>5α-Reductase Inhibitor Finasteride 5 mg qd (N = 768)</th>
<th>α1-AR Antagonist Doxazosin 4–8 mg qd (N = 756)</th>
<th>Combination (N = 786)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
<td>2.5</td>
<td>2.5</td>
<td>4.8*</td>
<td>5.9*</td>
</tr>
<tr>
<td>Asthenia</td>
<td>2.2</td>
<td>1.7</td>
<td>4.5*</td>
<td>4.6*</td>
</tr>
<tr>
<td>Somnolence</td>
<td>0.4</td>
<td>0.4</td>
<td>0.9*</td>
<td>0.9*</td>
</tr>
<tr>
<td>Postural hypotension</td>
<td>2.5</td>
<td>2.7</td>
<td>4.4</td>
<td>4.6*</td>
</tr>
<tr>
<td>Syncope</td>
<td>0.3</td>
<td>0.5</td>
<td>0.5</td>
<td>0.7*</td>
</tr>
<tr>
<td>Impotence</td>
<td>3.6</td>
<td>4.9*</td>
<td>3.9</td>
<td>5.6*</td>
</tr>
<tr>
<td>Decreased libido</td>
<td>1.5</td>
<td>2.5*</td>
<td>1.7</td>
<td>2.8*</td>
</tr>
<tr>
<td>Abnormal ejaculation</td>
<td>0.9</td>
<td>1.9*</td>
<td>1.2</td>
<td>3.4*</td>
</tr>
</tbody>
</table>

* P <0.05 vs placebo.
Adapted from J Urol.10

**TABLE III.** Percentage of patients discontinuing due to adverse events in 3 direct comparative 6- to 12-month studies among an α1-adrenoceptor (AR) antagonist, a 5α-reductase inhibitor (finasteride), and their combination

<table>
<thead>
<tr>
<th>Study</th>
<th>Placebo (N = 737)</th>
<th>5α-Reductase Inhibitor 5 mg qd (N = 768)</th>
<th>α1-AR Antagonist Doxazosin 4–8 mg qd (N = 756)</th>
<th>Combination (N = 786)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA Cooperative9*</td>
<td>1.6</td>
<td>4.8</td>
<td>5.9</td>
<td>7.8</td>
</tr>
<tr>
<td>PREDICT45†</td>
<td>11.1</td>
<td>12.9</td>
<td>11.6</td>
<td>12.2</td>
</tr>
<tr>
<td>ALFIN7‡</td>
<td>—</td>
<td>5.2</td>
<td>7.0</td>
<td>6.9</td>
</tr>
</tbody>
</table>

PREDICT = Prospective European Doxazosin and Combination Therapy trial; VA = Veterans Affairs.
* Terazosin.
† Doxazosin.
‡ Alfuzosin.
stop treatment. Although abnormal ejaculation has been reported for the $\alpha_1A/\alpha_1D$-adrenoceptor antagonist tamsulosin predominantly, the incidence in placebo-controlled trials seems to be comparable to that with other $\alpha_1$-adrenoceptor antagonists when they are directly compared. Moreover, $\alpha_1$-adrenoceptor antagonists, such as tamsulosin, appear to improve sexual function, which is very important because patients with LUTS/BPH appear to improve sexual function, which is very important because patients with LUTS/BPH are at increased risk of impaired sexual function. This may further improve the QOL of patients with LUTS/BPH.

Medical treatment can thus be considered a first-line treatment option for LUTS/BPH. It is noninvasive, and the adverse events are reversible, in contrast to the irreversible complications of surgery. Among the medical treatment options, $\alpha_1$-adrenoceptor antagonists, and particularly tamsulosin, are the most frequently used and have an excellent efficacy/tolerability ratio, which may possibly affect QOL.

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prostatic hyperplasia. BJU Int 90(suppl 2): 12, 2002. Abstract P-1.2.01.