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MEDICAL AFFAIRS

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Background on Overactive Bladder (OAB)

The overall prevalence of OAB symptoms in the United States among adults ≥40 years has been estimated to be 23%. Of that group, approximately 76% of women and 43% of men report being affected "often" or "sometimes." (Figure 1) Prevalence has been found to increase with age.[1] Despite this, there is a significant gap between the number of people suffering from this condition and those who ultimately seek medical advice or treatment.[2,3] Additionally, even with at-risk populations, medical professionals often do not proactively inquire about urinary symptoms.[2] Nonetheless, overactive bladder contributes greatly to social stigma, lower quality of life, and reduced productivity and engagement in life.[4] Thus, increasing awareness among patients and healthcare providers is critically important.

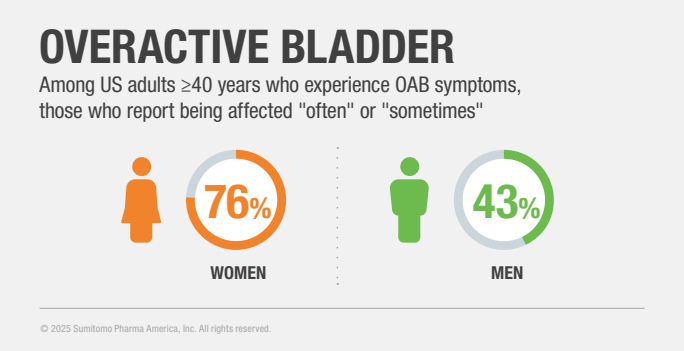


Figure 1. Adapted from Coyne, et al.[1]

It should be recognized that overactive bladder (OAB) is a syndrome consisting of typical symptoms as urinary urgency, increased daytime frequency and/or nocturia, with or without urgency urinary incontinence (UUI), in the absence of urinary tract infection (UTI) or other obvious pathology.[5,6] There are many medical diagnoses associated with overactive bladder symptoms, but OAB is fundamentally an idiopathic condition. In general, OAB occurs when the detrusor muscle experiences frequent involuntary contractions associated with increased activation of the parasympathetic nervous system. This is modulated by heightened stimulation of muscarinic neuronal receptors and subsequent acetylcholine leakage from the parasympathetic nerve terminal. This causes relative overactivity of the detrusor muscle and the accompanying urinary urgency sensation.[7]

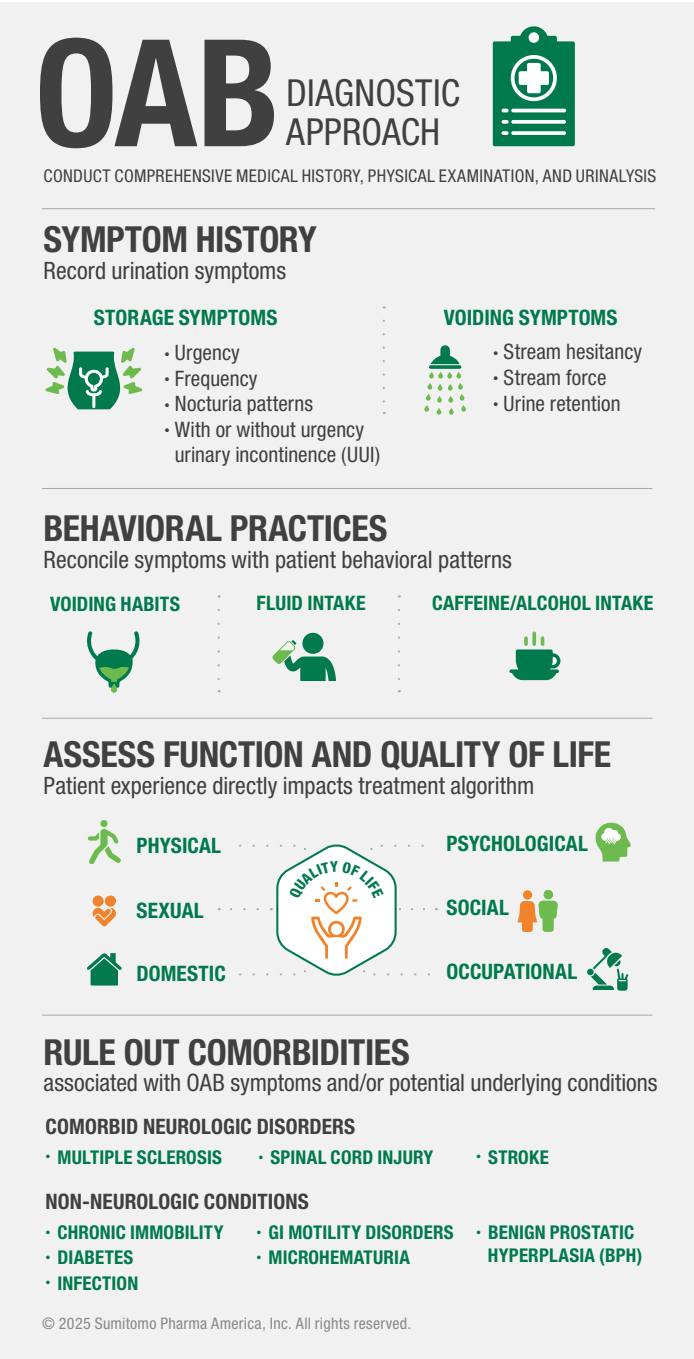
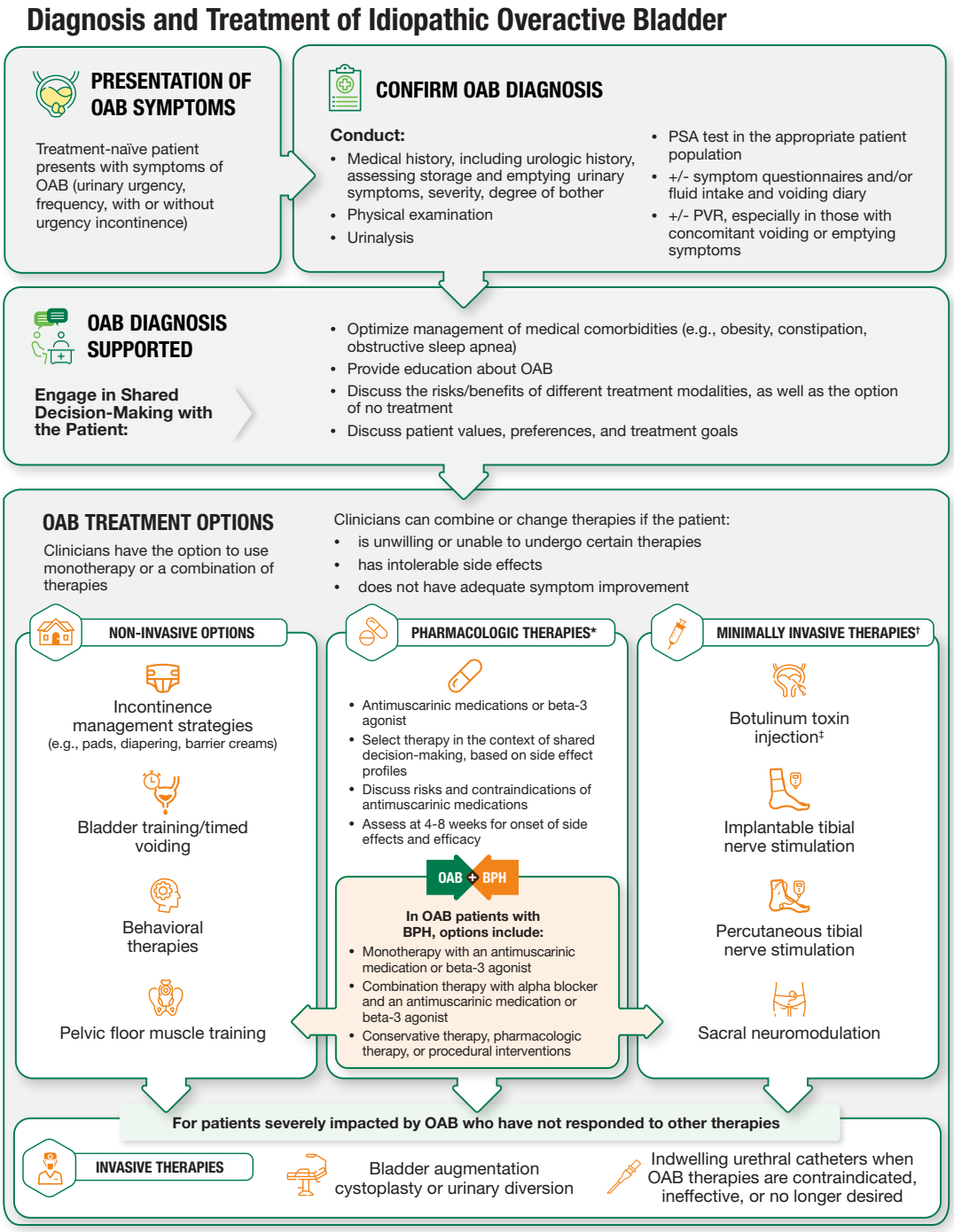


Figure 2. OAB diagnostic approach is adapted from AUA/SUFU Guideline on OAB.[6]

Although OAB symptoms are very common, the American Urological Association/Society of Urodynamics, Female Pelvic Medicine & Urogenital Reconstruction (AUA/SUFU) Diagnosis and Treatment of Idiopathic Overactive Bladder (OAB) Guideline recommends that a complete diagnostic approach be utilized to ensure that another treatable medical condition is not the cause of the patient's urinary symptoms.[6] This approach includes comprehensive medical history-taking, a physical examination, and urinalysis to exclude microhematuria and infection.[6] (Figure 2) The medical history should include questions about urine storage symptoms, including frequency, urgency, and nocturia patterns, and bladder-emptying symptoms, including stream hesitancy, stream force, and urine retention. Clinicians should reconcile these symptoms with the patient's behavioral practices, including voiding habits, daily fluid intake, and caffeine and alcohol intake.[6] This evaluation should also capture the degree to which the urinary symptoms impact the patient's daily functioning, productivity, and overall quality of life, especially given that many patients experiencing OAB symptoms may have delayed seeking medical advice.[6] Results from symptom questionnaires such as the Bristol Female Lower Urinary Tract Symptoms questionnaire,[8] Lower Urinary Tract Research Network's LUTS index (LURN-SI-29 or 10),[9] and others may be obtained at clinicians' discretion. The AUA/SUFU Guideline on OAB emphasizes the importance of evaluating patient-reported outcomes (PRO) in guiding both initial assessment and OAB symptom management. This comprehensive picture of the patient's experience will directly impact the subsequent treatment algorithm.[6]

Since idiopathic OAB symptoms are associated with other organic conditions, it is critical for clinicians to inquire about and be aware of comorbid neurologic disorders (e.g., stroke, spinal cord injury, multiple sclerosis) and any



BPH, benign prostatic hyperplasia; OAB, overactive bladder; PSA, prostate-specific antigen; PVR, post-void residual.

* If patient experiences intolerable side effects or inadequate symptom improvement, clinician can/may prescribe different medication of same or different class. If patient has inadequate symptom improvement with single medication, consider combination with medication of a different class.

† These therapies may be offered without trial of behavioral, non-invasive, or pharmacologic management. If patient is refractory to one treatment, clinician can try another. Consider a trial off of pharmacologic therapy after appropriate response has been achieved via minimally invasive therapies.

‡ Obtain PVR prior to injection, if not previously obtained.

Figure 3. Idiopathic OAB diagnosis and treatment options infographic is adapted from AUA/SUFU Guideline on OAB algorithm.[6]

non-neurologic conditions (e.g., obesity, constipation, chronic immobility, diabetes, GI motility disorders, obstructive sleep apnea, benign prostatic hyperplasia (BPH), pelvic or vaginal surgery, recurrent UTIs, or a history of pelvic radiation therapy). Clinicians should inform patients about how these can affect bladder symptoms.[6] A comprehensive physical examination, including abdominal, genitourinary, and rectal assessments, when appropriate, should be completed.[6] Additionally, a cognitive examination is advisable, since this can impact some of the behavioral and pharmacologic treatment choices, particularly in the elderly population.[6] The Mini-Mental State Examination (MMSE)[10] or the Montreal Cognitive Assessment (MoCA)[11,12] would be reasonable options for in-clinic cognitive examinations.

Depending on the patient specifics, additional diagnostic testing, such as urine culture and post-void residual (PVR) measurement, may be helpful to assess the cause of urinary symptoms.[6] Fluid intake and voiding diaries to be completed at home can also be useful to better understand the impact on a patient's lifestyle and behavior; these diaries can also play a role in the eventual treatment recommendations.[6]

The AUA/SUFU Guideline on OAB includes clear emphasis on clinician and patient engaging in shared decision-making, with consideration of the patient's expressed values, preferences, and treatment goals to ensure they can make an informed decision regarding the treatment option(s) they may select.[6]

In general, more advanced diagnostics, including urodynamic testing, cystoscopy, and ultrasound imaging are available to further assess bladder function and exclude other conditions when initial diagnosis is uncertain. These are not indicated for uncomplicated OAB symptoms, although this should be a shared decision between physician and patient.[6]

Non-Invasive Options

Behavioral and Lifestyle Approaches

If a treatable, primary cause of the patient's urinary symptoms is identified, then the primary treatment should focus on the management of that medical condition. If the patient is deemed to have idiopathic OAB, then the treatment algorithm may begin with behavioral and lifestyle techniques, possibly followed by pharmacologic options. The AUA/SUFU Guideline on OAB stresses that providers should always weigh the benefit-risk ratio for the various treatment options and should counsel patients about the expectations of the therapy for acceptable symptom control so that patients can make an informed decision.[6] Every treatment approach should be individualized. (Figure 4)

The least invasive treatment for idiopathic OAB targets behavioral techniques and lifestyle adjustments. For some patients, fluid intake management or restriction, a reduction in caffeine, and weight loss can be helpful. Daily fluid restrictions, coupled with reductions in caffeine intake (<100 mg a day), have successfully reduced urinary symptoms in some patients.[13] The AUA/SUFU Guideline on OAB also advises alcohol avoidance.[6]

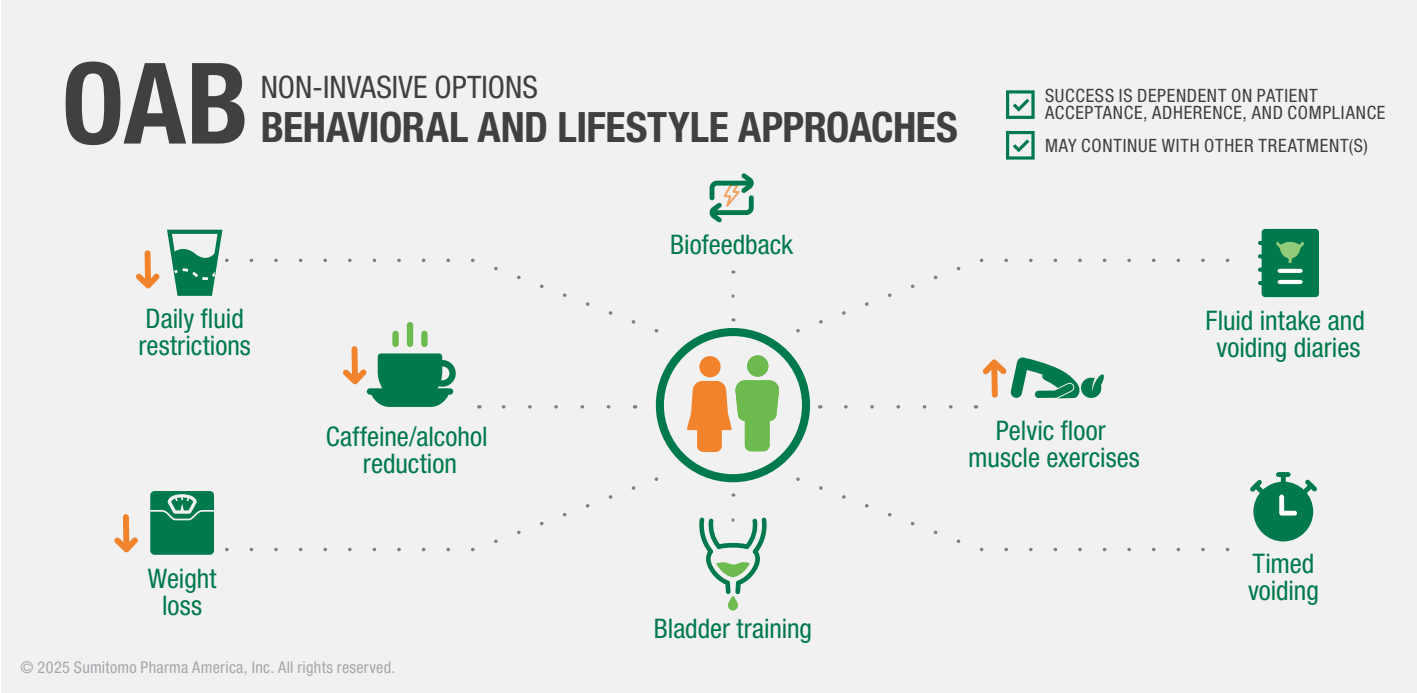


Figure 4. OAB non-invasive options are adapted from AUA/SUFU Guideline on OAB.[6]

Additionally, in a study of overweight/obese women by Subak et al., weight loss interventions over 6 months were shown to have positive impacts on reducing the weekly number of incontinence episodes, as found in the primary intervention study group, which achieved a mean 8% weight loss.[14] Fluid intake and voiding diaries can be an important tool in these approaches since daily insights can entrain enduring behaviors to improve urinary control. Additionally, fluid intake and voiding diaries allow for baseline metrics on fluid intake, caffeine and alcohol use, and weight to be established as comparators. Smoking cessation programs can also be beneficial in patients for whom smoking is an important variable.[6]

Concurrently, clinicians can recommend incontinence management strategies as needed for coping with urinary incontinence, such as diapers, pads, and barrier creams. While they are not a treatment, they can reduce adverse secondary effects, such as urine dermatitis.[6]

Non-invasive behavioral techniques for OAB symptom management include bladder training, pelvic floor muscle therapy, and biofeedback. Bladder training is a formal program that teaches timed voiding every 3-4 hours, along with the use of relaxation and distraction techniques to progressively delay voiding at the onset of urgency urinary sensations.[7] The goal of this technique is to maximally reestablish a patient's voluntary control over voiding, if possible. Pelvic floor muscle therapy focuses on physical exercises to improve the ability of the patient to voluntarily inhibit the contraction of the detrusor muscle.[7,15] Biofeedback approaches can also be assistive in this approach, using electrodes placed in the abdominal and pelvic region to help patients contract their pelvic floor muscles.[7] These approaches rely on robust patient participation and engagement, and are best used in combination. The AUA/SUFU Guideline on OAB thus suggests attempting a combination of behavioral and lifestyle approaches as non-invasive therapy options for patients with idiopathic OAB.[6] Note that these approaches can take 6-12 weeks to show an effect,[15] before considering pharmacologic options. It should also be noted that even if patients opt for pharmacologic therapy, this approach works best in conjunction with continued behavioral approaches. Multiple studies have shown that these behavioral techniques in combination with pharmacologic therapy lead to fewer incontinence episodes when compared with pharmacologic or single behavioral intervention alone.[16,17]

Pharmacologic Therapies

Antimuscarinic Medications

In addition to a range of behavioral and lifestyle interventions, there are multiple lines of pharmacologic treatments that can be used adjunctively or alone. Historically, antimuscarinic medications (a subgroup of the anticholinergic class) served as the primary options for most patients with OAB symptoms. These medications specifically act through the muscarinic pathway, inhibiting the involuntary detrusor muscle contractions that are felt to be a source of OAB symptoms. There are many antimuscarinic medications available (including trospium, darifenacin, fesoterodine, solifenacin, tolterodine, and oxybutynin), although oxybutynin and tolterodine are the most used in practice in the United States.[7] This category of therapy has been studied in many randomized controlled trials, with moderate efficacy demonstrated in terms of reduced episodes of UUI and voids.[18] Analyzed as a class, anticholinergic therapies have been shown to reduce UUI by 1.73 episodes per day and urinary voids by 2.06 episodes per day, from 2.79 and 11.28 at baseline, respectively. Placebo arms showed lesser reductions of daily UUI by 1.06 and void episodes by 1.2 per

OAB PHARMACOLOGIC THERAPIES

OXYBUTYNIN CHLORIDE

MECHANISM OF ACTION
Inhibition of the acetylcholinergic muscarinic receptor (non-selective blockade will have off-target effects on GI tract, salivary glands, eyes, and brain)

STUDY DESIGN
Multiple RCT studies, placebo- and active-controlled, showed improvements in urge incontinence, urgency, and frequency

ROUTE OF ADMINISTRATION
Oral tablet, oral tablet extended release, transdermal gel, transdermal patch, oral syrup

SIDE EFFECTS
≥5%: Dry mouth, constipation, diarrhea, dizziness, headache, somnolence

>1%: Abdominal pain, flatulence, vomiting, dysuria, fatigue, insomnia, dry eye, blurred vision, cough, dry nose, dry throat

CONTRAINDICATIONS
Urinary retention, gastric retention, uncontrolled narrow-angle glaucoma, prior hypersensitivity to oxybutynin chloride

WARNINGS
Angioedema, CNS effects, pre-existing dementia in patients treated with cholinesterase inhibitors, Parkinson's disease, myasthenia gravis, decreased gastrointestinal motility with autonomic neuropathy, urinary retention, gastric retention

Table 1. Information shown is abbreviated; please refer to full prescribing information[19] for complete details.

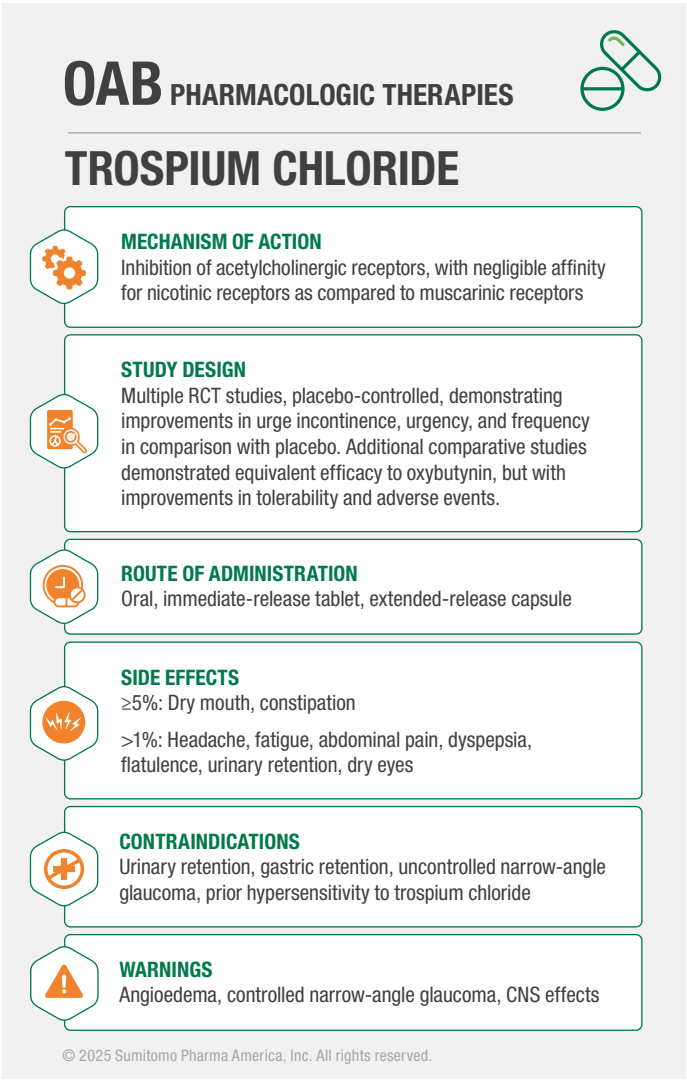


Table 2. Information shown is abbreviated; please refer to full prescribing information[20] for complete details.

day.[18] Studies have also demonstrated no consistent differences in efficacy across the various individual antimuscarinic medications in the anticholinergic class.[21,22] One of the most commonly used medications in this class, oxybutynin (Table 1), is available in multiple formulations, including extended-release and transdermal preparations to improve compliance. Newer medications in this class, such as trospium (Table 2), may have lesser side effects than the older agents, while retaining the same degree of effectiveness. Some of these medications (trospium, fesoterodine, darifenacin) are believed to have less blood-brain barrier penetration, thus potentially reducing the impact on cognitive side effects.[6] These newer medications also come in multiple formulations, including extended-release versions.

Despite their class efficacy, these medications have well-known and undesirable side effects due to their broad anticholinergic action; this includes dry mouth and constipation, which often limit patient satisfaction with the therapy. In comparative studies, oxybutynin has been found to have statistically significant higher rates of dry mouth and constipation in comparison with tolterodine.[6] However, all the medications in this class possess high self-reported rates of dry mouth (25%-44%) and constipation (8%-15%).[6] It should be noted that the extended-release versions (oxybutynin and tolterodine) and transdermal preparations (oxybutynin) appear to have slightly lower rates of these side effects while maintaining the same efficacy.[6,23]

In addition to the typical anticholinergic side effects, it is important to recognize that this medication class has also been increasingly associated with an increased risk of cognitive impairment, particularly in the aging population.[24,25,26] This is especially salient since this population of patients is at heightened risk for OAB symptoms and commonly takes medications that may have interactions with anticholinergic agents.[24,27] Additionally, several case-control and cohort studies have found a heightened risk of a dementia diagnosis in patients who have taken medications from the anticholinergic class.[28,29,30,31,32] Although these studies do not establish a causative relationship, this body of evidence does raise caution regarding the use of these medications in patients at risk for cognitive or memory impairment. Although not entirely contraindicated, caution is warranted when using muscarinic antagonists (anticholinergics) in the elderly population, and thus CNS effects should be monitored in any patient starting or increasing the dose of one of these types of medication.

Beta-3 Adrenergic Agonist Medications

In contrast with the older anticholinergic class of medications, 2 more recent therapies have been approved by the FDA for the treatment of OAB symptoms that act as beta-3 adrenergic receptor agonists. These medications (mirabegron and vibegron) act by actively relaxing the detrusor smooth muscle during the storage phase of the bladder, thereby increasing fill capacity.[33] Both medications appear to have superior efficacy compared with placebo,[6] and both have low potential for crossing the blood-brain barrier.[34,35] The first of these medications, mirabegron (Table 3), was approved by the FDA in 2012, and is indicated as monotherapy for OAB symptoms or in combination with the antimuscarinic medication solifenacin succinate (refer to mirabegron label[36]). In a study by Herschorn et al., 2 strengths of mirabegron were used (25 mg or 50 mg daily) in comparison with placebo over 12 weeks. The mirabegron treatment arms decreased daily incontinence episodes by 1.36 (25 mg) and 1.38 (50 mg) compared to 0.96 in the placebo group, and daily micturitions decreased by 1.65 (25 mg) and 1.60 (50 mg) in the treatment arms vs only 1.18 for the placebo arm, with both active arms reaching statistical significance.[37]

The mirabegron trials were associated with treatment-emergent side effects. A study by Chapple et al. showed lower rates of

OAB PHARMACOLOGIC THERAPIES

MIRABEGRON

MECHANISM OF ACTION
Activation of the beta-3 adrenergic receptor (more selective to the bladder)

STUDY DESIGN
Multiple RCT 12-week studies (placebo- or placebo- and active-controlled) and 52-week study (active-controlled) showing improvements in incontinence episodes and micturitions per day

ROUTE OF ADMINISTRATION
Oral tablet extended-release, oral suspension extended-release

SIDE EFFECTS
≥5%: Hypertension

>1%: Tachycardia, abdominal pain, constipation, diarrhea, dry mouth, cystitis, urinary tract infection, dizziness, headache, nasopharyngitis, sinusitis, fatigue

CONTRAINDICATIONS
Prior hypersensitivity to mirabegron

WARNINGS
Increases in blood pressure, urinary retention, angioedema, co-administration with medications metabolized by CYP2D6

Table 3. Information shown is abbreviated; please refer to full prescribing information[36] for complete details.

dry mouth vs tolterodine.[38] Hypertension is one of the more common adverse events (AEs) associated with mirabegron as compared with placebo (11.3% [25 mg] and 7.5% [50 mg] vs 7.6% [placebo]); currently, mirabegron does carry a warning and is not recommended for patients with severe uncontrolled hypertension. The most common AEs in the mirabegron trials were hypertension, nasopharyngitis, urinary tract infection, and headache (all >2% and greater than the placebo arms).[36]

A second selective beta-3 adrenergic agonist, vibegron (Table 4), was approved by the FDA in 2020 for the treatment of OAB symptoms, based on data from multiple randomized controlled trials. In 2024, an expanded indication for vibegron was approved for the treatment of OAB symptoms in adult males receiving pharmacological therapy for benign prostatic hyperplasia (BPH) (Table 4). A pivotal 12-week treatment trial vs placebo demonstrated statistically significant improvements in both daily voids ($p< 0.001$) and urge incontinence episodes ($p< 0.0001$) at 12 weeks.[39] A subsequent phase 3 study of once-daily vibegron vs active control was completed with a primary

OAB PHARMACOLOGIC THERAPIES

VIBEGRON

MECHANISM OF ACTION
Activation of the beta-3 adrenergic receptor (more selective to the bladder)

STUDY DESIGN
RCT 12-week study (placebo- and active-controlled) and 52-week extension study (active-controlled) showing improvements in incontinence episodes, urgency, and frequency

RCT 24-week, double-blind, placebo-controlled study in male patients with OAB on pharmacological therapy for benign prostatic hyperplasia (BPH)

ROUTE OF ADMINISTRATION
Oral tablet

SIDE EFFECTS
≥5%: Hypertension*
*Reported in male patients with OAB on pharmacological therapy for BPH

>1%: Headache, nasopharyngitis, diarrhea, nausea, upper respiratory tract infection, dry mouth, constipation, urinary retention, hot flush; urinary tract infection

CONTRAINDICATIONS
Prior hypersensitivity to vibegron

WARNINGS
Urinary retention, angioedema

Table 4. Information shown is abbreviated; please refer to full prescribing information[40] for complete details. For additional safety information, please click here for full prescribing information.

outcome focused on safety, as measured by the incidence of AEs. No clinically meaningful differences in AEs were found between the treatment groups in the study. This 52-week extension analysis also demonstrated greater improvements in urinary incontinence episodes, urinary episodes, and total incontinence episodes, which were secondary endpoints in the study. In comparing the 2 treatment arms, 61% of patients on vibegron experienced a ≥75% reduction in urge urinary incontinence episodes vs 54% for patients on active control.[41] Additionally, in the 24-week, phase 3 COURAGE trial evaluating vibegron 75 mg in men with OAB symptoms who were being pharmacologically treated for BPH, vibegron was associated with significant reductions in mean daily micturitions and urgency episodes when compared to placebo at week 12.[42]

The primary AEs reported for vibegron vs placebo were headache (4.0% vs 2.4%), nasopharyngitis (2.8% vs 1.7%),

diarrhea (2.2% vs 1.1%), and nausea (2.2% vs 1.1%). The incidence of hypertension in the primary vibegron trial at 12 weeks was equal to that of placebo (1.7% vs 1.7%).[39] In the 52-week extension study for vibegron, the incidence of hypertension was 8.8% but this was comparable to the group of patients taking active control (8.6%). At the end of the trial, the mean changes in blood pressure were not statistically different from baseline in the treatment cohort.[41] In the 24-week COURAGE trial of men with OAB and pharmacologically treated BPH, the incidence of the treatment-emergent AE of hypertension was comparable in the vibegron and placebo arms (9.0% vs 8.3%, respectively).[42]

For patients on a single OAB medication who experience side effects that are intolerable or without adequate improvement, clinicians may offer a different medication in the same class or different class for greater tolerability and/or efficacy.[6]

Medication Combinations

If individual medications are not fully effective and the patient desires an alternative approach, combinations of antimuscarinics and beta-3 adrenergic agonists can also be attempted.[6] In particular, the combination of solifenacin and mirabegron has been studied in comparison with placebo, mirabegron in monotherapy, and solifenacin in monotherapy.[43,44,45] These studies demonstrated stronger effect sizes for the combination arms in terms of both UUI and voiding episodes. Perhaps as expected, however, the combination therapies also exhibited a higher rate of AEs (49%), including dry mouth and constipation, as compared with either of the monotherapy arms (41% for mirabegron and 44% for solifenacin).[45] Despite these findings, for a motivated patient with difficult-to-treat symptoms, a combination approach may be warranted.

It is again noted that if antimuscarinic medications are included, clinicians should consider either extended-release or transdermal formulations, which have been shown to carry fewer side effects with the same degree of clinical effectiveness.[6,23]

Special Considerations

As mentioned earlier, given the potential for impact on cognitive functioning, antimuscarinic treatment options should be used cautiously or avoided in the elderly, patients with pre-existing cognitive impairment, and patients who are taking other medications with anticholinergic properties. Even though a trial of an antimuscarinic medication is recommended through the AUA/SUFU Guideline on OAB, a decision should be made between the clinician, patient, and caregivers with consideration of the benefit-risk balance.[6] If an antimuscarinic trial is pursued in these patient populations, a low dose should be employed first, with regular monitoring of cognitive functioning and titrating according to efficacy and side effects.[2]

If symptoms do not respond adequately to monotherapy, clinicians may offer to combine one or more therapies (i.e., non-invasive, pharmacologic, minimally invasive). Clinicians should provide counsel on side effects of all oral medications. Caution should be exercised when prescribing antimuscarinics for patients with glaucoma, impaired gastric emptying, or history of urinary retention. Assessment of pharmacologic therapy efficacy and onset of side effects should be ongoing.[6]

Additionally, for all patient populations, it must be recognized that commitment to a long-term treatment approach is a critical element of success. For both behavioral and pharmacologic techniques, it requires habit-forming and patience to make incremental improvements and improve health outcomes.[2,46] Unfortunately, for many patients with OAB symptoms, maintaining a persistent therapy is often challenging. Unmet treatment expectations, medication switches, and side effects are some of the key reasons behind this.[47] Therefore, clinicians should always ensure that patients have a realistic expectation and plan for their long-term treatment approach to OAB symptoms.

Minimally Invasive Therapies

Botulinum Toxin and Neurostimulatory Approaches

The AUA/SUFU Guideline on OAB states that clinicians may offer minimally invasive therapies if the patient with OAB is unable or unwilling to engage in behavioral, non-invasive, or pharmacologic management options, or if shared physician-patient decision-making leads to selection of this treatment option first.[6] For selected patients who have intractable symptoms and have not responded to other therapeutic options, onabotulinumtoxinA injections (Table 5) have been approved by the FDA and can be considered. The AUA/SUFU Guideline on OAB recommends measuring post-void residual (PVR) prior to these injections, if not previously obtained, as well as post-injection should symptoms not improve or worsen.[6] Botulinum toxin works by inhibiting the release of acetylcholine from presynaptic nerve terminals, thus preventing bladder detrusor muscle activity. This procedurally involves direct intradetrusor bladder injections performed under local anesthesia via cystoscopy.[48] In comparative assessments with antimuscarinics and mirabegron, onabotulinumtoxinA injections were also shown to decrease the number of daily incontinence and urgency urinary episodes.[49] Placebo-controlled studies of

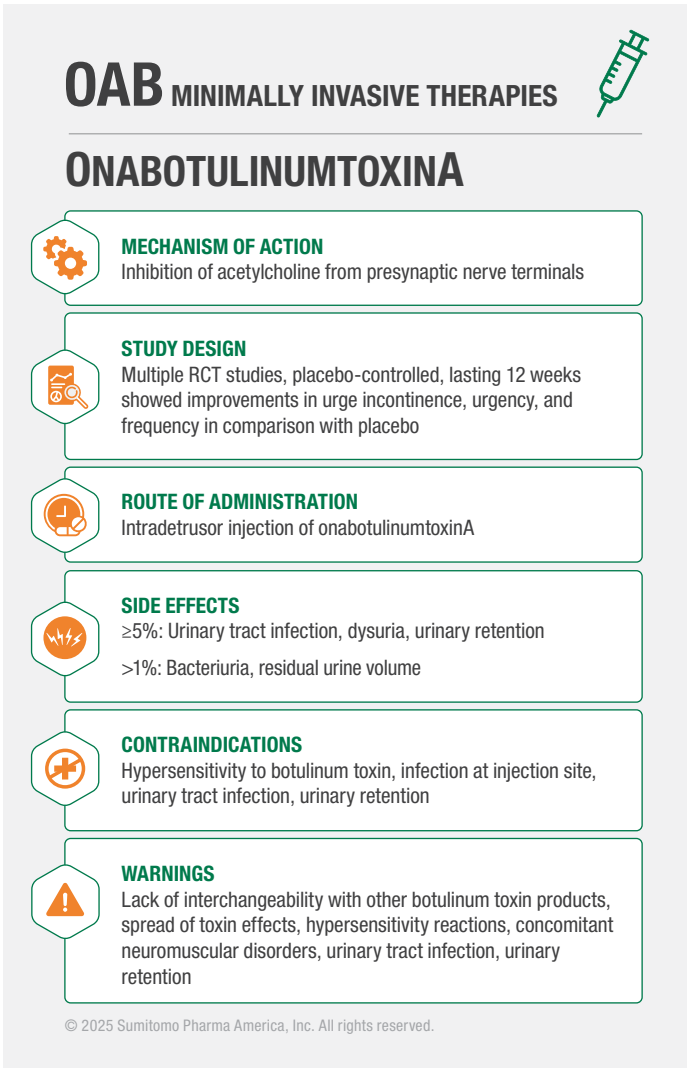


Table 5. Information shown is abbreviated; please refer to full prescribing information[50] for complete details.

botulinum toxin injections also demonstrated a benefit on the number of daily incontinence episodes.[51] However, long-term follow studies on the use of botulinum toxin injections show a high rate of discontinuation, primarily due to intolerability of the procedure (43%) and lack of sufficient efficacy (27%).[48]

Neurostimulation therapy has also been studied as a potential option for some patients with intractable OAB symptoms. Percutaneous tibial nerve stimulation (PTNS) and transcutaneous tibial nerve stimulation (TTNS) are 2 approaches based on the concept that peripheral nerve stimulation may impact bladder functioning. PTNS involves an office-based procedure in which a small needle is used to administer a mild electrical stimulation in the area of the posterior tibial nerve (behind the ankle) for 30 minutes, usually with 12 weeks of sessions. This procedure has been shown in systematic reviews to improve symptoms in some patients against placebo,[6] although head-to-head trials in comparison with traditional antimuscarinic medications did not show evidence of superiority.[52] Sacral neuromodulation is another FDA-approved procedure in which a small neurostimulator is surgically implanted after testing the patient for candidacy.[6] Studies have demonstrated that between 56% and 68% of intractable patients have at least a 50% improvement in urinary incontinence episodes and urinary frequency[53,54]; however, in a study of 272 patients, 13% required surgical intervention, with the majority being revision or replacement of the device.[55] Thus, this therapy should only be considered for refractory patients with continuing symptoms that severely impact quality of life.

If patients with OAB symptoms improve in response to a minimally invasive procedure, clinicians should discontinue oral medications but may restart pharmacologic therapy if efficacy is not maintained.[6]

Invasive Therapies

Surgical Approaches

The final category of therapies for patients with intractable OAB symptoms involves primary surgical solutions. It is stressed that these approaches are only for rare refractory and complicated patients who have attempted many prior types of treatment. Clinicians may offer bladder augmentation cystoplasty, in which a segment of the bowel is added to the bladder to make it larger.[56] Urinary diversion is another surgical procedure in which an alternative pathway for urine release is created due to blockage of the body's natural anatomy.[57] Lastly, indwelling (urethral or suprapubic) catheters are an option when other OAB therapies are contraindicated or ineffective.[6]

In summary, physicians managing the complexities of OAB symptoms should consult the AUA/SUFU Guideline on OAB, which advocates for a collaborative, patient-centered approach to shared decision-making, ensuring treatment choices align with each patient's values, preferences, and goals.[6]

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