

A SR performed by the Panel of studies on alpha-blockers used specifically for women with BOO included one placebo-controlled RCT, one RCT comparing two types of alpha-blockers, and six prospective non-comparative studies [148]. In the only placebo-controlled RCT reporting subgroup analyses in women with urodynamically proven BOO (based on the Blaivas-Groutz nomogram) no significant difference was observed in the changes of IPSS, IPSS sub scores, $Q_{\max}$, PVR volume and bladder diary after eight weeks of alfuzosin ($n = 58$) vs. placebo ($n = 59$). Of note, no EMG and/or voiding cysto-urethrography was used to distinguish between dysfunctional voiding and primary bladder neck obstruction [567].

Information on the comparative effectiveness of the different types of alpha-blockers was limited to one RCT. A small trial of 37 women with IPSS > 8, $Q_{\max} < 12$ mL/s and PVR volume > 50 mL, compared tamsulosin and prazosin over a three-month treatment period. More patients treated with tamsulosin were completely satisfied with their treatment (16/20 vs. 9/20). Both treatment groups showed significant improvement in symptom scores from baseline but no further statistical comparison between the groups was done. However, a larger decrease in AUA symptom score was seen in the tamsulosin group compared with the prazosin group. More adverse events were reported with prazosin (thirteen vs. one case) [568].

4.5.4.2.2 Striated muscle relaxants

Baclofen is a gamma-aminobutyric acid (GABA) agonist that exerts its effect on the GABAergic interneurons in the sacral inter-mediolateral cell column responsible for the relaxation of the striated urethral sphincter during voiding.

A randomised placebo-controlled crossover trial investigated the efficacy and safety of a four-week course of oral baclofen 10 mg three times/day in 60 women diagnosed with BOO, based on increased EMG activity with sustained detrusor contraction during voiding. It showed a lower number of voids, significant improvements in $Q_{\max}$ and $P_{\det}Q_{\max}$ with baclofen compared with placebo. Post-void residual volume, maximum cystometric capacity and MUCP were not significantly different between the groups. Adverse event rates were also similar, with the most common being somnolence, dizziness, and nausea. An important limitation of this study was the lack of patient-reported outcomes to assess symptoms and QoL [569].

4.5.4.2.3 Oestrogens

The relative reduction in urethral wall compliance seen in atrophic urethritis due to oestrogen deprivation may be responsible for urethral obstruction in post-menopausal women. Oestrogen therapy is thus theoretically expected to improve the condition. There are no published studies on the use of oestrogens specifically for the management of female BOO.

4.5.4.2.4 Sildenafil

Sildenafil, by inhibiting phosphodiesterase five, might be expected to increase the levels of nitric oxide in the female urethral sphincter, thereby promoting urethral relaxation.

A placebo-controlled, randomised crossover trial that included twenty women with partial or complete retention or obstructive voiding, with high MUCP and elevated US-estimated sphincter volume (> 1.6 cm) showed that while there was significant improvement in symptom scores and urodynamic parameters from baseline with sildenafil treatment, this difference was not significant when compared with placebo [570].

4.5.4.2.5 Thyrotropin-releasing hormone

Intravenous thyrotropin-releasing hormone (TRH) has been postulated as a neurotransmitter that induces urethral relaxation [571]. The exact mechanism is unclear.

In a small RCT of sixteen women with voiding difficulty, eight (three with BOO) were randomised to receive 200 µg intravenous bolus of TRH, and eight (three with BOO) received saline. No difference in the decline in functional profile lengths and maximum urethral closure pressures were noted between treatment groups, despite a significant decline noted from baseline in the treatment group. No subgroup analysis of women with BOO was reported [571].