Hypothesis / aims of study
Urethral sphincter injection of BoNT-A can reduce urethral resistance and facilitate spontaneous voiding in patients with low detrusor contractility. If the low detrusor contractility is induced by a poor relaxation of urethral sphincter, treatment of this underlying cause might reduce the inhibitory effect of detrusor contractility resulting in better detrusor function. This study investigated the effect of urethral injection of BoNT-A on idiopathic low detrusor contractility and its relation to baseline urodynamic characteristics.

Study design, materials and methods
Twenty-seven patients with idiopathic low detrusor contractility received urethral injection of BoNT-A. Videourodynamic studies were performed at baseline and after treatment. Recovery of detrusor contractility was defined as an increase of detrusor pressure and maximum flow rate and reduced postvoid residual urine. The therapeutic results and changes of urodynamic parameters were compared between patients with and without recovery of detrusor contractility.

Results
Recovery of detrusor contractility after urethral BoNT-A injection occurred in 13 (48%) of the patients. Patients with recovery of detrusor contractility had baseline data characterized by normal bladder sensation during bladder filling combined with a poor relaxation or hyperactive urethral sphincter activity. In contrast, patients without recovery of detrusor contractility had poor bladder sensation and a non-relaxing urethral sphincter. A significant reduction of PVR and a significant increase of Qmax were noted in both groups. Patients without recovery of detrusor contractility had significantly reduced voiding pressure of Pves. By contrast, Pdet significantly increased in patients with recovery of detrusor contractility. Among the 13 patients with recovery of detrusor contractility, 10 had an excellent therapeutic result and 3 had an improved result. Among the 14 patients without recovery of detrusor contractility, 1 had an excellent result, 10 had an improved result and 3 had a failed result (p<0.001). The overall success rate of the treatment was 89%. Patients with recovery of detrusor contractility could all void without the aid of abdominal straining but all patients without recovery of detrusor contractility continued to void with increased abdominal pressure. The maximum effect of urethral BoNT-A injection lasted from 5 months to more than 24 months. Five patients with recovery of detrusor contractility after urethral BoNT-A injection had effects which lasted for more than 12 months without relapse of difficult urination. However, the therapeutic duration was less than 8 months in all patients without recovery of detrusor contractility. Patients with recovery of detrusor contractility had a significantly longer duration of therapeutic effect than those without recovery (14±7.6 vs 4.6±2.5 months, p<0.001).

Interpretation of results
Voiding dysfunction may be caused by true detrusor underactivity or urethral hyperactivity. This study showed that patients with low detrusor contractility and urethral sphincter hyperactivity had a higher rate of recovery of detrusor contractility after urethral BoNT-A treatment, suggesting that a hyperactive urethral sphincter might be the cause of low detrusor contractility. Patients with low detrusor contractility due to urethral sphincter hyperactivity had a longer duration of therapeutic effect without repeat BoNT-A injections. By comparison, fewer patients with poor bladder sensation and true detrusor underactivity, had recovery of detrusor contractility.

Concluding message
Patients with detrusor underactivity with normal bladder sensation combined with a poor relaxation or hyperactive urethral sphincter were significantly more likely to recover normal detrusor function.

FUNDING:  NONE
DISCLOSURES:  NONE
HUMAN SUBJECTS:  This study was approved by the IRB of Buddhist Tzu Chi General Hospital, Taiwan and followed the Declaration of Helsinki. Informed consent was obtained from the patients.