Introduction
The two primary functions of the lower urinary tract are storage of urine at low pressure and voiding of urine. These functions are performed by the synergistic action of two muscular structures, the urinary bladder and the urethra, which are neurally controlled by the sacral nerves. The latter are influenced by higher centers, particularly the pontine micturition center, whereas the suprapontine influences the pontine micturition center to switch between storage and voiding [1]. Neurologic disturbances inflicted on one or more of the suprapontine structures (eg, the frontal lobe cortex, cerebellum, and the basal nuclei) result in voiding dysfunction, leading to neurogenic bladder.

Neurogenic Bladder Dysfunction
Patients with injury or disease of upper motor neurons may present with neurogenic detrusor overactivity (NDO) or detrusor–sphincter dyssynergia (contraction of the detrusor and urethral sphincter simultaneously rather than reciprocally). The term neurogenic detrusor overactivity is defined as involuntary detrusor contractions during the filling phase of relatively low bladder volume [2]. The behavior of voiding symptoms depends on the level and nature of the lesion and may explain individual variation in urinary symptoms and degree of reported urinary incontinence.

Causes of neurogenic bladder dysfunction include spinal cord injury or trauma; central nervous system tumors; demyelinating disease involving the spinal cord; spinal cord ischemia; cerebral palsy; and myelomeningocele, the most common cause of neurogenic bladder dysfunction in children.

Another study restricted definition of patients with neurogenic bladder dysfunction to those with positively diagnosed neurogenic pathology (one with benign tumors of medulla oblongata, cerebral infarcts, encephalitis disseminata, disc prolapse, polyneuropathy, or after spinal operations [3].

Regardless of the underlying pathophysiology, neurogenic bladder dysfunction often leads to progressive bladder and upper urinary tract deterioration.

Management of Neurogenic Bladder Dysfunction
The primary goal of NDO management is to preserve renal function by establishing low pressure storage and preventing recurrent urinary tract infections. The secondary goal is to improve storage capacity, resulting in better quality of life.

Management options for NDO include pelvic floor training and biofeedback. Pharmacotherapy includes anticholinergic medications, such as tolterodine or oxybutynin, as an initial line of treatment. Clean, intermittent self-catheterization as a conservative modality of treatment options with or without other forms of therapies constituted a breakthrough in the management of voiding dysfunction. However, major surgical procedures (eg, bladder augmentation, urinary diversion, and bladder neck reconstruction) are considered last resorts, as they potentially lead to serious side effects.
Between the two extreme limbs of therapy options (conservative vs major surgical procedures), different modalities of electrical neuromodulation are well-tolerated, effective, less invasive, and relatively reversible procedures to treat patients with neurogenic bladder dysfunction.

**Historical Overview of Electrical Neuromodulation for Voiding Dysfunction**

The concept of neuromodulation or nerve stimulation to treat voiding dysfunction emerged in 1811 when Bell first conducted experiments on the spinal nerve roots. He concluded that anterior nerve roots conduct sensory and motor impulses, whereas posterior nerve roots account for the vital functions [4]. In 1863, Giannuzzi [5] stimulated the spinal cord in dogs and concluded that the hypogastric and pelvic nerves are involved in regulating the bladder function. In 1976, Brindley et al. [6] began implanting sacral anterior root stimulators in paraplegic patients with incontinence. He presented his experience in 1986 with the first 50 patients. Thirty patients were completely continent and five were continent at night. Forty-three patients regularly used their implants for micturition and 26 of 38 male patients achieved erection under stimulation [6].

The pioneering work by Tanagho and Schmidt [7] in 1982 resulted in the design of a spiral electrode to minimize nerve damage. They presented the first result of sacral root stimulation in paraplegic dogs. Then, in 1988, Schmidt [8] described the three stages of electrode placement, starting with placement of a needle for test stimulation into the sacral foramina near the sacral roots. If a patient qualified, the needle was replaced by a temporary wire for percutaneous stimulation. A permanent neural implant was implanted if the patient experienced more than 50% improvement in urinary incontinence. The electrode was inserted unilaterally at either S3 foramina level or at the level of the pudendal nerves [8].

In 1990, Tanagho [9] presented results of neurostimulation for incontinence. Results showed 50% or more subjective improvement in 70% of patients with urge incontinence and in 40% of 25 patients after prostatectomy incontinence. In 1992, Tanagho [10] also published results of neuromodulation in 27 children. In this study, five of seven patients with meningomyelocele achieved continence.

In 1998, Shaker and Hassouna [11] published the result of sacral root neuromodulation in 20 patients with idiopathic nonobstructive chronic urinary retention. Results showed 50% improvement in voided volume and postvoid residual volume. They concluded that sacral root neuromodulation is an appealing, efficacious treatment for patients with idiopathic nonobstructive retention.

**Types of Electrical Neuromodulation for Neurogenic Voiding Dysfunction**

**Finetech-Brindley bladder system**

The Finetech-Brindley bladder system (Finetech Medical, Hertfordshire, United Kingdom), also known as Vocare bladder system, was developed from experiments on baboons from 1969 to 1977. As mentioned, the first device was implanted in 1976, but no useful micturition was obtained. Another six devices were implanted in 1978 and 1979 and were successful, with two still in use in 2002 [12]. In 1999, the US Food and Drug Administration (FDA) approved the device to treat neurogenic bladder secondary to spinal cord injury (through a Humanitarian Device Exemption; randomized clinical trials were not required for approval) [13]. Nearly 3000 implants have since been performed in patients with spinal lesions.

**Mechanism of action**

The system enables micturition by inducing detrusor contraction by implantation of the electrodes on the ventral branch of the sacral nerve roots (S2–S4/5). The ventral branch of the sacral nerve roots contains most of the efferent fibers that innervate several muscles of the pelvic floor, legs, and the anal and urethral sphincter. So, in addition to detrusor muscle contraction, electrical stimulation of these nerve roots leads to contraction of the urethral sphincter, thus complicating voiding and causing lower limb movements. To overcome this problem and enable voiding, the Finetech-Brindley bladder system uses the poststimulus voiding principle. Poststimulus voiding takes advantage of the difference in biomechanical characteristics of smooth and striated muscle tissue. The relaxation time of the smooth detrusor muscle is longer than that of the external urethral sphincter striated muscle. Thus, when stimulating with interrupted pulse trains (usually 3–6 seconds on, 6–9 seconds off), voiding can be achieved between the pulse trains due to the sustained high intravesical pressure [14].

**Patient selection**

Any patient with stable supra-sacral spinal cord lesion (paraplegia, tetraplegia) with reflex bladder (detrusor sphincter dyssynergia and incontinence) resistant to pharmacotherapy can benefit from the Brindley technique [15]. It is not recommended for patients with incomplete spinal cord injury, as they show poor tolerance to the stimulation, or male patients who desire to maintain their erectile function, as dorsal rhizotomy carries the risk of causing erectile dysfunction.

**The procedure**

The procedure involves implantation of three components: the tripolar electrodes, cables, and a pulse generator. The external component composed of an external portable control unit is programmed by the physician to power the implant. Electrode implantation can be achieved