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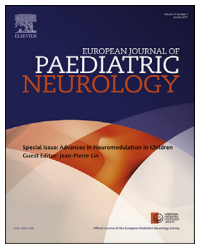

Review article

Electroneurostimulation for the management of bladder bowel dysfunction in childhood

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ABSTRACT

Both non-invasive and invasive electroneurostimulation (ENS) modalities for bladder bowel dysfunction have been studied and reported in children. A summary of the proposed mechanism of actions and the more commonly used and recently reported techniques and outcomes are described. This includes transcutaneous electrical nerve stimulation, functional electrical nerve stimulation, intravesical electrical nerve stimulation, percutaneous tibial nerve stimulation and sacral neuromodulation in conditions including overactive bladder, enuresis, dysfunctional voiding, constipation, combined bladder bowel dysfunction and neuropathic bladder and bowel dysfunction. There is a need for further high quality randomised trials as well as long-term outcomes to establish whether ENS is able to alter the long-term trajectory for an individual child with bladder bowel dysfunction.

Contents

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Abbreviations

ENS	electroneurostimulation
LUT	lower urinary tract
LUTD	lower urinary tract dysfunction
IVES	intravesical electrostimulation
SNM	sacral neuromodulation
TENS	transcutaneous electrical nerve stimulation
FES	functional electrical nerve stimulation
PTNS	percutaneous tibial nerve stimulation
NRCT	non randomised clinical trial
RPT	randomised prospective trial
OAB	overactive bladder
DV	dysfunctional voiding
NB	neuropathic bladder
CBBD	Childhood Bladder bowel dysfunction
CNS	central nervous system

1. Introduction

Electroneurostimulation (ENS) has well documented use in adult bladder bowel dysfunction with an increasing body of evidence emerging for childhood. Childhood lower urinary tract dysfunction (LUTD) is distinct from adult conditions both in the spectrum of pathophysiology and the management and the reader is referred to the Standardisation documents from the International Children's Continence Society for a full description.[1–3] LUTD in children, both non-neuropathic and neuropathic, is associated with urinary tract infection, vesicoureteric reflux, kidney damage and incontinence and is associated with and worsened by any accompanying bowel dysfunction and faecal incontinence. These affect the health and quality of life of affected children and their families.

The commonest underlying etiology of childhood bladder bowel dysfunction is functional or idiopathic and there is increasing epidemiological evidence that this is the second commonest chronic condition of childhood after the atopy/allergy complex. See Fig. 1 for the prevalence and classification of functional childhood incontinence. The reader is referred to the Standardisation documents of the International Children's continence society for further definition and reading. The two main functional bladder disorders causing daytime urinary incontinence are overactive bladder (OAB, with the underlying pathophysiology usually being detrusor overactivity during the storage phase of the bladder cycle), and Dysfunctional voiding (DV, failure of the external urethral sphincter to sufficiently relax during micturition causing functional obstructed micturition). At the other end of the spectrum are the neuropathic causes for CBBD which are far less common but associated with greater morbidity and mortality; congenital spina bifida is the commonest diagnosis in this group but other acquired causes include cerebral palsy, transverse myelitis, tumours, trauma etc. Constipation of the retentive type is the commonest cause of bowel dysfunction.

Management of CBBD is tailored to the condition and can involve cognitive-behavioural techniques, urotherapy, medication, biofeedback programmes, clean intermittent catheterisation, retrograde colonic enemas, injectable botulinum toxin and in some cases surgery with variable limits to success and adverse effects. ENS has been a useful addition to management strategies for both non-neuropathic and neuropathic CBBD although further high quality evidence is required.

2. Neuroanatomy and neurophysiology of the lower urinary tract (LUT)

Stimulation of the hypogastric plexus (spinal level T10-L2) results in sympathetic relaxation of the detrusor muscle and contraction of the intrinsic sphincter thereby facilitating the storage phase of the bladder cycle with inhibition of micturition. Stimulation of the parasympathetic nerves via the sacral micturition centre (spinal level S2–S4) travel in the pelvic nerve resulting in contraction of the detrusor muscle (via release of predominantly cholinergic transmitter) and relaxation of the sphincter thereby facilitating micturition. Somatic nerves from S2–S4 innervate the pelvic floor and external urethral sphincter via the pudendal nerve with 70% of external urethral sphincter pressure dependent upon efferent activity from S3 ventral root, and S2 (and possibly S4)contributing the remaining 30%.[4] Afferent pathways provide a sense of bladder fullness and allow initiation of micturition. There are two types of bladder afferent that travel via the pelvic nerve to the sacral micturition centre (S2–S4); A-δ fibres from mechanoreceptors in the bladder wall detecting fullness and C-fibres carrying noxious or painful signals eg. during a urinary tract infection. The smooth co-ordination between the two bladder states of storage and micturition depend on intact neural pathways in the central nervous system (cortical and pontine micturition centres) which provide amplification, coordination and timing. The bladder is an unusual autonomic visceral organ in that once toilet-training is achieved voluntary initiation of voiding over a wide range of bladder volumes can be initiated at will in an "on-off" manner facilitated via a series of positive feedback loops using supraspinal pathways. The initial concept that babies micturate via a reflex spinal pathway is no longer held