

## Bladder, Bowel and Sexual Function/Dysfunction in Diabetic Peripheral Neuropathy: A Look Beyond the Horizon

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### Abstract

This article was aimed at throwing light on the relatively under-researched area in diabetic peripheral neuropathy (DPN), the presence of visceropelvic disorders secondary to bladder, bowel and sexual dysfunction through a scoping overview of current research evidence. The three studies on bladder dysfunction in DPN uniformly found increased residual urine, urinary retention and slowing of nerve conduction velocities. Three studies on bowel dysfunction found fecal incontinence, increased rectal sensitivity thresholds, prolonged pudendal distal motor latency, asymptomatic sensorimotor deficit, higher mucosal electrosensitivity thresholds and increased fibre density of the external anal sphincter. Three studies on sexual dysfunction demonstrated erectile dysfunction, altered copulatory behaviors and infertility which suggested role of 'central' neuropathy in DPN. The study findings indicate future surgical research on appropriate management for these problems in this patient population.

**Keywords:** Visceropelvic disorders; Urogenital surgery; Surgical neurology; Diabetic neuropathy.

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visceropelvic disorders secondary to bladder, bowel and sexual dysfunction through a scoping overview of current research evidence.

### *Bladder function/ dysfunction*

Andersen and Bradley[1] studied the segmental and supraspinal innervation of the detrusor muscle and periurethral striated musculature in 27 patients with diabetes mellitus by gas cystometry, integrated sphincter electromyography, and spinal evoked-response latency measurements. Neuropathy in the segmental innervation of bladder was evident in presence of slowing of neural conduction velocities.

Beylot *et al*[2] determined the presence of residual urine using post void bladder ultrasonography in 37 control subjects and 102 unselected insulin-dependent and non-insulin-dependent diabetic patients. The presence of residual urine was strongly associated with peripheral neuropathy in the subjects.

Buck *et al*[3] applied urodynamic and electrophysiological techniques to assess the frequency and extent of autonomic and peripheral neuropathy in 60 subjects with diabetes mellitus; 38 were diabetics with suggestive symptoms and the others were representative newly diagnosed (11) or treated (11) diabetics. The study had following findings: "neuropathic bladder dysfunction was detected in 43 of them (71.7%). The

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commonest abnormality was a hypotonic, insensitive large capacity bladder, which condition was usually asymptomatic. Less frequently (15%) was this complicated by bladder decompensation and sphincter involvement, resulting in excessive residual urine and infection; some of these had bladder paralysis with chronic painless retention of urine (7%)."

The three studies on bladder dysfunction in DPN uniformly found increased residual urine, urinary retention and slowing of nerve conduction velocities.

#### *Bowel function/dysfunction*

Erckenbrecht (1988)[4] studied the relationship between fecal incontinence in diabetes mellitus and manifestations of diabetic autonomic or peripheral neuropathy at other organ sites in 12 incontinent and 15 continent diabetics by evaluating stool frequency and stool continence, basal and squeeze anal sphincter pressures, and continence to rectally infused isotonic saline solution (1500 ml). Incontinent diabetics exhibited decreased basal and squeeze anal sphincter pressures, and reduced continence for fluid compared to their continent controls. The degree of incontinence correlated well with the maximal volume of retained rectally infused saline solution, but neither with basal and squeeze anal sphincter pressures, nor with the severity of autonomic or peripheral neuropathy at other organ sites.

Pinna Pintor *et al*[5] investigated the pathophysiology of fecal incontinence in diabetes mellitus, by studying two groups of diabetic patients: 14 subjects with fecal incontinence (Group A) and 15 subjects without fecal incontinence but affected by somatic peripheral neuropathy and third group (C) of 10 healthy volunteers as controls. "Maximum squeeze pressure was found to be lower in A compared to C and sustained for a shorter period in A compared with B and C. All rectal sensitivity thresholds were higher in A compared with B and C. Pudendal Nerve Terminal Motor Latency was prolonged in 93% of patients studied in group A and in 73% of

patients in group B, with a significant difference in comparison with C."

Rogers *et al*[6] studied mucosal electrosensitivity, rectal distension for the quantitative assessment of anorectal sensation, and manometric and electromyographic tests for the assessment of anorectal motor function in 21 patients with diabetic peripheral neuropathy, 18 with idiopathic fecal incontinence and 11 normal controls. An asymptomatic sensorimotor deficit, higher mucosal electrosensitivity thresholds and increased fibre density of the external anal sphincter were found in anal canal of DPN subjects. Pelvic floor sensorimotor neuropathy in diabetic patients thus had several similar features to that of patients with idiopathic fecal incontinence.

Three studies on bowel dysfunction found fecal incontinence, increased rectal sensitivity thresholds, prolonged pudendal distal motor latency, asymptomatic sensorimotor deficit, higher mucosal electrosensitivity thresholds and increased fibre density of the external anal sphincter.

#### *Sexual function/dysfunction*

McVary *et al*[7] defined the neural components of erectile failure by characterizing diffuse neuropathic changes without a confounding vasculopathy in their analysis of sexual function in a diabetic rat (BB/WOR) model as follows: "Copulatory behavioral testing demonstrated that diabetic males were severely impaired: had lesser frequency of mounting, greater latency for mounting, the controls mounted three times more than the diabetics and had about one-half the latency to first mount. The diabetics had about one-fourth the number of intromissions and took nearly twice as long to achieve first ejaculation. The number of ejaculations was drastically reduced as well. Reflex testing demonstrated that spinal sexual reflexes were also severely impaired: the onset latency of reflexes was more than doubled, and the duration of reflexes was less than one-half. More than one-half of the diabetic rats showed no penile

erections. Neural studies showed even more derangement in reflex measures in rats, without erection. Nerve conduction velocity experiments suggested a peripheral neuropathic change in hypogastric nerve and motor pudendal nerve fibers. These dysfunctional findings were seen without any androgen deficiency.”

Ali *et al*[8] studied 100 insulin-dependent diabetes mellitus patients, 314 non-insulin-dependent diabetes mellitus patients with and without an objective evidence of neuropathy, and their age-matched nondiabetic controls for serum and urinary levels of pituitary-gonadal hormones and found low serum total and serum free (urinary) testosterone level and a high serum and urinary FSH and LH and serum prolactin level, specifically in the neuropathic diabetic patients.

Nofzinger[9] emphasized the role of central neuropathy in DPN for the pathophysiological sexual dysfunction since sexual behavior involved the complex integration of higher intellectual function, such as associative memory and the experience of drives and motivations, with basic instinctual or reflexive physiological responses coordinated at the spinal level. Diabetic male erectile dysfunction occurred secondary to a peripheral vasculopathy or neuropathy on forebrain mechanisms of sexual function which was demonstrated by functional brain imaging of human rapid eye movement (REM) sleep, a brain state known to be associated with the periodic occurrence of penile tumescence.

Three studies on sexual dysfunction demonstrated erectile dysfunction, altered copulatory behaviors and infertility which suggested role of ‘central’ neuropathy in DPN.

The study findings indicate future surgical research on appropriate management for these problems in this patient population.

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