Evaluation of different electrophysiological studies in the detection of urinary and sexual dysfunction in diabetic women

Hanan El Sebaie El Hefnawy[a], Nadia Hamed El Arousy[a], Hassan Sayed Shaker[b], Nevine Mahmoud Taha Fouda[a] and Samia Mohamed Rashad Abd El-Raheem[a]

[a]Department of Rheumatology and Physical Medicine and [b]Department of Urology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

Correspondence to Hanan El Sebaie El Hefnawy, MD, Department of Rheumatology and Physical Medicine, Faculty of Medicine, Ain Shams University, Cairo, Egypt
Tel: +01006052551;
e-mail: hanan.sebaie@gmail.com

Received 5 November 2012
Accepted 10 December 2012

Egyptian Rheumatology & Rehabilitation
2013, 40:39–49

Background
Diabetes mellitus is the most common cause of urinary and sexual dysfunction. Although diabetes mellitus can be diagnosed clearly and simply, diabetic neuropathy and diabetic cystopathy (DC) can progress insidiously over time without any symptoms, manifesting itself at a later stage, which increases the risk of secondary complications. Therefore, early diagnosis in the asymptomatic stage of DC with a simple noninvasive method is of utmost importance.

Aim of the work
To evaluate the different electrophysiological studies [including genital sympathetic skin response (SSR), somatosensory-evoked potential (SSEP) of the tibial nerve] in the early detection of urinary and sexual dysfunction in diabetic women.

Patients and methods
This study was carried out on 30 diabetic women and 10 healthy women served as a control group. All patients were divided into two groups (group I and group II) with respect to lower urinary tract symptoms and signs. They were subjected to a full assessment of medical history, full neurological examination, and assessment by the female sexual function index questionnaire. Urodynamic studies including: uroflowmetry and cystometry were carried out for all patients. Electrophysiological studies were carried out for both patients and controls and included nerve conduction studies of both tibial and peroneal nerves, sensory nerve conduction studies of both sural nerves, SSEP of the tibial nerve and genital, hand, and foot SSR.

Results
In group I: Abnormal findings of motor studies were recorded in 1/15 (6.6%) patients, prolonged genital SSR in one patient (6.6%) and absent in two patients 2/15 (13.3%). Prolonged SSEP were recorded in 2/15 (13.3%) of patients. As regards urodynamic study, abnormal findings were detected in 3 patients (20%). In group II: Abnormal findings of motor studies were recorded in 8/15 (53.3%) patients, absent foot SSR in four patients (26.6%), absent genital SSR in seven (46.6%) patients. Prolonged SSEP P40 were recorded in 6/15 (40%). As regards urodynamic study, abnormal findings were detected in 12 patients (80%). There was statistically significant difference between both groups as regards all electerophysiologic parameters except foot latency. There was a statistically highly significant difference between urodynamic diagnosis and genital SSR and SSEP P40 of tibial nerve.

Conclusion
Although urodynamic is essential for the actual diagnosis and the detection of variable pathophysiological changes, electrophysiological studies represent an easy, valid, and noninvasive objective method for the evaluation of DC and sexual dysfunction.

Keywords:
diabetic cystopathy, electerophysiological study, female sexual dysfunction, urodynamic study

Egypt Rheumatol Rehabil 40:39–49

1110-161X

Introduction

Diabetes mellitus (DM) is a common disease worldwide and a growing public health burden. Neuropathy is one of the most prevalent, devastating, and costly complications of diabetes. It may lead to dysfunction of the peripheral as well as the central nervous system, the somatic as well as the autonomic nervous system [1].

DM is the most common cause of urinary dysfunction secondary to peripheral nervous system pathology. The prevalence rate of bladder dysfunction increases with the duration of DM; the rate is around 25% after 10 years and greater than 50% after 45 years of diabetes [2].

Diabetic autonomic neuropathies are a heterogeneous and progressive disease entity that commonly complicate

both type I and type II DM. Diabetic autonomic neuropathies affecting the urogenital tract show two clinically relevant manifestations: the diabetic neurogenic voiding dysfunction [diabetic cystopathy (DC)] and complex sexual functional disturbances in men and women [3].

DC is characterized by a steady increase in residual urine and bladder capacity with diminished bladder sensation and decreased bladder contractility. It can progress insidiously over time without any symptoms, manifesting itself at a later stage. This insidious progress increases the risk of secondary complications [4].

Urodynamic tests are the most important functional tests in urinary dysfunction. However, the morbidity of an invasive urodynamic study (UDS) in diabetic patients includes urinary tract infection, fever, urinary retention, and gross hematuria. Therefore, the UDS indication should be evaluated carefully, especially in diabetic men who have high residual urine volume and diabetic women who have pyuria before UDS [5].

Electrophysiological studies of peripheral nerve function are the most sensitive, reliable, and reproducible measures of nerve function, which also correlate with the morphologic findings on nerve biopsy. The nerve conduction studies can define nerve dysfunction in a simple and noninvasive way [6,7].

Aim of the work

To evaluate the different electrophysiological studies [nerve conduction studies, genital sympathetic skin response (SSR), somatosensory-evoked potential (SSEP)] in the early detection of urinary and sexual dysfunction in diabetic women.

Patients and methods

This study included 30 diabetic married middle-aged women. Patients were diagnosed according to the diagnostic criteria of the American Diabetes Association [8]. They ranged in age from 30 to 60 years. They were recruited from the Diabetic Outpatient Clinics of Ain Shams University Hospital.

Ten healthy individuals matched for age and sex served as a control group after measurement of fasting blood glucose levels to exclude diabetes. Both patients and controls provided informed consent for participation in the study after a full explanation of the procedure was provided.

Patients with any neurological disease that could affect the urinary tract and those with lower urinary pathology other than DC were excluded by clinical examination or routine investigation (e.g. urine analysis and culture – renal function test).

The patients were divided into two groups, group I (asymptomatic group number = 15) and group II (symptomatic group number = 15), with at least two symptoms out of a list of 10 lower urinary tract symptoms (LUTS) and/or abnormal findings in the frequency volume charts (Table 1) according to the International Continence Society standardization [9].

Table 1 Lower urinary tract symptoms and signs suggestive of lower urinary tract dysfunction [6]

Symptoms	Yes	No
LUTS		
Symptoms of over active bladder		
Urgency		
Urge incontinence		
Stress incontinence		
Frequency		
Obstructive LUTS		
Hesitancy		
Slow urine stream		
Splitting or spraying of urine stream		
Intermittent urine stream		
Straining		
Terminal dribble/postmicturition dribbles		
Feeling of incomplete emptying		
LUTD		
Frequency volume chart	Normal	Abnormal
Functional bladder capacity (ml)	≤ 550	>550
Frequency (voids)	≤ 7	>7

LUTD, lower urinary tract dysfunction; LUTS, lower urinary tract symptoms.

All patients were subjected to the following:

(1) Full assessment of history with a focus on duration of disease and history of medication. Sexual function was evaluated using the female sexual function index (FSFI) questionnaire [10], which assesses the six key (domains) of sexual function in women including desire, arousal, lubrication, orgasm, satisfaction, and pain.
(2) Neurological assessment of the patients for sensation, temperature, vibration, reflexes, and muscle power and also pelvic neurological examination for perianal sensation, anal, and bulbocavernous reflex to assess genitourinary autonomic function.
(3) Electrophysiological studies were carried out for both patients and controls and included the following:
    (a) Examination of the integrity of the somatic peripheral nervous system – Motor nerve conduction studies of both tibial and peroneal nerves – Sensory nerve conduction studies of both sural nerves.
    (b) Examination of central somatosensory pathways:
        (i) SSEP of the tibial nerve.
    (c) Examination of the autonomic nervous system:
        (ii) Hand and foot SSR.
        (iii) Genital SSR.

Apparatus

The electromyography apparatus used was Tonnies version 1.59 (Germany).

Electrodes

Motor nerve recording electrode: The active electrode E-l was applied to the motor point of the muscle and the

reference electrode E-2 was placed at or just beyond the muscle tendinous insertion.

Sensory nerve recording electrode: Sensory nerve recording electrode was applied as the surface electrode.

Stimulation electrode: This is a bipolar surface electrode. The distal tip is the cathode and it is placed distally.

Methods

Electrophysiological studies were performed in a quiet room with a constant temperature set at 27°C using thermostat of air condition. The patient was placed in a lying position, and allowing maximum relaxation.

(1) Motor nerve conduction of the posterior tibial nerve:
   (a) The recording active electrode was secured 1 cm posterior and inferior to the navicular tubercle on the medial aspect of the foot on the abductor hallucis muscle.
   (b) The reference electrode was secured to the distal aspect of the first digit.
   (c) Site of stimulation: A distal stimulus posterior to the medial malleolus and a proximal stimulus at the crease of the popliteal fossa.
(2) Motor nerve conduction of the common peroneal nerve.
   (a) The recording active electrode was secured over the extensor digitorum brevis muscles.
   (b) The reference electrode was secured 3 cm distal to the active electrode on the dorsum of the foot.
   (c) Site of stimulation: A distal stimulus midway between the malleoli on the anterior surface of the limb (between the tibialis anterior and extensor hallucis proprius tendons) and a proximal stimulus at the head of the fibula.

Somatosensory-evoked potentials

The posterior tibial nerve was stimulated at the ankle using surface electrodes. The stimulation rate was 3/s, with a stimulus duration of 0.5 ms for the tibial. (The intensity of the stimulus was such that it produced small contraction of the intrinsic foot muscles.) The filter settings included a low-frequency filter 5 Hz and a high-frequency filter 2 kHz. The gain was 5–8 μv and the sweep speed was 10 ms.

The site of the recording electrodes G1 (reference electrode) was over the upper mid forehead and G2 (active electrode) placed 1 cm behind Cz, which is the point half-way between inion (the external occipital protuberance) and nasion (the point in the skull where the frontal and nasal bones unite). The ground electrode was strapped around the patient's neck.

Averaging of about 300 responses was performed and the cortical evoked potentials (P40) were assessed.

Sensory antidromic nerve conduction of the sural nerve

The recording active electrode was placed behind the lateral malleolus. The reference electrode was placed 3 cm distally.

Stimulation

Stimulation was 14 cm proximal from the active electrode, slightly lateral to the midline in the lower third of the posterior aspect of the leg, with the cathode placed distally.

Sympathetic skin response

Hand sympathetic skin response

To stimulate the median nerve at the wrist, an active electrode was placed on the palm, a reference electrode was placed on the dorsum of the hand, and a ground electrode was placed on the forearm.

Foot sympathetic skin response

To stimulate the median nerve at the wrist, an active electrode was placed on the planter surface of the foot, a reference electrode was placed on the dorsum of the foot, and a ground electrode was placed on the forearm.

Genital sympathetic skin response

To stimulate the median nerve at the wrist, an active electrode was placed on the mons pubis, a reference electrode was placed on the anterior superior iliac spine, and a ground electrode was placed on the forearm.

To avoid any habituation, stimulations were carried out at randomized intervals and various intensities. The duration of the stimulus was between 0.1 and 0.2 ms and the stimulus intensity ranged from 10 to 40 mA. A sensitivity of 500 μv to 2 mv per division and a sweep speed of 1–2 s were used for recording. Low-frequency and high-frequency filters were adjusted between 0.1 and 1000 Hz.

Interpretation of the response

(1) If we did not elicit any response to 10 consecutive stimuli, the response was considered to be absent.
(2) The latency was measured from the onset of the stimulus artifact to the first deflection of the signal baseline.
(3) The amplitude was measured from peak to peak [11].
   (a) The shape of the response was a triphasic, a biphasic, or a monophasic wave of either P-type or N-type [11].

Urodynamic studies

Urodynamic studies were carried out using Dantec UD 5500 equipment (Dantec Inc., Copenhagen, Denmark). A double-lumen 6–8 F urethral catheter was introduced and normal saline was instilled at a rate of 5–20 ml/min to fill the bladder. Vesical, abdominal, and detrusor pressures were monitored simultaneously during the filling and voiding phases.

Bladder volume at the first sensation, maximum bladder capacity, bladder compliance, filling pressure, opening pressure, and detrusor pressure at maximum flow were recorded; the maximum and average flow rates were also recorded [8].

The value for first desire to void was 200–330 ml in female participants, and the maximum bladder capacity ranged between 300 and 500 ml. The end filling pressure

was considered normal if it was less than 20 cm H_2O at full bladder capacity and there should be no detrusor overactivity [12].

Statistical analysis

The data collected were revised, coded, tabulated, and entered into a personal computer using the Statistical package for Social Science (IBM SPSS Statistics 19.0, for windows; IBM Corporation, New York, New York, USA, August 2010). Data were presented and the relevant analysis was carried out according to the type of data obtained for each parameter. *P*-value level of significance; *P* greater than 0.05: nonsignificant (NS), *P* less than 0.05: significant (S), *P* less than 0.01: highly significant (HS).

Results

The results of this study are shown in Tables 2–28 and Figs 1 and 2.

A total of 30 diabetic women were enrolled in this study. Their age ranged from 31 to 60 years (mean 41 + 5). The duration of DM ranged from 2 to 37 years (mean 19 + 6). All the patients had type 2 diabetes. Twenty-eight patients (93.3%) were on oral hypoglycemics and two (6.6%) were on insulin.

The patients were divided into two groups: group I [asymptomatic group, $n = 15$ (50%)] and group II [symptomatic group, $n = 15$ (50%)] with at least two symptoms of a list of 10 LUTS and/or abnormal findings in the frequency volume charts.

Demographic and clinical data of group I ($n = 15$)

Their ages ranged from 30 to 55 years (mean 37 ± 7 years). The duration of diabetes ranged from 2 to 20 years (mean 8.52 ± 4.29 years). Fourteen patients (93.7%) were receiving oral hypoglycemic drugs and only one patient (6.7%) was on insulin therapy. The LUTS, autonomic, and neurologic symptoms of group I are shown in Table 2. Assessment of the patients by FSFI is shown in Table 3.

Urodynamic data in group I

The results of different urodynamic parameters (cystometry and uroflowmetry) are shown in (Table 4). According to these parameters, 12 patients (80%) were normal, two patients (13.3%) had detrusor instability, and one patient (6.7%) had DC (Table 5).

Electrophysiological data in group I

The parameters of nerve conduction studies (latency, amplitude, and velocity) of the tibial, common peroneal, and sural nerves and the SSR (hand–foot–genital) are shown in Tables 6 and 7. Abnormal findings of motor studies were recorded in 1/15 (6.6%) patients, whereas genital SSR were absent in two patients 2/15 (13.3%) and prolonged in one patient (6.6%). The values of SSEP P40 ranged from 42.6 to 50 (mean 44.7 ± 1.8). Prolonged SSEP were recorded in 2/15 (13.3%) of patients.

Table 2 Clinical assessment of group I

Symptoms	Number of patients (%)
Urinary symptoms	
Urgency	2 (13.3)
Urge incontinence	0 (0)
Stress incontinence	4 (26.7)
Hesitancy	0 (0)
Weak urine stream	1 (6.7)
Splitting or spraying of urine stream	0 (0)
Intermittent urine stream	1 (6.7)
Straining	1 (6.7)
Terminal dribble/postmicturition dribbles	1 (6.7)
Feeling of incomplete emptying	0 (0)
Autonomic symptoms	
Orthostatic hypotension	2 (13.3)
Sweating	5 (33.3)
Neurological symptoms	
Hypothesia	
Unilateral	1 (6.7)
Bilateral	3 (20)
Diminished vibration sense	2 (13.3)
Abnormal reflexes	1 (6.7)

Table 3 Assessment of the patients by female sexual function index (Rosen, 2000)

	Group I	
	Min–max	Mean ± SD
Desire score	1–4	2.66 ± 1.11
Arousal score	1–4	2.8 ± 0.8
Lubrication	1–3	2.16 ± 0.7
Orgasm score	1–4	2.46 ± 0.9
Satisfaction score	1–5	3 ± 1.44
Pain score	1–4	2.8 ± 0.8
FSFI total score	6–23	15.9 ± 5.7

FSFI, female sexual function index; max, maximum; min, minimum.

Table 4 Urodynamic parameters in group I

	Min–max	Mean ± SD
Cystometry parameters		
First desire to void (ml)	110–168	136.3 ± 17.5
Maximum bladder capacity (ml)	265–625	450 ± 81
Compliance (ml/cmH_2O)	3.1–60	34.3 ± 16.6
Uroflowmetry parameters		
Maximum flow rate (ml/s)	18.1–34	28 ± 4.27
Average flow rate (ml/s)	7.7–16.3	12.2 ± 2.5
Voided volume (ml)	200–719	380 ± 108.9
Voiding time (s)	24–53	33.3 ± 7.7
Time to maximum flow (s)	5–16	7.4 ± 2.9
Residual urine (ml)	30–70	50.4 ± 12

Max, maximum; min, minimum.

Table 5 Urodynamic diagnosis in group I

Urodynamic diagnosis	Number of patients (%)
Normal	12 (80)
Detrusor instability	2 (13.3)
Diabetic cystopathy	1 (6.7)

Comparison of electrophysiological studies between the control group and group I showed a statistically nonsignificant difference in all parameters ($P > 0.05$), except for foot SSR amplitude, which showed statistically significant difference ($P < 0.05$).

Table 6 Parameters of nerve conduction studies of tibial, common peroneal and sural nerves in group I

	Latency		Amplitude		Velocity	
Nerve	RT	LT	RT	LT	RT	LT
Tibial nerve	3.96 ± 0.6	4.09 ± .65	14 ± 3.6	13 ± 2.7	45.6 ± 4.7	45.5 ± 4.6
Common peroneal nerve	4 ± 0.5	4.1 ± 0.5	6 ± 0.9	5.7 ± 0.9	44.7 ± 4.6	44.5 ± 4.4
Sural nerve	3.6 ± 1	3.5 ± 1.06	5.14 ± 1.18	5.26 ± 1.18	40.3 ± 8.3	41.5 ± 9.3

LT, left; RT, right.

Table 7 Parameters of (hand–foot–genital) sympathetic skin response in group I

	Group I	
Parameters of SSR	Min–max	Mean ± SD
Hand latency (s)	1.25–1.9	1.41 ± 0.18
Hand amplitude (μv)	260–450	377 ± 60.88
Foot latency (s)	1.8–2.6	1.98 ± 0.2
Foot amplitude (μv)	100–170	143 ± 23.12
Genital latency (s)	1.5–1.98	1.44 ± 0.59
Genital amplitude (μv)	100–240	211 ± 36.7

Max, maximum; min, minimum; SSR, sympathetic skin response.

Table 8 Clinical assessment of group II

Symptoms	Number of patients (%)
Lower urinary tract	
Urgency	3 (20)
Urge incontinence	0 (0)
Stress incontinence	12 (80)
Hesitancy	6 (40)
Weak urine stream	4 (26.7)
Splitting or spraying of urine stream	0 (0)
Intermittent urine stream	2 (13.3)
Straining	10 (66.7)
Terminal dribble/postmicturition dribbles	1 (6.7)
Feeling of incomplete emptying	8 (53.3)
Autonomic symptoms	
Orthostatic hypotension	9 (60)
Diarrhea	4 (26.7)
Sweating	1 (6.7)
Neurological symptoms	
Hypothesia	
Unilateral	3 (20)
Bilateral	10 (66.7)
Diminished vibration sense	10 (66.7)
Abnormal reflexes	6 (40)

Table 9 The Score of desire, arousal, lubrication, orgasm, pain, satisfaction, and female sexual function index total score

	Group II	
	Min–max	Mean ± SD
Desire score	1–3	1.76 ± 0.72
Arousal score	1–4	1.93 ± 0.88
Lubrication	1–3.5	1.83 ± 0.74
Orgasm score	1–3	1.80 ± 0.75
Satisfaction score	1–5	2.4 ± 1.2
Pain score	1–3	1.9 ± 0.81
FSFI total score	6–20	11.7 ± 4.55

FSFI, female sexual function index; max, maximum; min, minimum.

Demographic and clinical data of group II (*n* = 15)

The age of the patients in group II ranged from 31 to 60 years (mean 46 ± 9 years). The duration of diabetes ranged from 4 to 37 years (mean of 8.52 ± 4.29 years). Fourteen patients (93.7%) were receiving oral hypoglycemic drugs and only one patient (6.7%) was on insulin therapy. The LUTS, autonomic, and neurologic symptoms of group I are shown in Table 8. Assessment of the patients by FSFI is shown in Table 9.

Table 10 Urodynamic parameters in group II

	Median	IQR
Cystometry parameters		
First desire to void (ml)	145	85–169
Maximum bladder capacity (ml)	420	369–724
Compliance (ml/cmH_2O)	21.6	5–30
Uroflowmetry parameters		
Maximum flow rate (ml/s)	18.1	12.7–30
Average flow rate (ml/s)	7.7	4.2–12.7
Voided volume (ml)	338	300–430
Voiding time (s)	30	28–35
Time to maximum (s)	7	5–8
Residual urine flow (ml)	60	30–110

IQR, interquartile range.

Table 11 Urodynamic diagnosis in group II

Urodynamic diagnosis	Number of patients (%)
Normal	3 (20)
Diabetic cystopathy	4 (26.6)
Detrusor instability with incontinence	3 (20)
Large capacity and detrusor hypocontractility	2 (13.3)
Detrusor hypocontractility	2 (13.3)
Urinary retention	1 (6.7)

Urodynamic data in group II

The results of different urodynamic parameters (cystometry and uroflowmetry) are shown in Table 10. According to these parameters, 3 patients (20%) were normal, 4 patients (26.6%) had diabetic cystopathy, 3 patient (20%) had detrusor instability with incontinence, 2 patients (13.3%) had Large capacity and detrusor hypocontractility, 2 patients (13.3%) had detrusor hypocontractility and one patient (6.7%) had urinary retention detrusor instability, and one patient (6.7%) had DC (Table 11).

Electrophysiological data in group II

The parameters of nerve conduction studies (latency, amplitude, and velocity) of the tibial, common peroneal, and sural nerves and the SSR (hand–foot–genital) are shown in Tables 12 and 13. Abnormal findings of motor studies were recorded in 8/15 (53.3%) patients. Foot SSR were absent in four patients (26.6%), whereas genital

Table 12 Parameters of nerve conduction studies of tibial, common peroneal, and sural nerves in group II

	Latency		Amplitude		Velocity	
Nerve	RT	LT	RT	LT	RT	LT
Posterior tibial nerve	5.56 ± 1.11	5.72 ± 1.14	8.84 ± 2.7	9 ± 2.6	36.8 ± 6.4	36.7 ± 5.65
Common peroneal nerve	5.6 ± 1.02	5.64 ± 0.94	4.9 ± 0.8	4.88 ± 0.89	34.7 ± 4.8	34.2 ± 4.2
Sural nerve	5.76 ± 1.38	5.68 ± 1.45	3.55 ± 1.41	3.48 ± 1.55	25.6 ± 6.7	26 ± 8

LT, left; RT, right.

Table 13 Parameters of (hand–foot–genital) sympathetic skin response in group II

Parameters of SSR	Min–max	Mean ± SD
Hand latency (s)	1–2	1.76 ± 0.26
Hand amplitude (μv)	100–410	257.33 ± 89.3
Foot latency (s)	2–2.9	1.75 ± 1.13
Foot amplitude (μv)	97–150	78.8 ± 51.7
Genital latency (s)	1.7–2.5	1.12 ± 1.11
Genital amplitude (μv)	97–235	148.7 ± 61.67

Max, maximum; min, minimum; SSEP, somatosensory-evoked potential; SSR, sympathetic skin response.

Table 14 Comparison between group I and group II as regards clinical data

LUTS	χ^2	*P*-value	Significance
Urinary symptoms			
Urgency	0.166	0.684	NS
Urge incontinence	0.166	0.684	NS
Stress incontinence	8.571	0.003	HS
Obstructive urinary symptoms			
Hesitancy	7.500	0.006	HS
Slow urine stream	2.16	0.142	NS
Splitting or spraying of urine stream	2.16	0.142	NS
Intermittent urine stream	0.370	0.543	NS
Straining	11.627	0.001	HS
Terminal dribble/postmicturition dribbles	1.034	0.309	NS
Feeling of incomplete emptying	7.778	0.005	HS
Autonomic symptoms			
Orthostatic hypotension	7.033	0.008	HS
Sweating	0.159	0.690	NS
Diarrhea	1.034	0.309	NS
Neurologic symptoms			
Hypothesia	11	0.004	HS
Temperature	11.627	0.001	HS
Vibration sense	8.889	0.003	HS
Reflexes	4.658	0.031	S

HS, highly significant; NS, nonsignificant; S, significant.

Table 15 Comparison between group I and group II as regards female sexual function index

	T-value	*P*-value	Significance
Desire score	2.621	0.014	S
Arousal score	2.319	0.028	S
Lubrication	1.220	0.232	NS
Orgasm score	2.180	0.038	S
Satisfaction score	1.287	0.2	NS
Pain score	2.804	0.009	HS
FSFI total score	2.258	0.032	S

FSFI, female sexual function index; HS, highly significant; NS, nonsignificant; S, significant.

SSR were absent in seven (46.6%) patients. The values of SSEP P40 ranged from 46 to 52 (mean 38.5 ± 20). Prolonged SSEP P40 were recorded in 6/15 (40%) patients.

Comparison of electrophysiological studies between control group and group II revealed a statistically high significant difference ($P<0.01$) as regards all parameters, except left common peroneal amplitude, genital latency, and SSEP P40 showed a statistically significant difference ($P<0.05$). Foot SSR latency showed statistically nonsignificant difference ($P>0.05$).

Comparison between group I and group II

Demographic and clinical data

There were statistically significant differences between both groups in the duration of diabetes ($P<0.05$). Comparison between both groups showed a statistically highly significant difference ($P<0.01$) in stress incontinence, hesitancy, straining, and feeling of incomplete emptying, whereas other urinary symptoms showed statistically nonsignificant differences ($P>0.05$) (Tables 14 and 15).

Urodynamic data

There was a statistically nonsignificant difference ($P>0.05$) between both groups in all urodynamic parameters, except for the average flow rate and compliance, which showed a highly significant statistical difference ($P<0.01$) (Table 16).

Comparison between both groups in abnormal cases detected by urodynamic studies showed a statistically highly significant difference ($P<0.01$) (Table 17).

Electrophysiological data

Comparison of electrophysiological studies between group I and group II showed a statistically highly significant difference ($P<0.01$) in all parameters, except the left common peroneal amplitude, genital latency, and SSEP P40, which showed a statistically significant difference ($P<0.05$). Foot latency showed a statistically nonsignificant difference ($P>0.05$) (Table 18).

Correlation studies of group I

A positive urodynamic diagnosis was associated significantly with a higher incidence of DM complications and longer duration of DM (Tables 19 and 20).

A positive urodynamic diagnosis was significantly associated with positive findings of SSEP P40 ($P=0.029$) and positive findings of genital SSR ($P=0.009$) (Tables 21 and 22).

Correlation studies of group II

A positive urodynamic diagnosis was significantly associated with a higher incidence of DM complications

Table 16 Comparsion between both groups as regards parameters of urodynamic study

	Group I		Group II				
	Median	IQR	Median	IQR	*Z*	*P*	Significance
Cystometry parameters							
First desire to void (ml)	130	125–150	145	85–169	−0.872	0.383	NS
Maximum bladder capacity (ml)	440	400–500	420	369–724	−0.498	0.618	NS
Compliance (ml/cmH_2O)	35	24.7–44	21.6	5–30	−2.491	0.013	S
Uroflowmetry parameters							
Maximum flow rate (ml/s)	28	25–32	18.1	12.7–30	−1.931	0.053	NS
Average flow rate (ml/s)	13	10–14	7.7	4.2–12.7	−2.35	0.019	S
Voiding volume (ml)	360	340–400	338	300–430	−1.06	0.289	NS
Voiding time (s)	30	28–35	41	28–77	−1.330	0.184	NS
Time to maximum flow (s)	7	5–8	11	6–27	−1.649	0.099	NS
Residual urine flow (ml)	50	45–60	60	30–110	−0.52	0.603	NS

HS, highly significant; IQR, interquartile range; NS, nonsignificant; S, significant.

Table 17 Comparisons between group I and group II as regards urodynamic diagnosis

	Positive urodynamic findings	Negative urodynamic findings
Group I	3	12
Group II	12	3
P-value	0.001	

Table 18 Comparisons between group I and group II as regards electrophysiological data

	T-value	*P*-value and significance
Right post. T. latency (ms)	−4.811	0, HS
Left post. T. latency (ms)	−4.784	0, HS
Right post. T. amplitude (μv)	4.419	0, HS
Left post. T. amplitude (μv)	4.025	0, HS
Right post. T. conduction velocity (m/s)	4.263	0, HS
Left post. T. conduction velocity (m/s)	4.656	0, HS
Right C.P. latency (ms)	−4.891	0, HS
Left C.P. latency (ms)	−5.284	0, HS
Right C.P. amplitude (μv)	3.506	0.002, HS
Left C.P. amplitude (μv)	2.517	0.01, S
Right C.P. conduction velocity (m/s)	5.785	0, HS
Left C.P. conduction velocity (m/s)	6.495	0, HS
F-wave tibial	−3.082	0.005, HS
Right sural latency (ms)	−4.835	0, HS
Left sural latency (ms)	−4.598	0, HS
Right sural amplitude (μv)	3.364	0.002, HS
Left sural amplitude (μv)	3.541	0.001, HS
Right sural conduction velocity (m/s)	−4.835	0, HS
Left sural conduction velocity (m/s)	4.859	0, HS
Hand latency (s)	−4.099	0, HS
Hand amplitude (μv)	4.288	0, HS
Foot latency (s)	0.764	0.4, NS
Foot amplitude (μv)	4.384	0.000, HS
Genital latency (s)	−0.974	0.03, S
Genital amplitude (μv)	2.923	0.009, HS
SSEP P40	0.925	0.02, S

CP, common peroneal nerve; HS, highly significant; IQR, interquartile range; NS, nonsignificant; Port T, post tibial nerve; S, significant; SSEP, somatosensory-evoked potential.

($P = 0.036$) and a longer duration of DM ($P = 0.024$) (Tables 23 and 24).

A positive urodynamic diagnosis was significantly associated with positive findings of SSEP P40 ($P = 0.044$) and positive findings of genital SSR ($P = 0.001$) (Tables 25 and 26).

The sensitivity and specificity of electrophysiological studies in a urodynamic diagnosis in group I and group II are shown in Tables 24 and 25 and Figs 1 and 2.

Table 19 Correlation between urodynamic diagnosis and diabetes mellitus duration in group I

	Diabetes mellitus duration				
Urodynamic diagnosis	1–5 years	5–10 years	10–15 years	>15 years	Total
Positive findings	0	0	1	2	3
Negative findings	5	6	1	0	12
Total	5	6	2	2	15
P-value			0.008		

Table 20 Correlation between urodynamic diagnosis and diabetes mellitus complications in group I

	Diabetes mellitus complication			
Urodynamic diagnosis	No complication	Hypertension	Eye complication	Total
Positive findings	1	1	1	3
Negative findings	10	2	0	12
Total	11	3	1	15
P-value		0.034		

Table 21 Correlation between somatosensory-evoked potential P40 and urodynamic diagnosis in group I

	Urodynamic diagnosis		
SSEP P40	Positive finding	Negative finding	Total
Positive finding	2	0	2
Negative finding	1	12	13
Total	3	12	15
P-value		0.029	

P-value by Fisher's exact test.
SSEP, somatosensory-evoked potential.

Discussion

DC can progress insidiously over time without any symptoms, manifesting itself at a later stage. It has been reported that detailed and meticulous investigations can identify DC in only 25–50% of diabetic patients with no evident symptoms. This insidious progress increases the risk of secondary complications [3].

Therefore, early diagnosis in the asymptomatic stage of DC with a simple noninvasive method is of utmost importance.

The LUTS questionnaire showed that in group I (asymptomatic), the main symptoms were overactive symptoms of the bladder, whereas in group II (symptomatic) the main symptoms were an obstructive lower urinary tract.

This might be explained by the fact that diabetic bladder dysfunction includes time-dependent manifestations of storage and emptying problems. Detection of bladder dysfunction in the compensated phase with overactive symptoms; prevents the decompensate phase which causes obstructive urinary symptoms up to urinary retention [13].

This was in agreement with Rapidi *et al*. [14], who used the same questionnaire to classify their patients. However, other investigators such as Soylu *et al.* [15], Kebapci *et al.* [16], Esteghamati *et al.* [17], and Bansal *et al.* [18] used the international prostate symptom score to evaluate urinary symptoms. The reason we did not use the international prostate symptom score because it is a screening tool that is used for rapid diagnosis, tracking the symptoms of, and suggesting management of the symptoms of benign prostatic hyperplasia and because it cannot be used for female patients.

In our study, assessment of sexual function in diabetic women was carried out using FSFI of Rosen *et al.* [10] and still used in clinical studies by Gerstenberger *et al.* [19], Takahashi *et al.* [20]; this can be explained by the fact that the sexual response involves a temporal sequencing and coordination of several phases. Thus, problems in one area may interact with those in another, resulting in a considerable overlap among the diagnostic categories. As FSFI is a multidimensional questionnaire covering all sexual domains, it can be used in women and can lead to early detection of sexual dysfunction.

This was in agreement with Abu Ali *et al.* [21], Esposito *et al.* [22], and Wallner *et al.* [23], who used the same FSFI questionnaire, whereas other investigators such as Fatemi *et al*. [24] used a questionnaire prepared from the DSM-IV (American psychiatric association diagnostic and statistical manual) algorithm on sexual satisfaction and the Arizona sexual experience scale form.

In agreement with our study, Abu Ali *et al.* [21] assessed the prevalence of female sexual dysfunction among diabetic Jordanian women, and used the same FSFI questionnaire and concluded that desire, arousal, and orgasm are more significantly affected in diabetic women. This supports our results that desire, arousal, and orgasm are the mostly affected domains in diabetic women.

In our study, there was a significant correlation between FSFI questionnaires, especially the FSFI total score with

Table 22 Correlation between genital sympathetic skin response and urodynamic diagnosis in group I

	Urodynamic diagnosis		
Genital SSR	Positive finding	Negative finding	Total
Positive finding	2	1	3
Negative finding	1	11	12
Total	3	12	15
P-value		0.009	

SSR, sympathetic skin response.

Table 23 Correlation between urodynamic diagnosis and diabetes mellitus duration in group II

	Diabetes mellitus duration				
Urodynamic diagnosis	1–5 years	5–10 years	10–15 years	>15 years	Total
Positive findings	0	2	2	8	12
Negative findings	1	2	0	0	3
Total	1	4	2	8	15
P-value			0.024		

Table 25 Correlation between somatosensory-evoked potential P40 and urodynamic diagnosis in group II

	Urodynamic diagnosis		
SSEP P40	Positive finding	Negative finding	Total
Positive finding	9	3	12
Negative finding	3	0	3
Total	12	3	15
P-value		0.044	

P-value by Fisher's exact test.
SSEP, somatosensory-evoked potential.

Table 26 Correlation between genital sympathetic skin response and urodynamic diagnosis in group II

	Urodynamic diagnosis		
Genital SSR	Positive finding	Negative finding	Total
Positive finding	10	2	12
Negative finding	2	1	3
Total	12	3	15
P-value		0.001	

SSR, sympathetic skin response.

Table 24 Correlation between urodynamic diagnosis and diabetes mellitus complications in group II

	Diabetes mellitus complication				
Urodynamic diagnosis	No complication	Hypertension	Eye complication	Both eye complication and hypertension	Total
Positive findings	2	2	6	2	12
Negative findings	3	0	0	0	3
Total	5	2	6	2	15
P-value			0.036		

Table 27 The sensitivity and specificity of different parameters in group I as regards urodynamic diagnosis

Parameters	Sensitivity (%)	Specificity (%)
Sural latency	100	91.6
SSR genital latency	100	91.6
SSR genital amplitude	100	83.3
SSR hand	100	91.6
SSR foot	100	91.6
SSEP P40	33.3	91.6

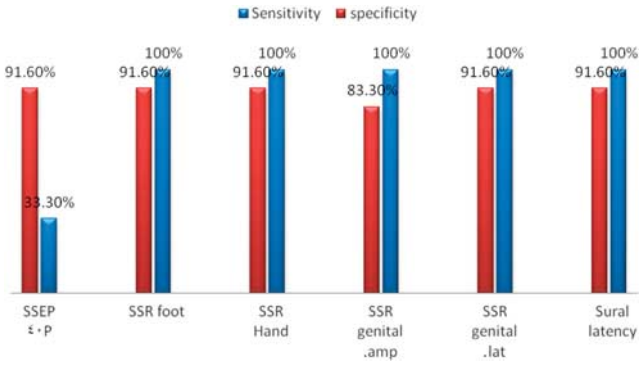
SSEP, somatosensory-evoked potential; SSR, sympathetic skin response.

Table 28 The sensitivity and specificity of different parameters in group II as regard urodynamic diagnosis

Parameters	Sensitivity (%)	Specificity (%)
Sural latency	100	33.3
SSR genital latency	100	66.6
SSR genital amplitude	91.6	66.6
SSR hand	100	91.6
SSR foot	100	33.3
SSEP P40	75	100

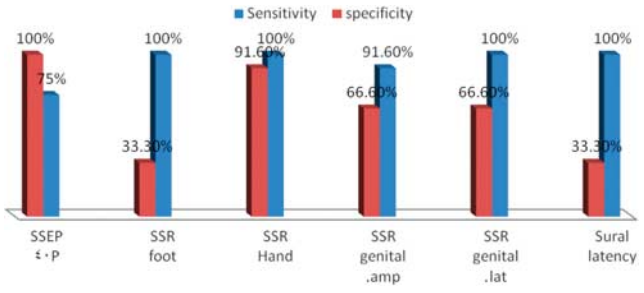
SSEP, somatosensory-evoked potential; SSR, sympathetic skin response.

Figure 1



Sensitivity, specificity of different parameters in group I as regards urodynamic diagnosis. SSEP, somatosensory-evoked potential; SSR, sympathetic skin response.

Figure 2



Sensitivity, specificity of different parameters in group II as regards urodynamic diagnosis. SSEP, somatosensory-evoked potential; SSR, sympathetic skin response.

the genital SSR. This can be attributed to the fact that any pathological process altering the function of the lumbosacral sympathetic divisions can affect both sudomotor and sexual activity. Therefore, genital SSR is an objective and potentially useful method to assess genitourinary and sexual dysfunction in diabetic women, which is particularly difficult to analyze [25,26].

Our results indicated that, in group I, 12 patients (80%) had a normal urodynamic diagnosis, two patients (13.3%) had detrusor instability, and one patient (6.7%) had DC, whereas in group II, three patients (20%) were normal, four patients (26.6%) had DC, three patients (20%) had detrusor instability with incontinence, two patients (13.3%) had large capacity and detrusor hypocontractility, two patients (13.3%) had detrusor hypocontractility, and one patient (6.7%) had urinary retention. From the above results, we can conclude that diabetic patients present with different urodynamic findings and diagnoses.

This was in agreement with Kaplan and Blaivas [27], who carried out a video UDS of 182 patients and analyzed as follows: 100 (55%) had detrusor hyperreflexia, 42 (23%) had impaired detrusor contractility, 20 (11%) had indeterminate findings, 19 (10%) had detrusor areflexia, and one (1%) was normal. Bladder outlet obstruction was present in 66 patients (36%), all men (57%). The most common urodynamic diagnoses were either impaired detrusor contractility in 21 patients (50%) or detrusor areflexia in 10 patients (24%).

Bansal *et al.* [18] have reported that patients with DM and LUTS can present with various urodynamic findings, apart from the classic urodynamic features of DC (delayed first sensation, high PVR, and increase in bladder compliance) and other abnormalities such as detrusor overactivity in 36.1% and bladder outlet obstruction in 28.8%; only 8% of patients are normal.

Accordingly, the classical features of DC are not the most common urodynamic findings in diabetic patients presenting with voiding dysfunction, and in fact, these patients present with various pathophysiological findings, which supports our results.

Urodynamic findings with DM duration and complications showed a statistically significant correlation between a urodynamic diagnosis and DM complications (such as hypertension, eye problems, e.g. cataract, glaucoma) and a statistically highly significant correlation between a urodynamic diagnosis and DM duration in both groups successively.

A similar correlation was also obtained by Kebapci *et al.* [16]. They found a strong correlation between duration of diabetes and urodynamic findings. This supports our results that DM duration and DM complications can be risk factors when screening for bladder dysfunction.

In our study, there was a statistically highly significant correlation between urodynamic findings and SSR genital, respectively. Similar results were obtained by Rodic *et al.* [28], who compared hand, foot, and genital

SSR recorded by video UDS for 90 patients; they found that genital SSR represents a sensitive diagnostic tool for assessing sympathetic nerve function within the thoracolumbar spinal cord and is of diagnostic value for evaluating neurogenic bladder in spinal cord lesion.

Soylu *et al.* [15] investigated the diagnostic value of genital SSR and hand SSR in type 1 diabetic children and its association with bladder dysfunction. He suggested that the SSR test, particularly genital SSR, is a noninvasive approach for the assessment of DC, and may be of significance during the early asymptomatic period of DC.

Thus, we can conclude that hand, genital, and foot SSR had almost the same sensitivity and specificity in group I; thus, any one can be used in early diagnosis, whereas in group II, all had equal sensitivities but different specificities, where hand SSR had 96.1%, genital SSR had 66.6%, and foot SSR had 33.3%. We may conclude that SSR is the best in aiding the detection of DC.

In agreement with these results, Ueda *et al.* [29] confirmed the strong association between bladder dysfunction and hand and foot SSRs abnormalities in adult diabetic patients as well as its diagnostic significance in DC. The explanation is that bladder symptoms should not be attributed to peripheral neuropathy unless there are other features of autonomic involvement in DM, that is, to test the function of myelinated sensory fibers, complex central autonomic connections, myelinated preganglionic, and nonmyelinated postganglionic thoracolumbar sympathetic fibers innervating the perineal skin by recording SSR [30].

In our study, there was a statistically significant correlation between urodynamic findings and SSEP P40 in both groups of studied patients.

This is in agreement with Rapidi *et al.* [14], who concluded that there is a possible contribution of central nervous system dysfunction in the pathogenesis of DC, mainly in conjunction with peripheral neuropathy. They concluded that tibial SSEP is well correlated to abnormal urodynamic in diabetic patients with and without LUTD/LUTS.

In our study, we also recorded SSEP P40 sensitivity and specificity in group I: 33.3% sensitivity and 91.6% specificity, and in group II: 75 and 100%, respectively. SSEP P40 of tibial nerve shows a significant correlation to DC. The tibial nerve is a mixed sensory-motor nerve that contains fiber originating from spinal roots L4 through S-3. It comprises the outflow of the sacral nerves that modulate the somatic and autonomic nervous supply to the pelvic floor, innervating directly the bladder, urinary sphincter, rectum, and anal sphincter, because of the fact that normal spinal reflexes of micturition have an afferent limb through S1, S2 roots. The existence of these reflexes provides a possible connection point between the neural pathways explored by tibial SSEP and bladder function [31] (quoted from La Portilla *et al.* [32]).

Accordingly, SSEP P40 of the tibial nerve remains the most specific electrophysiological test of urodynamic diagnostic diabetic dysfunction.

We investigated the most specific parameters of UDS related to diabetic bladder dysfunction (first desire to void–bladder capacity–residual urine volume) and correlated these parameters with different electrophysiological tests; we found that residual urine volume correlated with genital SSR, both latency and amplitude, in the early and late stages of DC. Thus, genital SSR (latency, amplitude) can be used in the screening and follow-up of DC. In contrast, correlations of foot SSR and SSEP P40 were only recorded in the early stage. Therefore, these two tests cannot be used in follow-up of disease progression. Moreover, the residual urine volume correlated with the common peroneal nerve conduction velocity, sural amplitude only in late stages of DC, and thus it cannot be used in early detection or screening.

Thus, hand, foot, and genital SSR are significantly correlated with DC; however, genital SSR is superior because it is sensitive and specific, and can be used in early detection and screening as well as for follow-up.

Conclusion

Electrophysiological studies offer an easy, valid, and noninvasive objective method for the evaluation of DC and sexual dysfunction. Genital SSR is an objective parameter that has clinical acceptance for the evaluation of DC and sexual dysfunction and can be used to follow up disease progression. SSEP of tibial nerve and sural nerve latency are also reliable measures for DC; however, SSEP of tibial is more specific.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

References

1 Edwards JL, Vincent AM, Cheng HT, Feldman EL. Diabetic neuropathy: mechanisms to management. Pharmacol Ther 2008; **120**:1–34.

2 Koubaa S, Ben Salah FZ, Miri I, Ghorbel S, Lebib S, Dziri C, Rokbani L. Neurogenic bladder in diabetes mellitus. Tunis Med 2009; **87**: 279–282.

3 Burakgazi AZ, Alsowaity B, Burakgazi ZA, Unal D, Kelly JJ. Bladder dysfunction in peripheral neuropathies. Muscle Nerve 2012; **45**:2–8.

4 Gomez CS, Kanagarajah P, Gousse AE. Bladder dysfunction in patients with diabetes. Curr Urol Rep 2011; **12**:416–426.

5 Yenilmez A, Kebapci N, Isikli B, Hamarat M, Donmez T. Morbidity after urodynamic study in diabetic patients. Acta Diabetol 2008; **46**:197–202.

6 Weiss L, Silver J, Weiss J. *Easy EMG. A guide to performing nerve conduction studies and electromyography*. Butterworth- Heinemann/Elsevier Inc.; 2004.

7 Soliman E. 2005. Diabetic neuropathy. [cited March 27, 2012]; Available at: *htpp//www.emedicine-diabetic neuropathy article by Emad soliman, MD, Msc.htm.* [Accessed 1 July 2012].

8 American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2006; **29**:543–548.

9 Abrams B, Cardozo L, Fall M. The standardization of terminology of lower urinary tract function. Neurourol Urodyn 2002; **21**:167–178.

10 Rosen R, Browen C, Leiblum S, Meston C, Shabsigh R, Ferguson D, Dagostino R. The female sexual function index (FSFI): a multidimensional self-report instrument for the assessment of female sexual function. J Sex Marital Ther 2000; **26**:191–208.

11 Kucera P, Goldenberg Z, Kurca E. Sympathetic skin response: review of the method and its clinical use. Bratisl Lek Listy 2004; **105**:108–116.

12 Pfisterer MH, Griffiths DJ, Rosenberg L, Schaefer W, Resnick NM. Parameters of bladder function in pre-, peri- and postmenopausal

continent women without detrusor overactivity. Neurourol Urodyn 2007; **26**:356–361.

13 Daneshgari F, Liu G, Birder L, Hanna-Mitchell AT, Chacko S. Diabetic bladder dysfunction: current translational knowledge. J Urol 2009; **182 (6 Suppl):** S18–S26.

14 Rapidi CA, Karandreas N, Katsifotis C, Benroubi M, Petropoulou K, Theodorou C. A combined urodynamic and electrophysiological study of diabetic cystopathy. Neurourol Urodyn 2006; **25**:32–38.

15 Soylu A, Akinci A, Yilmaz U, Sarier M, Aslan M, Ozcan C. Sympathetic skin responses in type-1 diabetic children: relationship to urodynamic findings. Neurourol Urodyn 2006; **25**:243–248.

16 Kebapci N, Yenilmez A, Efe B, Entok E, Demirusutu C. Bladder dysfunction in type 2 diabetic patients. Neurourol Urodyn 2007; **26**:814–819.

17 Esteghamati A, Rashidi A, Nikfallah A, Yousefizadeh A. The association between urodynamic findings and microvascular complications in patients with long-term type 2 diabetes but without voiding symptoms. Diabetes Res Clin Pract 2007; **78**:42–50.

18 Bansal R, Agarwal MM, Modi M, Mandal AK, Singh SK. Urodynamic profile of diabetic patients with lower urinary tract symptoms: association of diabetic cystopathy with autonomic and peripheral neuropathy. Urology 2011; **77**:699–705.

19 Gerstenberger EP, Rosen RC, Brewer JV, Meston CM, Brotto LA, Wiegel M, Sand M. Sexual desire and the female sexual function index (FSFI): a sexual desire cutpoint for clinical interpretation of the FSFI in women with and without hypoactive sexual desire disorder. J Sex Med 2010; **7**:3096–3103.

20 Takahashi M, Inokuchi T, Watanabe C, Saito T, Kai I. The female sexual function index (FSFI): development of a Japanese version. J Sex Med 2011; **8**:2246–2254.

21 Abu Ali RM, Al Hajeri RM, Khader YS, Shegem NS, Ajlouni KM. Sexual dysfunction in Jordanian diabetic women. Diabetes Care 2008; **31**: 1580–1581.

22 Esposito K, Maiorino MI, Bellastella G, Giugliano F, Romano M, Giugliano D. Determinants of female sexual dysfunction in type 2 diabetes. Int J Impot Res 2010; **22**:179–184.

23 Wallner LP, Sarma AV, Kim C. Sexual functioning among women with and without diabetes in the Boston Area Community Health Study. J Sex Med 2010; **7**:881–887.

24 Fatemi SS, Tachavi SM. Evaluation of sexual function in women with type 2 diabetes mellitus. Diabetes Vasc Dis Res 2009; **6**:38–39.

25 Secil Y, Özdedeli K, Altay B, Aydoğdu I, Yilmaz C, Ertekin C. Sympathetic skin response recorded from genital region in normal and diabetic women. Neurophysiol Clin 2005; **35**:11–17.

26 Seçil Y, Yetimalar Y, Gedizlioglu M, Arpaci E, Tokuçoglu F, Inceoglu Kendir A, *et al*. Sexual dysfunction and sympathetic skin response recorded from the genital region in women with multiple sclerosis. Mult Scler J 2007; **13**: 742–748.

27 Kaplan SA, Blaivas JG. Urodynamic findings in patients with diabetic cystopathy. J Urol 1995; **153**:342–344.

28 Rodic B, Curt A, Dietz V, Schurch B. Bladder neck incompetence in patients with spinal cord injury: significance of sympathetic skin response. J Urol 2000; **163**:1223–1227.

29 Ueda T, Yoshimura N, Yoshida O. Diabetic cystopathy: relationship to autonomic neuropathy detected by sympathetic skin response. J Urol 1997; **157**:580–584.

30 Podnar S. Neurophysiology of the neurogenic lower urinary tract disorders. Clinl Neurophysiol 2007; **118**:1423–1437.

31 McGuire EJ, Shi-Chun Z, Horwinski ER. Treatment of motor and sensory detrusor instability by electrical stimulation. J Urol 1982; **129**:78–79.

32 La Portilla F, Rada R, Vega J, Gonzalez CA, Cisneros N, Maldonado VH. Evaluation of the use of posterior tibial nerve stimulation for the treatment of fecal incontinence: preliminary results of a prospective study. Dis Colon Rectum 2009; **52**:1427–1433.