RESEARCH ARTICLE

Accuracy of neurophysiological tests in patients with neurological diseases and pelvic floor symptoms: which tests for which patients?

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Abstract

Aims. To evaluate the sensitivity (SE), specificity (SP), positive predictive value (PPV), and negative predictive value (NPV) of neurophysiological tests in patients with neurological diseases and pelvic floor symptoms.

Methods.
Sixty-four of 111 outpatients who came to our attention for pelvic floor symptoms presented with neurological disorders (32 lower motor neuron disease [LMND], 30 upper motor neuron disease [UMND], 2 with mixed clinical picture). Forty-seven patients with chronic pelvic pain served as controls. All underwent neurological/perineal clinical evaluation and a battery of neurophysiological tests: concentric-needle electromyography (CNEMG) of external anal sphincter (EAS) muscle, pudendal evoked potentials (pPEPs), sacral reflexes, and perineal sympathetic skin response (pSSR). Upper and lower limits of normal values were collected and compared to patients' data. SE, SP, PPV, and NPV were calculated for each neurophysiological exam.

Results.
CNEMG of the EAS showed the highest SE, PPV, and NPV compared with the other tests in LMND; also in UMND, CNEMG of the EAS showed moderate SE and different patterns of abnormality. Combination of sacral reflexes and CNEMG increased the SE of single electrophysiological tests in LMND and UMND. PSEPs were altered in half of the patients with LMND and demonstrated high SE in UMND. PSSR had moderate SE in UMND but the lowest SP and PPV in patients with LMND or UMND.

Conclusions.
Targeted protocols including diverse neurophysiological tests should be chosen on the basis of neurological conditions and level of damage, while other tests should be used for research purposes.

Key-words: Pelvic floor neurophysiology**,** Electromyography, Sacral reflexes**,** Pudendal evoked potentials**,** Perineal sympathetic skin response

1. Introduction

Pelvic floor muscles are involved in complex mechanisms that ensure correct urinary, anorectal, and sexual function. Neural control of the pelvic floor relies on multiple interactions and coordination between the central and peripheral nervous systems.[1] The complexity of the neural system is reflected in the peculiar neuroanatomy of the pelvic floor.[1] Its functional integrity can be studied by means of electrodiagnostic methods [2] specifically designed to test specific body neural districts, localize pathological processes, and possibly reveal the underlying mechanisms and severity.[3]

Standardized methods are encouraged not only to have uniform reproducible techniques but also to obtain normative data that can be utilized across different laboratories.[3] Accordingly, a neurophysiological test battery should be tested for its sensitivity and specificity in evaluating diverse pathologies and supported by appropriate correlations with the patient's clinical background, a challenging goal rarely accomplished in clinical practice. [3,4]

In patients with pelvic floor disorders associated with well-defined neurologic diseases, neurophysiology testing may yield valuable information about lesion site, pathophysiological processes, prognosis of the disease, and sometimes the efficacy of treatment. [5] The aim of the present study was to evaluate the sensitivity, specificity, positive and negative predictive values of a neurophysiological battery in patients affected by upper and lower motor neuron diseases (UMND and LMND, respectively) and pelvic floor symptoms.

2. Materials and methods

2.1 Patients

Between 2010 and 2016, 64 out of 111 outpatients who came to our attention for pelvic floor symptoms presented with peripheral or central nervous system diseases diagnosed either clinically or with radiological support: 32 (18 males, 14 females; mean age 53 years) were affected by peripheral or lower motor neuron disease (LMND) (cauda equina syndromes in 23, pudendal plexopathies in 4, and polyneuropathies in 5); 30 (14 males, 16 females; mean age 52 years) were affected by central or upper motor neuron disease (UMND) (spinal cord disease in 15 and cerebral disease in 15); 2 male patients exhibited a mixed picture (a 69 year-old male patient with traumatic dorsolumbar myelopathy and a 53 year-old male patient with arteriovenous fistula at T12) (Table 1).

Table 1. Clinical data

Peripheral group (LMND) [a]	**No. of patients**
Lumbar vertebral column stenosis	4
Cauda equina syndrome due to herniated lumbar disc	2
Post-surgical cauda equina	9
Post-traumatic cauda equina	5
Cauda equina syndrome in conus-cauda tumor	2
Cauda equina after acute polyradiculoneuritis	1
Post-traumatic pudendal plexopathy	3
VZV [b] radiculoplexopathy	1
Alcoholic polyneuropathy	2
Idiopathic axonal sensory neuropathy	3
Central group (UMND) [c]	
Post-traumatic cervical myelopathy	3
Cervical myelopathy due to cervical spondylosis	3
Thoracic myelopathy due to herniated disc at level T9-T10	1
Thoracic myelopathy due to dorsal spondylosis	7
Tethered cord, lumbosacral lipoma	1
Parkinson's disease	2
Multiple sclerosis	3
Suspected MSA [d]	3
Cognitive impairment	3
Cranial frontal trauma	1
Oligophrenia	1
Leukodystrophy	1
Hydrocephalus	1
Mixed group (UMND + LMND)	
Traumatic thoracolumbar spinal cord lesion	1
T12 Arteriovenous fistula	1

Legend to Table 1

[a] LMND: lower motor neuron disease

[b] VZV: varicella zoster virus

[c] UMND: upper motor neuron disease

[d] MSA: multisystem atrophy

Controls for this study were 47 patients with chronic pelvic pain without neurological clinical signs or other symptoms. The study was approved by the Local Ethical Committee of Azienda Ospedialiera Universitaria Integrata of Verona and written informed consent was obtained from all subjects. All subjects underwent neurological/perineal clinical evaluation and neurophysiological tests.

2.2. Neurophysiological tests

A neurophysiological test battery was performed using a Keypoint apparatus (Dantec, Denmark).

2.2.1 Concentric needle EMG (CNEMG): the subcutaneous part of the external anal sphincter (EAS) muscle was assessed bilaterally according to Podnar's technique [6] for qualitative evaluation of tonic and reflex activity, presence of denervation, motor unit potentials (MUPs) morphology and type of recruitment; quantitative electromyography (QEMG) with automatic multi-MUP analysis was carried out in uncertain cases. Concurrent EMG recording of the EAS muscle and rectus abdominis (RA) muscle and qualitative evaluation of their activity in both resting state and straining were performed in patients presenting at conventional EMG with a lack of inhibition of EAS during straining or complaining of constipation/difficult evacuation. Band-pass was fixed at 20-10 kHz.

2.2.2 Bulbocavernosus/clitoro-cavernosus reflex (BCR): bipolar stimulation of the dorsal penile/clitoral nerves (cathode at the midline at the base of penis shaft or/on the clitoris and anode 2 cm distal for men or 2 cm away from the cathode between the labia majora and minora for women) was performed using single or paired pulses of 0.1 msec duration and an interstimulus interval (ISI) of 3 msec; the concentric needle was positioned in the bulbocavernosus (BC) muscle. Band-pass was set at 20 Hz-10 kHz.

2.2.3 Pudendo-anal reflex (PAR): bipolar stimulation of the dorsal penile/clitoral nerves (single or paired pulses of 0.1 msec duration and 3 msec ISI) was performed for needle recording at the subcutaneous part of the EAS. Band-pass was set at 20-10 kHz.

2.2.4 Pudendal evoked potentials (pSEPs): pSEPs were obtained using bipolar stimulation with pre-gelled surface electrodes positioned above the dorsal penile/clitoral nerves (cathode at the midline at the base of penis shaft or/on the clitoris and anode 2 cm distal for men or 2 cm away from the cathode between the labia majora and minora for women); scalp recording was obtained with monopolar needles at Cz' (active) and Fz (reference) according to 10-20 International Recording System; stimuli of 0.1 msec duration and intensity of 3x sensory threshold were delivered; final recording was obtained after an average of 200 stimuli. Band-pass was set at 10 Hz-3 kHz.

2.2.5 Perineal sympathetic skin response (pSSR): stimuli of 0.1 msec duration were applied at the second digit of the left hand at an intensity of 5x the sensory threshold; recording

was obtained with pre-gelled surface electrodes at the right hand palm, feet sole, and perineum (at penis/ labia minora with reference at groin). Band-pass was set at 0.5 Hz-2 kHz.

Upper and lower limits of control values were collected and compared to patients' data. Normal limits of the neurophysiological data were set at mean ± 2 standard deviation (M ± 2 SD) for latencies (as they showed Gaussian distribution) and 95th percentile of upper and lower limits for amplitudes.

CNEMG was considered pathological if the qualitative study showed neurogenic signs or the QEMG was abnormal according to Podnar's published criteria (upper/lower limits: M ±2 SD for mean values, and 95th percentile of the third highest and lowest values of 20 MUPs for detection of outliers).[7] Pathological criteria were: reduced recruitment due to decreased central activation, as well as muscular overactivity (e.g., involuntary EAS contraction) or dyssynergic pattern (lack of EAS inhibition during straining or co-contraction of EAS and abdominal wall muscles during straining). In the patients' group, latency/amplitudes above/below normal limits or the absence of neurophysiological signals were considered abnormal.

Sensitivity, specificity, positive and negative predictive values for each neurophysiological exam were calculated. Test sensitivity (SE) (%) was calculated as the number of patients with an abnormal test/number of evaluated patients *100. Test specificity (SP) (%) was calculated as the number of controls with a normal test/number of evaluated controls* 100. The positive predictive value (PPV) (%) was established as the number of patients with an abnormal test/number of evaluated subjects with abnormal test *100. The negative predictive value (NPP) (%) was defined as the number of controls with a normal test/number of evaluated subjects with normal test *100. Cohen's *k* agreement test was used to determine the relationship between the presence of abnormal electrophysiological data and patients' symptoms. Statistical analyses were performed using SPSS (Statistical Package for Social Sciences, IBM-SPSS, Armonk, NY, USA). The significance level was set at $P \leq 0.05$.

3. Results

Neurophysiological values for the control group are summarized in Table 2. The neurophysiological tests results are presented in Tables 3 and 4.

Table 2. Reference values from control group

Reference values	**BCR** [a]	**pSEPs** [b]	**pSSR** [c]	**PAR** [d] **Right side**	**PAR Left side**
Latency (msec)					
Mean (M)	35.3	39.3	1734.2	39.6	**38.5**
Standard Deviation (SD)	4.8	4	404.7	4.4	4.8
Upper Limit (M+2SD)	**44.9**	**47.4**	**2543.6**	**48.3**	**48.1**
Amplitude (uV)					
Median	Not considered	1.1	324	Not considered	Not considered
Lower Limit (95th percentile)	Not considered	**0.36**	**143.1**	Not considered	Not considered

Legend to Table 2

[a] BCR: bulbocavernosus reflex

[b] pSEPs: pudendal somatosensory evoked potentials

[c] pSSR: perineal sympathetic skin response

[d] PAR: pudendo-anal reflex

Table 3. Sensitivity, specificity, positive and negative predictive values of neurophysiological tests

		BCR [g]	**pSEPs** [h]	**pSSR** [i]	**PAR** [l]	**CNEMG** [m]
LMND [a]						
Patients	32 (+2 mixed)					
	SE [c] %	60	50	52	60	67
	PPV [e] %	100	80	62	95	100
	NPV [f] %	69	67	56	77	81
UMND [b]						
Patients	30 (+2 mixed)					
	SE %	31	95	69	36	63 (mixed pattern)
	PPV %	100	82	58	92	100
	NPV %	45	69	75	67	80
	SP [d] %	100	89	65	98	100

Legend to Table 3

[a] LMND: lower motor neuron diseases

[b] UMND: upper motor neuron diseases

[c] SE: sensitivity

[d] SP: specificity

[e] PPV: positive predictive value

[f] NPV: negative predictive value

[g] BCR: bulbocavernosus reflex

[h] pSEPs: pudendal somatosensory evoked potentials

[i] pSSR: perineal sympathetic skin response

[l] PAR: pudendo-anal reflex

[m] CNEMG: concentric needle electromyography

3.1 CNEMG

CNEMG of the EAS had the highest PPV, NPV (100% and 81%, respectively), and the highest SE (67 %) as compared with the other tests in LMND. In UMND, CNEMG showed diverse abnormal patterns: 6 patients (2 with cognitive impairment, 1 with leukodystrophy, 1 with Parkinson's disease (PD), 1 with thoracolumbar traumatic myelopathy, 1 with dorsal arteriovenous fistula) disclosed reduced EMG activation, 4 patients displayed a neurogenic pattern (3 with suspected multisystem atrophy (MSA) and 1 with tethered cord associated with lumbosacral lipoma); 4 others (1 with PD, 1 with multiple sclerosis (MS), and 2 with myelopathy) presented a mixed pattern (reduced EMG activity together with neurogenic MUPs); 6 patients displayed muscular overactivity: 3 with thoracic myelopathy and 2 with cervical myelopathy presented a dyssynergic pattern (Fig. 1a), while 1 oligophrenic patient exhibited involuntary EAS contraction (Fig. 1b). The overall SE was 63%.

Table 4. Sensitivity, specificity, positive and negative predictive values of combined CNEMG and sacral reflexes

		BCR [g] +PAR [h]	**BCR + PAR + CNEMG [i]**
LMND [a]			
Patients	32 (+ 2 mixed)		
	SE [c] %	60	73
	PPV [e] %	95	96
	NPV [f] %	78	84
UMND [b]			
Patients	30 (+ 2 mixed)		
	SE %	39	75 (mixed pattern)
	PPV %	92	96
	NPV %	69	85
	SP [d] %	100	100

Legend to Table 4

[a] LMND: lower motor neuron diseases

[b] UMND: upper motor neuron diseases

[c] SE: sensitivity

[d] SP: specificity

[e] PPV: positive predictive value

[f] NPV: negative predictive value

[g] BCR: bulbocavernosus reflex

[h] PAR: pudendo-anal reflex

[i] CNEMG: concentric needle electromyography

3.2 Sacral reflexes

BCR and PAR had moderate sensitivity (60%) and high PPV (100% and 95%, respectively) in LMND, whereas in UMND sensitivity was low (31% for BCR, and 36% for PAR) but PPV high (100% for BCR, and 92% for PAR). Combination of the two methods (sacral reflexes and CNEMG) increased the SE of single electrophysiological tests from 60 to 73% in LMND and from 39 to 75% in UMND (Table 4).

3.3 PSEPs

PSEPs were altered in half of the patients with LMND and showed a high PPV (80%); in those with UMND, pSEPs demonstrated high SE (95%) and a high PPV (82%).

3.4 PSSR

Also pSSR was abnormal in half of the patients (52%) and had a moderate PPV (62%) in LMND; conversely, it had moderate SE in UMND (abnormal in 69%) but lower PPV (58%). Overall, SP was generally high (≥80%) in all tests except for pSSR (65%). There was no correlation between neurophysiological data (considered as pathological) and the presence of pelvic floor symptoms.

4. Discussion

We found moderate sensitivity for CNEMG in LMND. This finding is congruous with published data demonstrating that CNEMG may detect neurogenic signs in sacral neuropathic lesions.[8] Also quantitative EMG (QEMG) is widely used in clinical practice, although the normative limits are not unequivocally stated because of the variable sensitivity and specificity that different sets of electrophysiological diagnostic criteria may produce.[9] Intriguingly, we found diverse EMG patterns in UMND: 6 of the 20 patients with abnormal CNEMG disclosed reduced activation at maximal effort, typical of central disorders, while 4 showed neuropathic signs: one with mixed upper and lower motor neuron syndrome (a patient with tethered cord associated with lumbosacral lipoma) and 3 with suspected MSA. Studies have largely confirmed the presence of neurogenic MUPs at sphincter EMG in patients with a clinical diagnosis of MSA, with sensitivity varying from 20 to 100%;[10,11] nevertheless, this electrophysiological finding is not useful for distinguishing MSA from PD or other parkinsonisms, even at earlier phases of disease.[12]

Four patients, 3 of which with suprasacral lesions, disclosed a mixed pattern (reduced EMG activity together with neurogenic MUPs): this result is not an electrophysiological 'paradox', as neurogenic MUPs in spinal cord lesions have been described and the resulting axonal damage has been interpreted as being caused by transynaptic degeneration.[13]

Five patients with constipation and cervical/thoracic myelopathies displayed a dyssynergic pattern (Fig. 1a), while one patient with oligophrenia and fecal incontinence disclosed EAS overactivity characterized by involuntary EAS contraction (Fig. 1b).

FIGURE 1a and 1b

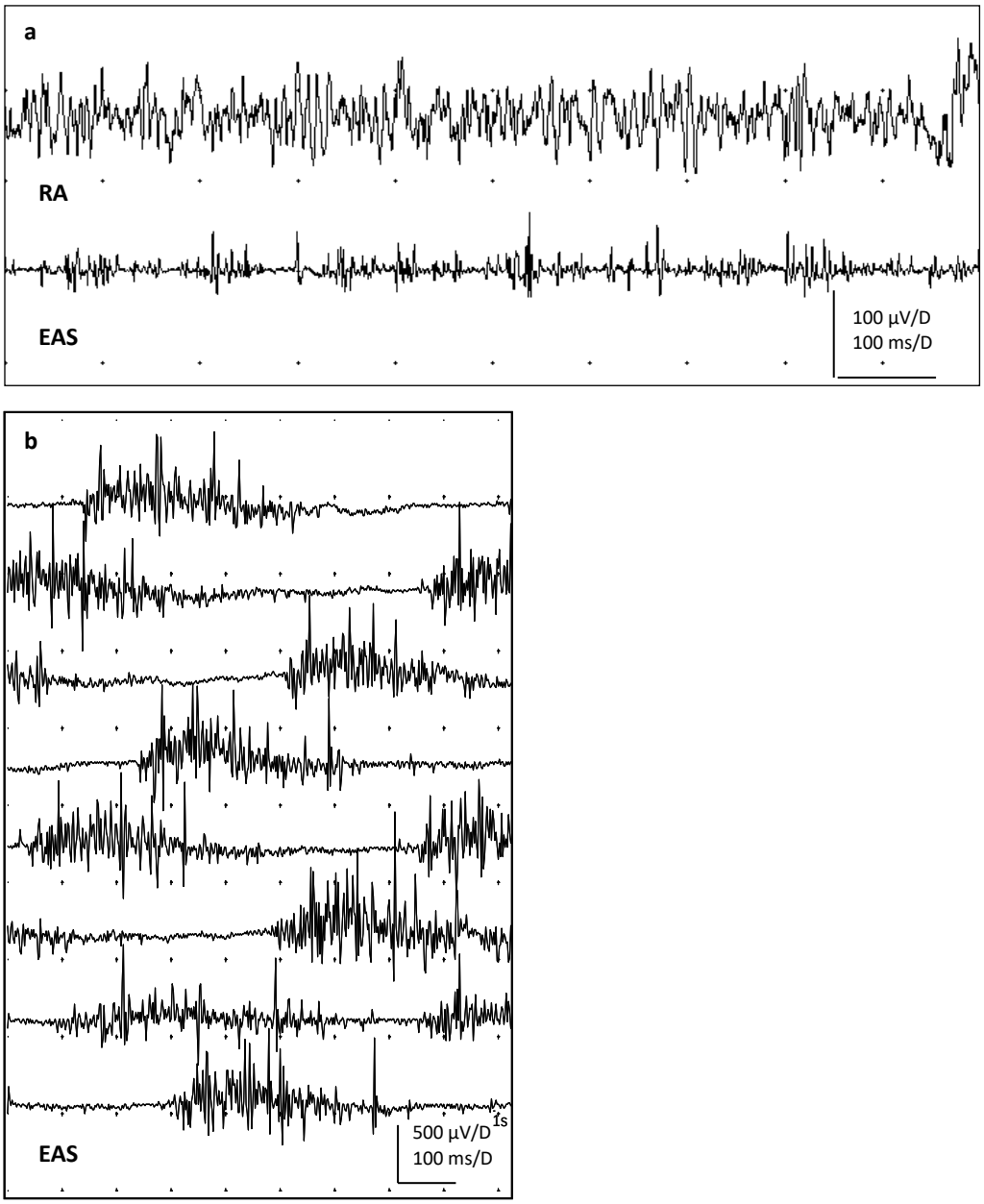

Figure 1a: Rectus abdominis (RA) and external anal sphincter (EAS) muscles during straining.

Figure 1b: External anal sphincter (EAS) muscle involuntary EMG burst at rest.

Though a lack of puborectalis inhibition or paradoxical activation of pelvic floor muscles during squeezing has been found in obstructive outlet syndrome with high sensitivity,[14] this finding has low specificity because increased EMG activity of the puborectalis muscle during straining at stool has been described in 25 to 50% of patients with chronic pelvic pain.[15] While pelvic EMG has been found to have a weaker correlation with constipation than defecography, which remains the gold standard diagnostic test,[16] EMG is considered a supportive test [17] in patients with constipation and inconclusive gastroenterological assessment.[15]

Although our sample was too small to draw any firm conclusions, we can state that patients with UMND and fecal incontinence or constipation can present muscular or dyssynergic overactivity that may correlates with clinical symptoms.

Sacral reflexes were altered in the patients with LMND, consistent with published data that demonstrated a high sensitivity of BCR in LMND.[18,19] We also found that combination of EMG and sacral reflexes increased test sensitivity in patients with LMND. This observation is shared by Podnar,[20,21] though he included only BCR and not PAR in his study.

Conversely, sacral reflexes were altered in a low percentage of cases in UMND. Although some authors have reported either decreased latency [22] or threshold,[23] others have described an absent or delayed response of sacral reflexes, similar to peripheral disorders.[24] These divergent findings were interpreted as being due to altered supraspinal control upon spinal cord interneurons, otherwise due to impairment of the sacral reflex arc.[25]

Concerning pSEPs, we found a higher percentage of alterations in patients with UMND than in those with LMND. While the literature is unanimous on pSEP alterations in UMND, even those of different origin (such as MS or spinal cord injury),[26,13] evidence is uneven for LMND: in cauda equina disease, pSEPs are reported as consensually altered,[19,27] while results are heterogeneous for polyneuropathies and mainly dependent on the origin of the neuropathy.[28,29] The reason for this discrepancy may be that the spinal cord damage is relatively independent of the cause but mostly related to lesion site and type (complete or partial), whereas pSEPs findings in polyneuropathies presumably rely upon the involvement of sensory myelinated fibers. It would be interesting to compare the SE of SEPs from the tibial nerve and pSEPS, as some authors have demonstrated a higher sensitivity of lower limb SEPs than pSEPs in patients with spinal cord lesion or MS.[30,31] This discrepancy may be due to the fact that tibial SEPs have a unilateral pathway in the spinal cord, while

pSEPs have a bilateral pathway and may be less sensitive.[31]

PSSR showed moderate SE in UMND, in line with previous findings (Schmid et al., 2003),[32,33] while its sensitivity was lower in LMND. Evidence in this regard is not uniform: abnormal pSSR has been described in cauda equina lesions [34] and familial amyloidotic polyneuropathy [29], but in other causes of polyneuropathy (such as diabetic or alcoholic) the results are dishomogeneous. [35-38] Such dissimilar results may depend not only on the different types of peripheral neuropathies involving somatic sensory large diameter or autonomic small diameter fibers, but also on the different techniques employed, such as site of stimulation and recording (genital area or median nerve for stimulation rather than hand-palm, foot-sole or perineum for recording).[29,37] Although any comparison is difficult owing to the lack of information about test specificity of pSSR described in other studies, our data show a low specificity, as pSSR could be absent also in normal conditions, so that the overall accuracy of the test may be questioned.

No correlation between symptoms and neurophysiological data was found, as neurophysiology has been reported to reveal subclinical involvement in asymptomatic patients with urinary symptoms [39] or have a limited diagnostic utility in patients with erectile dysfunction or anorectal disorders.[26,40,41]

5. Conclusions

The utility of pelvic floor neurophysiology is well recognized and supported by the evidence. Targeted protocols including diverse neurophysiological tests should be chosen on the basis of neurological conditions and level of damage, while other tests should be used for research purposes. CNEMG of the EAS and sacral reflexes had the highest sensitivity, specificity, and PPV in both LMND and UMND, and should therefore be utilized in combination with sacral reflexes. We wanted to highlight the importance of CNEMG also in central disorders, as different abnormal neurophysiological patterns may be found, from reduced activation to mixed central and neuropathic features or dyssynergic activity, that may correlate with anorectal symptoms in certain cases.

Conversely, pSEPs demonstrated low-moderate sensitivity in LMND (depending on the cause of peripheral involvement) and high sensitivity in UMND, so that their assessment may be tailored while relying upon diagnostic clues. Sacral SSR showed moderate sensitivity in UMND, but is nonspecific, as it may be altered also in healthy subjects so that it may be of some interest for research purposes.

Our study has the limitation of a small patient sample. Further studies with larger samples of patients with specific neurological diseases are needed to accurately investigate SE, SP, and predictive values for single neurological diseases.

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