A Novel Electrophysiological Method in the Diagnosis of Pudendal Neuropathy: Position-related Changes in Pudendal Sensory Evoked Potentials

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OBJECTIVE To examine the diagnostic value of pudendal somatosensory evoked potentials (SEPs) in pudendal nerve entrapment (PNE) neuropathy by stimulating the 2 sides separately after provocation by a standard sitting position. Routine pudendal SEPs performed in the supine position with bilateral simultaneous stimulation may fail to show the abnormality because the complaints of PNE appear or worsen in the sitting position.

METHODS Forty-nine patients with PNE and 16 controls were included. SEP recordings were performed by stimulating the dorsal nerve of penis or clitoris on either side. The recordings were performed at the initial supine position, at the beginning and end of the provocation by sitting position, and at the second supine position.

RESULTS Amplitude loss in the SEP responses after prolonged sitting was significantly more pronounced on the symptomatic sides of the patients. Approximately 45% decrease in the SEP amplitude or an amplitude value less than 1.5 μV at the end of sitting are the parameters to be used with high selectivity.

CONCLUSION The dynamic pudendal SEP study described herein seems to be more helpful in PNE diagnosis than in conventional SEPs. UROLOGY ■■: 1.e1–1.e7, 2016. © 2016 Elsevier Inc.

Pudendal nerves derive from S2-S4 sacral roots and give 3 major branches on either side: inferior anal, and perineal and dorsal nerves of the penis or clitoris.[1-3] Their entrapment has been described in the pudendal (Alcock's) canal, in the vicinity of the ischial spine, sacrospinous and sacrotuberous ligaments, and under the piriformis muscle.[4]

Pudendal nerve entrapment (PNE) is a common cause of chronic resistant perineal pain that generally increases with hip flexion. Patients usually describe it with the characteristics of neuropathic pain as burning, tearing, electrifying, or foreign body sensations. Although some types of trauma and exercises are accused, the etiology of PNE is not yet clearly understood.[4,5] Its clinical diagnosis primarily depends on clinical evidence, mostly on the typical patient history. Surgical decompression of the usual entrapment sites has been described for the cases resistant to conservative management.[6,7]

Neurophysiological tests can be used for the documentation of PNE. These tests consist of bulbocavernous muscle and/or sphincter electromyography, pudendal nerve conduction studies, sacral reflexes, and somatosensory evoked potentials (SEPs).[8,9] SEPs are the responses evoked in the cerebral cortex by electrical stimulation of a sensory nerve or a dermatome. In case of a lesion between the stimulation and recording sites, amplitude loss and/or latency prolongation of the potentials indicate the abnormality.[8,10] Several studies have provided normal values for pudendal SEPs.[11,12] However, evidence about the role of pudendal SEPs in confirming the pudendal entrapment neuropathies is inadequate. Most SEP abnormalities have been reported in patients already known to have severe neurological deficits or structural abnormalities, which were easily identifiable by clinical and/or radiological means.[13-16]

Because the complaints in PNE are completely or partially unilateral and worsen in the sitting position, routine

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SEP studies, which are generally performed in the supine position with bilateral simultaneous stimulation, may fail to show the nerve involvement. The hypothesis of the present study was that the diagnostic accuracy of pudendal SEPs can be increased by stimulating the symptomatic and asymptomatic sides separately before and after the provocation by sitting posture.

METHODS

The study was performed at Yeditepe University Clinical Neurophysiology Laboratory between March 2012 and March 2014, following approval of the local ethics committee, and informed consent was obtained from each subject.

Cases

All the patients were referred for electrophysiological examination by the urology department that treated 186 patients with complaints of chronic pelvic pain during the above-mentioned 2-year period. Forty-nine of these patients (29 females), who were diagnosed with PNE by using Nantes criteria, were referred for the electrophysiological studies.[17] The essentials of these criteria were described as follows: pain (1) in the anatomical territory of the pudendal nerve, (2) predominantly experienced while sitting, (3) does not wake the patient at night, (4) with no objective sensory impairment, (5) and relieved by diagnostic pudendal nerve block.[17] The exclusion criteria were the following: purely coccygeal or gluteal pain, paroxysmal pain, exclusive pruritus, having any systemic disease that could affect the peripheral or central nervous system, having a history of pelvic surgery, presence of imaging abnormalities that could explain the symptoms, and having symptoms or physical signs indicating a manifest urological or neurological abnormality. Complementary criteria, which were described elsewhere, were also used when needed in favor of the diagnosis of pudendal neuralgia.[17] Prior to the study, a complete neurological examination and bilateral tibial SEP studies were performed by one of the authors (BÖ) and gave normal findings in all the patients.

The symptoms were right sided in 20 patients, left sided in 16, and bilateral in 13. Sixteen healthy volunteers (5 females) who did not have any complaints that could be caused by neurological or urological diseases comprised the control group.

Electrophysiological Studies

Electrophysiological studies were performed by using a Keypoint-net (Natus Medical Inc., Middleton, WI) electrophysiology unit.

Stimulation was performed by using bar electrodes with saline-soaked felt pads (diameter: 4 mm, interelectrode distance: 2.5 cm). In the female subjects, the stimulator cathode was placed just laterally to the clitoris on either side, between the minor and major labia, and the anode was placed posteriorly. In the male subjects, the anode was placed just proximally to the corona of the glans on the most lateral side of the penis, with the cathode pointing proximally (Supplementary Fig. S1). The stimulator bar was secured to the position by adhesive tapes (Betafix). The ground electrode was placed on the ipsilateral iliac spine.

Square-wave electrical pulses of 0.2 ms duration were delivered at 2 Hz. The intensity of electrical stimulation was adjusted as 2 times the sensory threshold to avoid stimulating the nerve on the contralateral side. This intensity was well below the pain threshold and easily tolerated.

SEP responses were recorded using 9 mm cup electrodes placed on the points 2 cm behind the Cz and Fpz, according to the international 10-20 system.[18] The frequency filters were between 5 and 3000 Hz. Display sensitivity was 5 μV/division and sweep speed was 10 ms/division.

Each subject was requested to close his/her eyes, open his/her mouth slightly, and stay as relaxed as possible during the recordings. Recordings were performed by stimulating the right and left sides separately.

Three successive SEP recordings composed of at least 350 averaged responses were obtained in the supine position. Then the subject was made to do sitting position by hanging his/her legs down the lateral edge of the examination table without getting support from their arms, while the feet were not touching the ground and the hip and knee joints were flexed about 90 degrees. In the control cases, the duration of the sitting position was decided to be 20 minutes, which had been generally adequate to trigger the pain in most of our previous PNE patients. In the patient group, this period was also 20 minutes if the patient was able to tolerate it. If the patient was not able to maintain the position (because of severe pain or disappearance of the SEP responses), the study was terminated earlier. In the subjects who maintained the position for 20 minutes, approximately 10-12 recordings, each composed of at least 350 averaged responses, were made. If the subjects could maintain shorter sitting periods, fewer recordings could be obtained. Then the subjects were moved into supine position again and 4 consecutive averaged recordings were made. Although the artifacts caused by the muscle activity were rare due to the use of cephalic electrodes, the traces containing occasional muscle artifacts emanating from the neck and pharyngeal muscles were excluded. The study timeline is presented in Figure 1.

In the present study, "T" (ms) value represents the onset latency of the first negative deflection of the cortical response (generally entitled as P1 in pudendal SEP studies). "A" (μV) value represents the amplitude difference between P1 and N1 deflections. The T and A values obtained at the first supine position, at the beginning of the sitting position, at the end of the sitting position and at the end of the second supine position were named as T0, A0; T1, A1; T2, A2, and T3, A3, respectively. (Figs. 1, 2).

In the patients whose SEP responses were completely lost during the sitting position, T2 latency values were excluded and the A2 values were accepted as zero. Latency and amplitude differences caused by the sitting position and lying down again were calculated as T1-T0, T2-T0, T2-T1, T2-T3, T3-T0, and A0-A1, A0-A2, A1-A2, A3-A2,

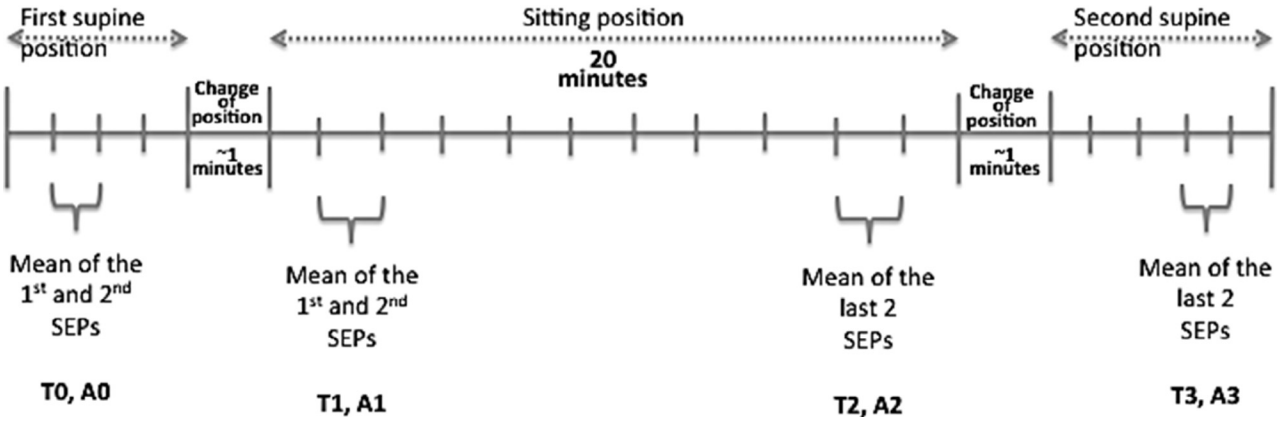

Figure 1. Timeline of the study. The time for "Change of position" includes the time for electrode impedance control. SEP, somatosensory evoked potential.

A0-A3. Amplitude changes caused by positioning were expressed as percentages [difference between the two steps / amplitude of the previous step × 100] due to high variability of the amplitudes between the subjects and relatively small absolute values of the changes.

In the control group, all data obtained from the right and left sides were compared and no statistically significant differences were found (Fig. 3). Therefore, data obtained from the right and left sides were pooled together and used for intergroup comparisons. Thus, 3 groups were formed as (1) controls, (2) symptomatic, and (3) asymptomatic sides of the patient group.

Statistical Analysis

Data were analyzed using SPSS 21.0 (Statistical Packages of Social Sciences) and MedCalc 10.2 (for the receiver operating characteristic [ROC] curves) software. Conformity of data to the normal distribution was assessed by Kolmogorov-Smirnov test. Descriptive statistics were shown as mean ± standard deviation for continuous variables and as frequencies and percentages for categorical variables. The Levene test was performed to assess homogeneity of variance between the groups. For comparison of the mean values of variables in more than 2 independent groups conforming to normal distribution, ANOVA or, when necessary,

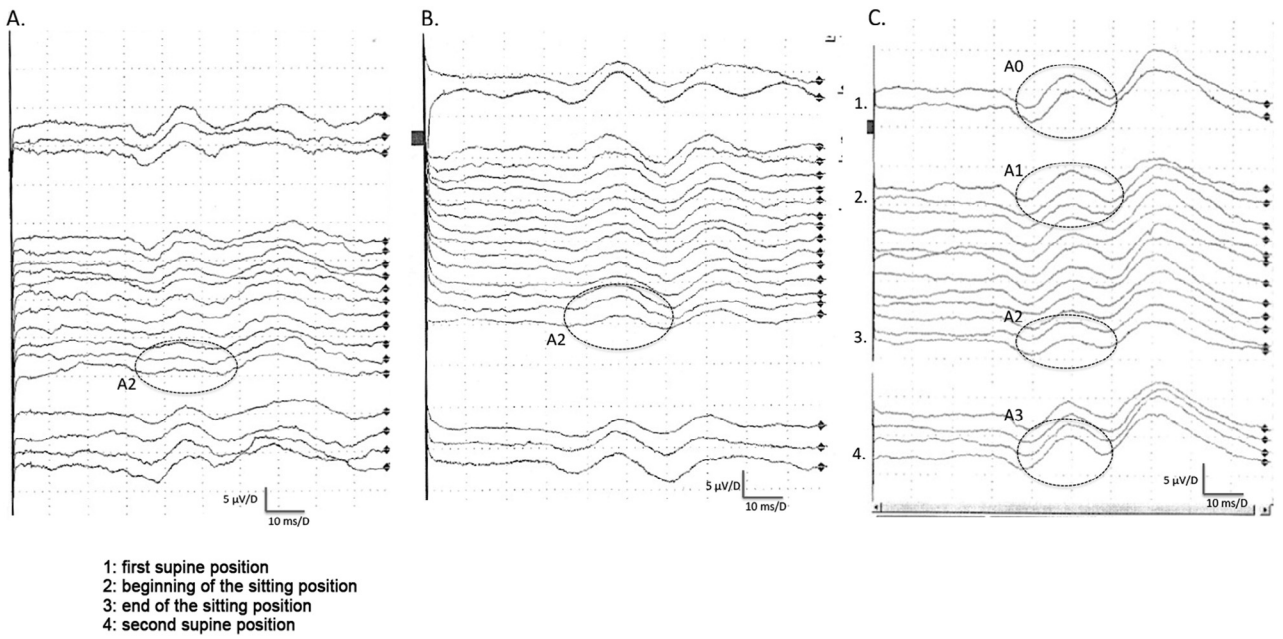

Figure 2. Examples for SEP recordings in each group. Please note that the A2 amplitude decreased severely on the symptomatic side of a patient **(A)**, moderately on the asymptomatic side **(B),** and mildly in a control case **(C)**. 1: Recordings made during first supine position, 2: recordings made during the beginning of the sitting position, 3: recordings made at the end of the sitting position, and 4: recordings made during the second supine position. The changing amplitudes at these positions are marked with A0, A1, A2, and A3. SEP, somatosensory evoked potential.

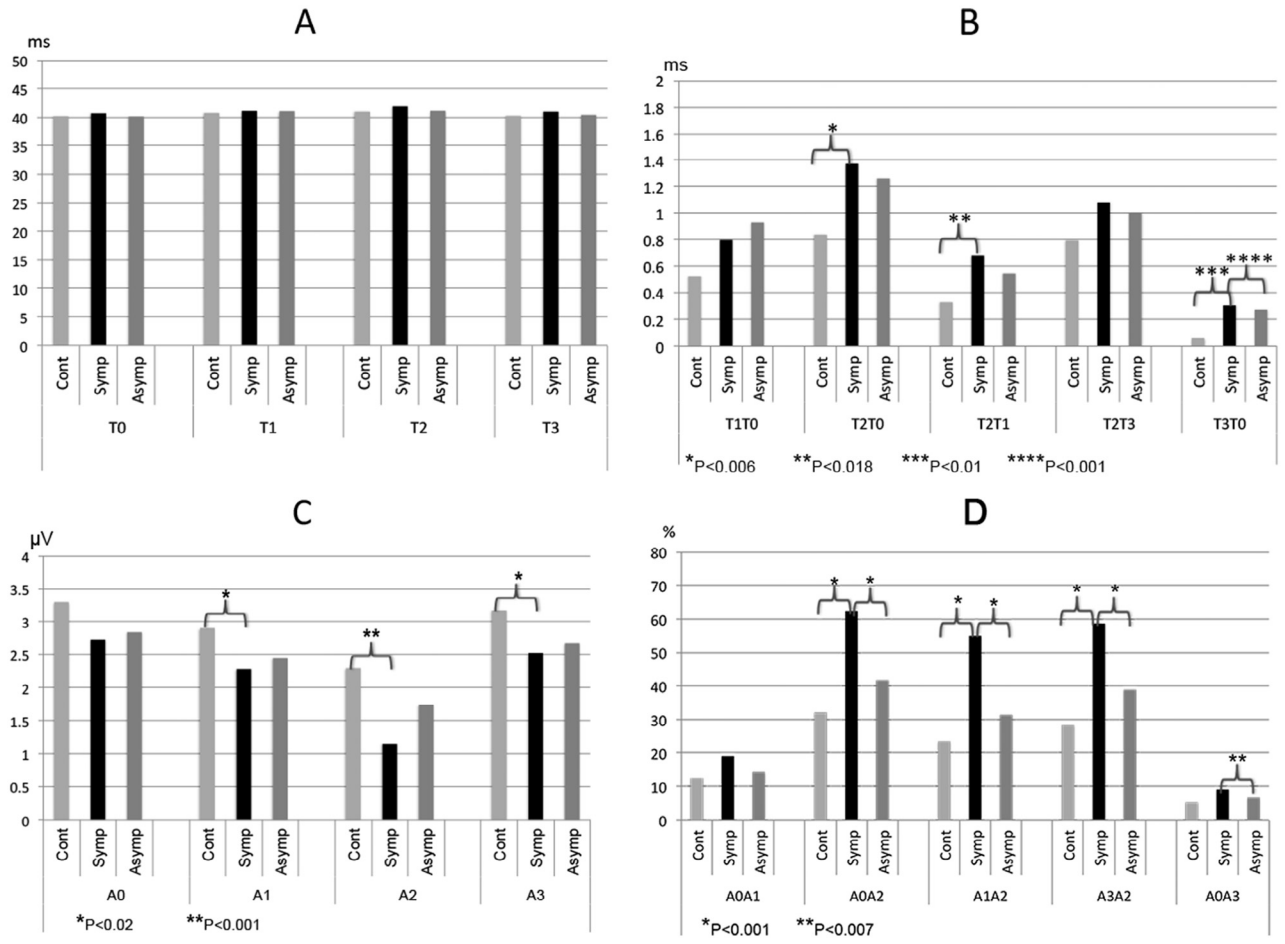

Figure 3. Mean latencies, amplitudes, and positional differences of the pudendal SEP responses found in the groups. **(A)** Latencies at T0, T1, T2, and T3 in the 3 groups; **(B)** positional latency changes; **(C)** amplitudes at A0, A1, A2, and A3; **(D)** positional amplitude changes. Intergroup comparisons: please see the stars at the bottom of each the figure. Although some statistically significant differences were found in the intergroup comparisons for the positional T differences, the absolute mean values of the differences were around 1 ms, which is in the range of recording errors in repeated studies and seems to be technically insignificant for a SEP study. Therefore, these findings were excluded from the conclusion of this study. SEP, somatosensory evoked potential.

the Welch test was used. For variables with significant differences between the groups, Tukey's multiple comparison tests or, when necessary, Dunnett's T tests were used. For comparison of the mean values of variables in more than 2 independent groups not conforming to normal distribution, the Kruskal-Wallis test was used. For variables with significant difference between the groups, the Mann-Whitney *U* test was used. For the estimation of best cutoff points to discriminate the abnormal findings elicited in the patient group from the normal values of the controls, ROC analysis was performed and the area under the curve was calculated.

Assessments by using amplitude values obtained by logarithmic transformation were also made but they provided no additional benefit in statistical analyses.

Post hoc power analysis was done to evaluate the achieved power of the statistically significant findings in the study by using G*Power 3.1 software.

$P < .05$ was considered to be a statistically significant difference.

RESULTS

Demographic data of the case groups are presented in Supplementary Table S1. Although the mean ages of the control subjects were lower than those of the patients, there were no statistically significant age differences between the case groups. Male subjects were significantly taller than the females ($P < .001$).

In the control group, there were no subjects with unobtainable pudendal SEP responses on either side. At the initial supine study, pudendal SEPs could not be obtained unilaterally in 9 patients (on the symptomatic sides [2 left, 7 right]) and bilaterally in 7 patients (bilateral complaints in 5 and right sided pain in 2). The sides with unobtainable responses were excluded from the steps of the

Table 1. Results of the electrophysiological studies and post hoc power analyses

	Controls	Asymptomatic Sides	Symptomatic Sides	Post Hoc Power†
Total number (N)	32	36	62	
Unobtainable SEP in the first supine position	0	2	23	
N for statistics	32	34	39	
T0 (ms)	40.19 ± 1.84	40.13 ± 4.47	40.69 ± 4.37	
T1 (ms)	40.74 ± 1.80	41.07 ± 4.68	41.13 ± 4.44	
T2 (ms)	40.96 ± 1.90	40.14 ± 6.60	41.90 ± 7.51	
T3 (ms)	40.25 ± 1.84	40.40 ± 4.53	40.98 ± 4.37	
T1-T0 (ms)	0.52 ± 0.19	0.93 ± 1.35	0.79 ± 0.64	
T2-T0 (ms)	0.83 ± 0.56	1.26 ± 0.81	**1.37 ± 0.87***	**86%**
T2-T1(ms)	0.32 ± 0.44	0.54 ± 0.59	**0.68 ± 0.64***	**76%**
T2-T3 (ms)	0.79 ± 0.49	1.00 ± 0.72	1.07 ± 0.73	
T3-T0 (ms)	0.05 ± 0.11	0.27 ± 0.32	**0.30 ± 0.32*†**	**99%**
A0 (μV)	3.30 ± 1.32	2.84 ± 0.99	2.73 ± 1.12	
A1 (μV)	2.90 ± 1.22	2.45 ± 0.92	**2.28 ± 1.03***	**93%**
A2 (μV)	2.29 ± 1.07	1.74 ± 0.95	**1.14 ± 1.11***	**99%**
A3 (μV)	3.16 ± 1.27	2.68 ± 0.96	**2.52 ± 1.07***	**72%**
A0-A1 (μV/%)	12.56 ± 4.97	14.30 ± 7.40	**19.06 ± 15.87***	**90%**
A0-A2 (μV/%)	32.06 ± 12.50	41.70 ± 20.39	**62.26 ± 26.61†**	**99%**
A1-A2 (μV/%)	23.55 ± 12.36	31.31 ± 20.74	**54.96 ± 30.34*†**	**99%**
A3-A2 (μV/%)	28.32 ± 13.62	38.84 ± 20.99	**58.64 ± 29.09*†**	**99%**
A0-A3 (μV/%)	5.13 ± 6.52	6.73 ± 6.89	**9.06 ± 9.90*†**	**53%**

SEP, somatosensory evoked potential.
For the meaning of the T and A values, please see the section "Electrophysiological Studies" in the text.
* Statistically significant difference between the symptomatic side of the patients and controls.
† Statistically significant difference between the symptomatic and asymptomatic sides of the patients.
† Post hoc power analyses were performed for the parameters that revealed significant intergroup differences (bold lines).

study (Table 1). At the end of the sitting position, SEP responses became unelicitable on 12 symptomatic sides and 1 asymptomatic side. In the control group, none of the SEP responses became unobtainable during the sitting position.

When the intergroup differences in the T and A values as well as their positional differences were analyzed (please see Table 1 and Fig. 3 for statistical significances), T0 latency was significantly longer in males as compared to females in the control group. When the 3 groups were statistically compared in terms of the T values (latencies), no significant differences were found. When the intergroup comparisons were made in terms of T latency changes, latency prolongation caused by the sitting position, which was the most marked in the symptomatic group, led to significant differences between the control and symptomatic groups in terms of T2-T0, T2-T1, and T3-T0 differences. Between the symptomatic and asymptomatic groups, the difference was statistically significant only for T3-T0.

There was no statistically significant intergroup difference in terms of amplitudes at the initial supine studies (A0). The amplitude loss caused by sitting, which was the most marked in the symptomatic group, led to significant differences between the control and symptomatic groups for A1, A2, and A3 values. Between symptomatic and asymptomatic groups, a significant difference was only found in A2. When the 3 groups were compared in terms of A differences, there was no significant intergroup difference in terms of A0-A1 difference. A0-A2, A1-A2, and A3-A2 differences were significantly higher in the symptomatic group as compared to the asymptomatic and control groups. The differences between the control and asymptomatic groups were not significant. For A0-A3, a significant difference was only found between the symptomatic and asymptomatic groups.

ROC analyses were performed to identify cutoff values indicating the abnormalities (please see the Supplementary Fig. S2).

When the data of *control and symptomatic groups* were analyzed, significant cutoff values could not be identified for the T values. For the T differences, the most valuable cutoff value was >1 ms for T2-T0 difference, with 55.81% sensitivity and 84.37% specificity. No significant cutoff value was found for the other T differences.

For the A values, the most valuable cutoff value was <1.5 μV in A2, with 75.9% sensitivity and 75.0% specificity. For the A differences, >47.05% A0-A2 difference provided 70.4% sensitivity and 96.9% specificity and >32.14% A1-A2 difference gave 75.9% sensitivity and 87.5% specificity. For the A3-A2 difference, >43.75% increase revealed 64.8% sensitivity and 96.9% specificity.

When ROC curves of *symptomatic and asymptomatic* groups were analyzed, no significant cutoff value could be identified for the T values and T differences. The most valuable cutoff value was <1.2 μV A2 with 72.2% sensitivity and 60.6% specificity. For the A differences, >48.3% A0-A2 difference gave 66.7% sensitivity and 72.7% specificity and >33.0% A2-A1 difference provided 74.1% sensitivity and 69.7% specificity. The most valuable cutoff value for the A3-A2 difference was >43.75%, with 64.8% sensitivity and 69.7% specificity.

No significant cutoff value could be identified for discrimination of control and asymptomatic groups.

There were no significant differences in the percentage of amplitude loss elicited by the sitting position (A0-A2 difference, %) between the patients having pain during this provocation (71%) and those who did not experience any induced pain (67.2%).

Post hoc power analyses for the statistically significant findings are presented in Table 1.

DISCUSSION

Neurophysiological tests have gained limited success for substantializing the nerve involvement in PNE.[10] SEP is one of the most frequently used technique due to its repeatable, noninvasive, and relatively painless methodology.[8,10,14-16] As far as we know, there is no satisfactory data in the literature on the role of pudendal SEPs in assisting the diagnosis in PNE. Although restriction of the stimulation site distal to the usual painful area could be the cause of poor results, another plausible reason for the rarity of abnormal SEPs could be the posture of the patient during examination. SEP studies are always performed with the patient lying supine rather than maintaining the sitting position during which the symptoms become manifest. Moreover, most of the previous pudendal SEP studies have been performed with bilateral stimulation by using ring-shaped electrodes instead of evaluating the symptomatic and asymptomatic sides individually. By considering that PNE is a kind of neuropathic pain of unilateral (or bilateral with one-sided dominance) distribution, it would be more productive to maintain the pain-provoking sitting posture in the examination and to avoid the diluting effect of normal responses evoked by asymptomatic side stimulation.

In 16 of 49 (32%) patients in our study, pudendal SEP responses could not be obtained bilaterally or unilaterally even in the initial supine position. Because there was no unrecordable pudendal SEP in the control group, absent responses in the supine position may also have a diagnostic role for severe cases. Bilateral absence of SEPs in 2 cases with unilateral symptoms may be explained by the presence of bilateral electrophysiological involvement resembling those observed in some other entrapment neuropathies.[19-21]

T0 values were significantly longer in male cases in the present study. This finding was interpreted as a consequence of significantly longer mean body heights of males.[22] When all T values (T0, T1, T2, T3) were compared among the groups, no significant differences were found (Table 1). This indicates that T values do not change in a degree sufficient to help the diagnosis of PNE during the sitting position. However, the small mean T2-T0 latency change found in this study could reflect the position effect. When the cutoff value for T2-T0 difference was taken as 1 ms, the sensitivity was 55.81% and the specificity was 84.37% for discriminating the abnormality. However, this 1 ms difference is a very small value for intra- and intersubject variabilities in SEP studies. Therefore, latency values seem to be mostly useless in interpreting the recordable pudendal SEP responses.

The amplitudes of the SEPs recorded at the initial supine position were insufficient to distinguish the abnormality of the patients with recordable SEPs in the initial study. In terms of A1, A2, and A3, the most significant change was observed at A2 (at the end of the sitting position). Although this phenomenon occurs in all groups, the decline is most obvious on the symptomatic side of the patients. It seems that a cutoff value of <1.5 μV is capable of discriminating the symptomatic group.

The distinguishing value of amplitudes was even more significant when the positional amplitude differences were analyzed. A0-A1 difference was insignificant among all groups, suggesting that the consequences of the stress exerted on the nerves were not evident immediately after taking the sitting position. However, at the end of the sitting period, the functional abnormalities in the pudendal nerves became more obvious (A0-A2 and A1-A2 differences). Amplitude loss more than the ratios provided above was able to discriminate the symptomatic side of the patients with satisfactory sensitivity and specificity values. The amplitude increase after taking the second supine position (A3-A2 difference) was also high enough on the symptomatic side of the patients to determine a cutoff value.

Post hoc power analyses showed that the statistical power achieved for the parameters with significant intergroup differences was quite high, especially for the data related to the amplitudes and amplitude differences (with more than 90% power values).

As a commentary for discrepancy between the latency and amplitude values in discriminating the abnormal conduction, it might be concluded that the main pathological process in this disorder is a compression or entrapment neuropathy causing more severe pain with additional compression, similar to the increased pain elicited by Phalen's sign in carpal tunnel syndrome.[21] In this kind of neuropathy, the conduction defect is confined to a short segment of the nerve and its major electrophysiological consequence is the conduction block that overtakes the slowed conduction even if it exists.[23-27] We might speculate that the amplitude loss during sitting is mainly caused by conduction block in the symptomatic pudendal nerve and the conduction slowing across the short entrapped segment was minor in degree or was diluted through the conduction in the afferent pathways.[28,29] We may think that although partial conduction block also occurs at the end of the sitting position in normal controls, the entrapment conditions already present in the patients lead to significantly more amplitude loss in the SEP responses (Fig. 2). Another discrepancy was observed between the degree of amplitude loss of SEP responses and the pain evoked by the sitting position. It has been shown that there was no correlation between the results of provocative tests in carpal tunnel syndrome (eg, Phalen's sign) and the electrophysiological findings.[30] Although there is no clear explanation for this phenomenon, our results might be interpreted as another indication of the resemblance of PNE to the other more renowned entrapment or compression neuropathies.

CONCLUSION

Although pudendal SEP abnormalities may be seen in supine position in some severe PNE cases, the power of the electrophysiological method can be enhanced with provocation by sitting in the majority of the patients. The decrease in amplitude, observed at the end of the sitting position, together with the increase elicited by switching to the supine position again can be claimed to be highly sensitive in discriminating the abnormality. The technique provided herein is easily applicable and noninvasive; however, the longer required test duration may be its disadvantage. Other weak points of the study are the relatively small number of the case groups and its initiation without making a sample size calculation. We tried to compensate them partly by post hoc power analysis.

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APPENDIX

Supplementary Data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.urology.2016.09.040.