REVIEW

The clinical value of EMG and SSEP in diagnosing chronic pelvic pain syndrome; a systematic review

M. C. Wissing MD¹ | S. E. I. van der Wal MD, PhD² | S. Bongarts MD³ | J. Aarnink MD¹ | K. J. B. Notten MD, PhD¹ | S. M. J. van Kuijk MD, PhD⁴ | A. H. D. M. Dam MD, PhD⁵ | K. C. P. Vissers MD, PhD² | K. B. Kluivers MD, PhD¹ | N. van Alfen MD, PhD^{6,7}

¹Department of Obstetrics and Gynaecology, Radboud University Medical Center, Nijmegen, The Netherlands

²Department of Anesthesiology, Pain and Palliative Medicine, Radboud University Medical Center, Nijmegen, The Netherlands

³The Department of Emergency, St. Jans Gasthuis Weert, Weert, The Netherlands

⁴Department of Clinical Epidemiology and Medical Technology Assessment, Maastricht University Medical Center+, Maastricht, The Netherlands

⁵Department of Obstetrics and Gynaecology, Clinique Ambroise Paré, Toulouse, France

⁶Department of Neurophysiology and Department of Neurology, Radboud University Medical Center, Nijmegen, The Netherlands

⁷Donders Center for Cognitive Neuroimaging, Radboud University Medical Center, Nijmegen, The Netherlands

Correspondence

M. C. Wissing, Department of Obstetrics and Gynaecology, Radboud University Nijmegen Medical Center, Geert Grooteplein 10, Nijmegen 6500HB, The Netherlands.

Email: myrthe.wissing@radboudumc.nl

Abstract

Background: Chronic pelvic pain syndrome (CPPS) is pain in the region of the lower pelvis for three months or longer. Which is often accompanied by complaints of organ systems in the lower abdomen. CPPS is often a subjective diagnosis where electrodiagnostic tests are recommended as a supplement in defining a diagnosis. **Objective:** Synthesize the clinical studies that describe electrodiagnostic testing in humans with a clinical diagnosis of CPPS.

Evidence Review: Registered in PROSPERO (CRD42024510404). A systematic search in Medline/PubMed, Embase, CINAHL, and Web of science, from inception till February 2024, complemented with reference examining. Two reviewers independently reviewed titles, abstracts, and full-text papers, and performed data extraction. Reviews were excluded, and papers were included if patients were clinically diagnosed with CPPS and underwent EMG and/or SSEP. The QUADAS-2 tool was used to assess the quality of studies.

Findings: Fourteen papers were included concerning EMG and/or SSEP, nine papers reported on EMG and five on SSEP. In total, 432 patients clinically diagnosed with CPPS underwent electrodiagnostic testing. 152/277 patients showed abnormalities on EMG and 102/155 patients had abnormal findings on SSEP. Due to the lack of quantitative data, no meta-analysis could be performed.

Conclusions: Abnormalities on electrodiagnostic testing are seen in half of the patients with CPPS, and therefore not recommended as a substitute in the diagnostic process. The low number of patients enrolled in this review needs to be taken into consideration when interpreting the results. Further research on the sensitivity of EMG and/or SSEP in PN is recommended.

KEYWORDS

electrodiagnostic testing, EMG, pudendal neuralgia, SSEP

INTRODUCTION

Chronic pelvic pain syndrome (CPPS) is characterized by pain in the pelvic region, either continuous or with intercurrent episodes for over 3 months, and often without evidence of pathology. Although data are limited, CPPS affects about 7%–24% of the general population,^{1–3} and is equally common among men and women. The etiology of CPPS is complex due to an interplay of gynecologic, urologic, gastrointestinal, musculoskeletal, neurological, and psychosocial comorbidities.³ Neurological abnormalities can cause

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made. © 2025 The Author(s). *Pain Practice* published by Wiley Periodicals LLC on behalf of World Institute of Pain.

CPPS, and a frequently involved nerve is the pudendal nerve, which arises from the ventral rami of S2–S4 of the sacral plexus.^{4–6} The pudendal nerve exits the pelvis through the greater sciatic foramen and enters the perineum through the lesser sciatic foramen; it then enters the pudendal canal. The pudendal nerve normally terminates in three branches: the inferior rectal branch, perineal branch, and dorsal sensory nerve of the clitoris.^{5–8}

CPPS is an umbrella term for multiple pain conditions in the pelvic region (including conditions caused by changes in peripheral nerves); part of this umbrella term are vulvodynia, Proctalgia Fugax (PF), Persistent Genital Arousal Disorder (PGAD), and Pudendal Neuralgia (PN).³ Vulvodynia is spontaneous or provoked chronic vulvar pain often described as burning, itching, and stabbing, and frequently paired with dyspareunia.⁹ PF is the sudden non-radiating pain in the anorectal area that is often described as cramping that lasts for approximately 30 min.¹⁰ PGAD is the unwanted sensation of genital arousal in the absence of sexual desire or stimulation, often accompanied with pelvic pain.¹¹ PN is recognized by the burning sensation in the distribution area of the pudendal nerve and the increase of the pain while sitting.^{3,6,7,12,13} The Nantes criteria have been developed in 2007 for the clinical diagnosis of PN.^{13,14} The limitation of these criteria, however, is that they are entirely subjective.^{13,14} A variety of electrodiagnostic tests (EDx) have been described as part of the evaluation of PN, but their clinical value is still unclear.^{12,14–17}

The European Association Urogynaecology (EAU) guidelines on CPPS shortly mention the option of EDx, but state that significant nerve damage is probably needed to show abnormal results.³ EDx are furthermore mentioned to have limited sensitivity and specificity and are recommended for use as complementary tests only.^{3,18} Abnormal test results may reveal altered function of the pudendal nerve and yield information on neurological lesions.^{16,17} But pain may also be associated with non-detectable nerve damage, and then these tests may be normal.

EDx is a term routinely used for the combination of nerve conduction studies (NCS) and needle EMG (nEMG). NCS assesses the function of a motor or sensory nerve by stimulating the nerve and recording the response further down- or upstream from a muscle or skin area. NCS can detect demyelination; this means that the myelin sheath surrounding axons is affected and can lead to loss of conduction velocity.^{19,20} nEMG can show axonal loss with denervation of muscle fibers and, if time progresses, a reinnervation of muscle fibers due to axonal regeneration.¹⁹ Lesions of the nerves (or neuropathy) can be caused by stretching of the nerve, compression of the nerve, direct injury of the nerve by a trauma, or inflammation of the nerve.^{21,22} In the case of a nerve lesion, the stimulus will lead to a diminished or absent response of the muscle or sensory nerve fibers, sometimes accompanied by a decrease in nerve conduction velocity.¹⁹ Somatosensory Evoked Potentials (SSEP) are a different form of EDx that includes stimulation of a skin area or major limb nerve and recording responses from the cerebral sensory cortex corresponding to that body part.^{19,23}

CPPS is a condition with high impact, and the diagnosis of CPPS is often made after many years of symptoms.^{2,3,24,25} Different diagnostic tools have been proposed, but systematic overviews on diagnostic value and clear recommendations are lacking. Meanwhile, patients may undergo multiple evaluations, trials of medications, and even surgical interventions. The evaluation of treatments is furthermore hampered when there is no optimized diagnostic phase. In this systematic review, we summarize the clinical studies that have described the results from neurophysiologic tests in humans with CPPS. The research question is the percentage of patients that show evidence of EDx abnormalities on EMG and SSEP testing in case of a clinical diagnosis of CPPS.

METHODS

A systematic literature review was performed to identify and summarize all studies that assess the use of EMG and SSEP in patients with CPPS. The review is reported in line with the PRISMA guidelines for systematic reviews and meta-analyses.^{26–28} The PRISMA checklist and PRISMA flow diagram can be found in Data S1 and S2. This review is registered in PROSPERO under registration number CRD42024510404.²⁹

Selection process and data extraction

A literature search was performed on 6 February 2024 and was conducted in Medline (using PubMed), Embase, CINAHL, and Web of Science. Clinical studies were selected if they investigated CPPS in humans and used EDx as a diagnostic instrument. The reference lists of the selected papers and relevant reviews were crossexamined for other papers that met the eligibility criteria. Identification, screening, eligibility, inclusion of studies, and data extraction were conducted by two authors (MW and JA) independent from each other. Senior researchers (SvdW, KK, KV, and KN) arbitrated any disparities.

The following keywords for NCS, nEMG, and/or SSEP in diagnosing CPPS were used and combined using Boolean operators: electromyography, electromyogram, electroneuromyography, EMG, myography, myogram, electrodiagnosis, electric diagnosis, electrical diagnosis, SSEP, SEP, evoked potentials, somatosensory, evoked potentials, pudendal, pudendus, pudendal neuralgia, and

TABLE 1 Study characteristics.

Author	Cohort study type	Type of pain	Nantes criteria	n	Female (%)	Abnormal nEMG/NCS	Abnormal SSEP
Benson 2005 ⁴⁶	Retrospective	Pain distribution area pudendal nerve	Applicable	64	72%	41/64 (64%)	_
Ormeci 2022 ⁴⁷	Prospective	CPP	Yes	43	58%	_	35/43 (81%)
Cappellano 2013 ⁴⁸	Retrospective	CPPS	Yes	62	100%	_	38/62 (61%)
Lee 2001 ⁴⁹	Prospective	CPPS	Not applicable	12	0%	-	2/12 (17%)
Kogan 2011 ⁵⁰	Prospective	CPPS (IIIB)	No	32	_	_	23/32 (72%)
Vodusek 2012 ⁵¹	Retrospective	Pelvic and perineal pain	No	38	54%	4/38 (11%) ^a	_
Frasson 2009 ⁵²	Prospective	Vaginism and vestibulodynia	No	20	100%	18/20 (90%) ^a	-
Villot 2016 ⁵³	Prospective	PGAD	No	23	100%	14/23 (61%)	_
Amarenco 2001 ⁵⁴	Retrospective	genital pain	Not applicable	6	0%	_	4/6 (67%)
Shafik 1998 ⁵⁵	Prospective	Vulvodynia	Applicable	11	100%	3/11 (27%) ^a	_
Shafik 1997 ⁵⁶	Retrospective	Anal pain	Not applicable	7	100%	7/7 (100%) ^b	-
GowChingGer 1993 ⁵⁷	Retrospective	Rectal pain	Not applicable	40	62%	20/40 (50%) ^b	-
Wexner 1991 ⁵⁸	Prospective	Rectal pain	Not applicable	19	74%	14/19 (74%) ^a	-
Damphousse 2012 ⁵⁹	Retrospective	Proctalgia fugax	No	55	82%	31/55 (56%) ^a	_
Case reports							
Insola 2010 ⁶⁰	Case report	Perineal pain	Yes	1	100%	1/1 (100%)	_
Origo 2019 ⁶¹	Case report	Genital pain	Yes	1	100%	1/1 (100%)	_
Kelly 2010 ⁶²	Case report	Genital pain	No	1	100%	_	1/1 (100%)
Huertas-Romero 2019 ⁶³	Case report	PN	No	1	0%	-	1/1 (100%)
Alvarado 2019 ⁶⁴	Case report	postoperative neuropathic pain	No	1	100%	_	1/1 (100%)
Isik 2017 ⁶⁵	Case report	PN	Yes	1	0%	1/1 (100%)	1/1 (100%)

Note: Applicable/not applicable=study is executed before the existence of the Nantes criteria (<2007), we assessed if the Nantes criteria could be applied based on eligibility criteria; -=not available/absent.

Abbreviations: CPPS, chronic pelvic pain syndrome; PGAD, persistent genital arousal disorder; PN, pudendal neuralgia.

^aNeedle electromyography.

^bUnclear if nEMG or NCS was used.

pudendal nerve(s). If applicable, MeSH terms were added. The detailed search strings can be found in Data S3. There were no language restrictions. No time frame was set.

The software platform Covidence was used for the selection process and data extraction.³⁰ Duplicates were removed automatically and manually. Relevant papers were selected by title and abstract based on the inclusion and exclusion criteria, followed by Full-text screening. Corresponding authors were contacted in case full-text papers were not available at (university) libraries in the Netherlands (Radboud University and interlibrary loan). We extracted data on study design, the aim of the study, sample size, characteristics of the study population, the investigated diagnostic tests, and characteristics of and results pertaining to those tests.

Risk of bias assessment

The QUADAS-2 tool (Quality Assessment of Diagnostic Accuracy Studies)^{31,32} was used to assess the quality of individual studies. The QUADAS-2 tool consists of four key domains: patient selection, index test, reference standard, and flow and timing.

Statistical analysis

In case of sufficient quantitative data, meta-analysis is performed to synthesize the results.²⁸

Case reports with less than 5 cases are presented, but not entered into the meta-analysis, to avoid the risk of bias due to the study design and having an influence on the results.³³

RESULTS

Study selection

The results of the search and study selection are shown in Data S2 flow diagram. The full text of the 58 papers that met the inclusion and exclusion criteria was attempted to be obtained in multiple libraries. In the end, for 12 of the studies, no full text could be obtained; 11/12 of these papers were published before the year 2000 and were not digitalized or not stored in an accessible library.^{34–45}

Study characteristics

The details of the studies enlisted are presented in Table 1. In all included papers, the patients were assessed to determine if the Nantes criteria were applicable, and in two papers, the Nantes criteria were embedded in the inclusion criteria, while in two other papers, the Nantes criteria were applicable. The patients of these four papers were suspected of PN with nerve entrapment. In ten studies, the patients underwent EDx, and the result was described as an outcome of the study, whereas in four studies, the results of EDx were described as a baseline characteristic of a cohort undergoing some sort of treatment.

Nine of the selected studies reported on NCS and/or nEMG, and five on SSEP. Five out of nine papers (56%) reporting on EDx used concentric needle EMG. The papers included a total of 432 patients (range 6–64). two hundred and seventy-seven of these patients (n=277) underwent EMG or NCS only, and 155 underwent SSEP.

The papers described neurophysiological changes or abnormalities in 254/432 patients (50%); these abnormalities are outlined in Table 1. In 55% (152/277) of the cases, abnormalities were seen on NCS and/or nEMG. SSEP showed abnormalities in 66% (102/155) of the cases.

There were two papers reporting on EMG in 75 patients who seemed to fulfill the Nantes criteria, of whom 44/75 (59%) patients showed abnormalities. In 73/105 (70%) patients with positive Nantes criteria who underwent SEPP had abnormalities were found. Conducting a meta-analysis was not possible due to the lack of sufficient data and the heterogeneity in outcome measurements used in the included papers.

Risk of bias assessment

All included papers were assessed with the QUADAS-2 tool; however, in 6 (43%) papers, the assessment was

incomplete due to lacking information. Therefore, we qualified these papers as low to moderate, based on items that were applicable. QUADAS-2 could be fully completed for 8 (57%) papers.

In two studies, the study population was not clearly defined.^{51,56} In six papers it was not clear whether the described neurophysiologic examinations were performed on all participants.^{46,48,49,51,53,57} The main results were well presented and yielded answers to the study aims in all studies.^{46–59}

Quality assessment could not be performed for the six case reports, $^{60-65}$ and two papers that were only available as conference abstracts. 50,51

DISCUSSION

This systematic review summarizes 14 papers on 432 participants with CPPS, of whom 254 (50%) had evidence of neurophysiological changes or abnormalities on EDx. In 277 of these 432 participants (64%), EDx consisted of either NCS or EMG. In this group, 152/277 (55%) showed an abnormal test result. Among the 155 participants who had undergone SEPP, 102 participants (66%) showed abnormalities on SEPP.

Main findings

Our review showed that electrophysiologic abnormalities are present in half of the patients with CPPS. In patients with CPPS, these tests may discriminate between the presence or absence of neuropathy, and seem to support a pathophysiologic or anatomic substrate for the pain in half of the patients. EDx results might contribute to defining the phenotypic characteristics in CPPS. However, at this stage, this knowledge on pudendal neuropathy does not lead to any change in treatment or counseling, and the clinical value of EMG and SSEP is insufficiently shown.

There are two possible explanations for the fact that in 50% of the patients we do not see any abnormalities: not all patients with CPPS have axonal loss or demyelination of the pudendal nerve, or the EDx are not able to show the axonal loss or demyelination. Based on the available studies, we do not know which of these explanations is more likely. It is known, however, that CPPS has a variable etiology and underlying pathology, and it is thereby likely that not all patients suffer from detectable nerve function loss. Some patients may nonetheless experience the so-called "positive" symptoms, that is, pain and paraesthesia. Comparable conclusions have been drawn in another review on CPPS.⁶⁶

The subcategory of patients with PN showed a lower proportion of participants (35%) without abnormalities, but even in cases of PN defined by the Nantes criteria (the gold standard), the neurophysiological tests showed normal test results (i.e., no evidence of pudendal lesions). Our results suggest that neurophysiological tests are not a substitute for the subjective diagnostic criteria, such as the Nantes criteria for PN, but might be used as complementary tools. They might help in extinguishing the phenotype of CPPS or confirm cases with suspected nerve injury when clinical findings are ambiguous. Nonetheless, CPPS and PN are still diagnosed based on clinical symptoms, because it is still unclear what the meaning of evidence of abnormal EDx is. A randomized controlled trial is needed to evaluate the potential role of EDx as a complementary tool to the clinical criteria.

A heterogenic pathology and etiology are not uncommon findings in nerve disorders, as this is similar to the situation in carpal tunnel syndrome or entrapment radiculopathies. No gold standard test exists either, and a clinical diagnosis based on subjective findings is often made, with the supplemental use of EDx.¹⁸

TABLE 2 Recommendations for clinical practice.

SSEP and EMG are not recommended to routinely use in the diagnostic process of CPPS

In diagnosing Pudendal Neuralgia, EMG and SSEP could be a supplement to the Nantes criteria

Electrodiagnostic tests might help in phenotyping patients

Clinical diagnostic criteria are the gold standard for diagnosing neuropathies

Abbreviations: CPPS, chronic pelvic pain syndrome; EMG, electromyography; SSEP, somatosensory evoked potential.

However, we argue that EDx might be useful in some patients with CPPS, especially in PN, with a suspected origin of nerve trauma or entrapment as an explanation of the origin of complaints. Patients with a trauma in the pelvic area due to vaginal delivery or an operation seem most prone to underlying nerve trauma or entrapment. Nonetheless, reports on symptom-free women who did give birth and had an abnormal EMG^{67,68} do exist. This might suggest that the pudendal nerve can show axonal loss or demyelination without symptoms, and this needs to be considered in further research.^{67,68} Table 2 and Figure 1 summarize the recommendations based on the main findings of this study.

Strengths and limitations

A strength of the study is that the PRISMA guidelines were followed. The two main researchers did the search, the selection, and the reviewing separately and compared their results to avoid researcher bias (MW and JA).

Each paper concerning patients with pelvic pain and electrodiagnostic testing was evaluated for the diagnosis CPPS, and especially for PN, thereby avoiding exclusion of papers on patients not evaluated by the Nantes criteria.

A limitation of the study is that there was considerable heterogeneity in the low number of papers available on this topic. Preferably, each paper had the same study aim and followed the same protocol; unfortunately, some papers did not have the aim to assess the diagnostic accuracy of EDx, but results were presented as extra data. Because

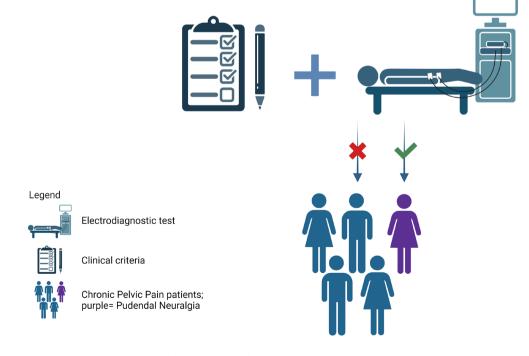


FIGURE 1 How to use electrodiagnostic tests in Chronic Pelvic Pain Syndrome; as a supplement to clinical diagnostic criteria and for patients with suspected nerve injury such as Pudendal Neuralgia.

of the lack of consistency, we compared the outcomes of abnormal versus normal EMG or SSEP and aimed to observe a trend in abnormalities seen in patients with CPPS. This was to formulate cautious conclusions for the use of EDx in CPPS, and we noticed a higher amount of abnormalities in patients with PN. Subsequently, further research is needed to confirm this trend.

Every paper was subjected to a risk of bias assessment; however, some papers missed information to complete the assessment. We qualified these papers as low to moderate. Despite this, we included these papers in the analysis because of the limited literature available on this topic.

Further research should be undertaken to investigate the clinical relevance of abnormalities on EDx in CPPS. A randomized controlled trial is recommended to evaluate the sensitivity and clinical relevance of EDx in CPPS. However, our results suggest that EDx might be less predictive in diagnosing CPPS than in PN. To answer this question, a study is needed on the relation between complaints and neurophysiological tests in patients with PN defined by the Nantes criteria, and a control group would be included. This would be the first step in assessing the role of EDx in patients with PN. Such a study is not available until now.

CONCLUSION

Concluding from the literature available on this subject, we found that EDx abnormalities, indicating pudendal nerve injury, were present in half of the patients with CPPS. We recommend a prospective study including participants with PN defined by the Nantes criteria and a control group to assess the sensitivity of EDx.

AUTHOR CONTRIBUTIONS

Concept: MW, SvdW, SB, KN, SvK, AD, KV, KK, and NvA. Acquisition: MW, JA. Analysis: MW, JA, SvdW, KV, KK, KN. Interpretation of data: SvK, NvA, MW, KN, KK. Writing manuscript: MW, SB. Revising manuscript: SvdW, KN, SvK AD, KV, KK, NvA.

ACKNOWLEDGMENTS

There were no contributors to this study.

FUNDING INFORMATION

This research did not receive any specific grant from funding agencies in the public, commercial, or not-forprofit sectors.

CONFLICT OF INTEREST STATEMENT

There were no competing interests. Prof. K. Vissers and S. van Kuijk are Editorial Board members of Pain Practice and co-authors of this article. To minimize bias, they were excluded from all editorial decision-making related to the acceptance of this article for publication. DATA AVAILABILITY STATEMENT

No new data are generated.

PATIENT CONSENT STATEMENT

Patient consent statement is not applicable.

ORCID

M. C. Wissing b https://orcid.org/0009-0000-6393-5258 S. M. J. van Kuijk b https://orcid. org/0000-0003-2796-729X K. B. Kluivers b https://orcid.org/0000-0002-4681-4304

REFERENCES

- Possover M, Forman A. Voiding dysfunction associated with pudendal nerve entrapment. Curr Bladder Dysfunct Rep. 2012;7(4):281-5.
- Richtlijn Chronische Bekkenpijn. 2021. Available from: https:// richtlijnendatabase.nl/richtlijn/chronische_bekkenpijn/startpagina_-_chronische_bekkenpijn.html
- Engeler D, Baranowski AP, Berghmans B, Birch J, Borovicka J, Cottrell AM, et al. EAU guidelines on chronic pelvic pain. 2022.
 [76]. Available from: https://d56bochluxqnz.cloudfront.net/ documents/full-guideline/EAU-Guidelines-on-Chronic-Pelvic-Pain-2022_2022-03-29-084111_kpbq.pdf
- Ramsden CE, McDaniel MC, Harmon RL, Renney KM, Faure A. Pudendal nerve entrapment as source of intractable perineal pain. Am J Phys Med Rehabil. 2003;82(6):479–84.
- Robert R, Prat-Pradal D, Labat JJ, Bensignor M, Raoul S, Rebai R, et al. Anatomic basis of chronic perineal pain: role of the pudendal nerve. Surg Radiol Anat. 1998;20(2):93–8.
- Khoder W, Hale D. Pudendal neuralgia. Obstet Gynecol Clin N Am. 2014;41(3):443–52.
- Maldonado PA, Chin K, Garcia AA, Corton MM. Anatomic variations of pudendal nerve within pelvis and pudendal canal: clinical applications. Am J Obstet Gynecol. 2015;213(5):727.e1– 727.e6.
- Marvel RP. Pudendal neuralgia: making sense of a complex condition. Curr Sex Health Rep. 2018;10(4):237–45.
- Schlaeger JM, Glayzer JE, Villegas-Downs M, Li H, Glayzer EJ, He Y, et al. Evaluation and treatment of vulvodynia: state of the science. J Midwifery Womens Health. 2023;68(1):9–34.
- Carrington EV, Popa S-L, Chiarioni G. Proctalgia syndromes: update in diagnosis and management. Curr Gastroenterol Rep. 2020;22(7):35.
- Pease ER, Ziegelmann M, Vencill JA, Kok SN, Collins CS, Betcher HK. Persistent genital arousal disorder (PGAD): a clinical review and case series in support of multidisciplinary management. Sex Med Rev. 2022;10(1):53–70.
- Fanucci E, Manenti G, Ursone A, Fusco N, Mylonakou I, D'Urso S, et al. Role of interventional radiology in pudendal neuralgia: a description of techniques and review of the literature. Radiol Med. 2009;114(3):425–36.
- Labat JJ, Riant T, Robert R, Amarenco G, Lefaucheur JP, Rigaud J. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (*Nantes criteria*). Neurourol Urodyn. 2008;27(4):306–10.
- Stav K, Dwyer PL, Roberts L. Pudendal neuralgia. Fact or fiction? Obstet Gynecol Surv. 2009;64(3):190–9.
- Elkins N, Hunt J, Scott KM. Neurogenic Pelvic Pain. Phys Med Rehabil Clin N Am. 2017;28(3):551–69.
- Bianchi F, Squintani GM, Osio M, Morini A, Bana C, Ardolino G, et al. Neurophysiology of the pelvic floor in clinical practice: a systematic literature review. Funct Neurol. 2017;22(4):173–93.
- 17. Lefaucheur JP, Labat JJ, Amarenco G, Herbaut AG, Prat-Pradal D, Benaim J, et al. What is the place of electroneuromyographic

studies in the diagnosis and management of pudendal neuralgia related to entrapment syndrome? Neurophysiol Clin. 2007;37(4):223–8.

- Assmus H, Antoniadis G, Bischoff C. Carpal and cubital tunnel and other, rarer nerve compression syndromes. Dtsch Arztebl Int. 2015;112(1-2):14-25; quiz 6.
- Aminoff MJ. Electrophysiologic testing for the diagnosis of peripheral nerve injuries. Anesthesiology. 2004;100(5):1298–303.
- Stadelmann C, Timmler S, Barrantes-Freer A, Simons M. Myelin in the central nervous system: structure, function, and pathology. Physiol Rev. 2019;99(3):1381–431.
- Finnerup NB, Haroutounian S, Kamerman P, Baron R, Bennett DLH, Bouhassira D, et al. Neuropathic pain: an updated grading system for research and clinical practice. Pain. 2016;157(8):1599–606.
- 22. pain) Iiaftso. IASP terminology 2011. [cited 2024]. Available from: https://www.iasp-pain.org/resources/terminology/
- Cavalcanti GA, Bruschini H, Manzano GM, Nunes KF, Giuliano LM, Nobrega JA, et al. Pudendal somatosensory evoked potentials in normal women. Int Braz J Urol. 2007;33(6):815–21. https:// doi.org/10.1590/S1677-55382007000600010
- 24. Filler AG. Diagnosis and treatment of pudendal nerve entrapment syndrome subtypes: imaging, injections, and minimal access surgery. Neurosurg Focus. 2009;26(2):E9.
- Itza-Santos F, Zarza-Luciáñez D, Salinas J, Gómez-Sancha F, Bathal-Gaude H. Pudendal nerve entrapment syndrome. Rev Neurol. 2010;50(3):157–66.
- Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71. https://doi.org/10.1136/bmj.n71
- Page MJ, Moher D, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. BMJ. 2021;372:n160.
- 28. Deeks JJ, Higgins JPT, Altman DG, McKenzie JE, Veroniki AA, on behalf of the Cochrane Statistical Methods Group. Chapter 10: Analysing data and undertaking meta-analyses. In: Higgins JPT, Thomas J, Chandler J, Cumpston M, Li T, Page MJ, et al., editors. Cochrane Handbook for Systematic Reviews of Interventions version 6.5 (updated August 2024). Cochrane; 2024. https://training.cochrane.org/handbook/current/chapter-10
- Page MJ, Shamseer L, Tricco AC. Registration of systematic reviews in PROSPERO: 30,000 records and counting. Syst Rev. 2018;7(1):32.
- 30. Covidence systematic review software. 2024. Available from: www.covidence.org
- 31. Bristol Uo. QUADAS-2. Available from: https://www.bristol.ac. uk/population-health-sciences/projects/quadas/quadas-2/
- Whiting PF, Rutjes AW, Westwood ME, Mallett S, Deeks JJ, Reitsma JB, et al. QUADAS-2: a revised tool for the quality assessment of diagnostic accuracy studies. Ann Intern Med. 2011;155(8):529–36.
- 33. Viswanathan MAM, Berkman ND, Chang S, Hartling L, McPheeters LM, Santaguida PL, et al. Assessing the risk of bias of individual studies in systematic reviews of health care interventions. AHRQ Publication No. 12-EHC047-EF; 2012. Available from: www.effectivehealthcare.ahrq.gov/
- Amarenco G, Kerdraon J, Le Cocquen A, Adba MA. Perineal neuralgia clinical and electrophysiological symptoms in 89 patients. Ann Read Adapt Med Phys. 1991;34(1):19–24.
- Amarenco G, Lanoe Y, Ghnassia RT, Goudal H, Perrigot M. Alcock's canal syndrome and perineal neuralgia. Rev Neurol. 1988;144(8–9):523–6.
- Amarenco G, Lecocquenamarenco A, Kerdraon J, Lacroix P, Adba MA, Lanoe Y. Perineal neuralgias. Presse Med. 1991;20(2):71–4.

- Chen MT, Wu ZA, Chang LS, Lin SN, Lin ADL, Yin JH, et al. Cortical evoked potentials from the pudendal nerve. J Surg Assoc. 1987;20(5):431–7.
- Gupta PR, Dorfman LJ, Greenleaf WJ, Davidson JM. Pudendal somatosensory evoked-potentials – evidence for central delay. Electroencephalogr Clin Neurophysiol. 1983;56(4):P34.
- Haldeman S, Bradley WE, Johnson BK. Pudendal somatosensory evoked-potentials (PER). Neurology. 1981;31(4):152.
- Kaplan PE. Somatosensory evoked responses obtained after stimulation of the pelvic and pudendal nerves. Electromyogr Clin Neurophysiol. 1983;23(1–2):99–102.
- 41. Kogan MI, Belousov II, Shornikov PV. Neurophysiologic evaluation of patients with chronic prostatitis (III B chronic pain syndrome). Urol (Mosc). 2012;(4):37–42.
- 42. Lee BW, Lasak AM. Levator ani syndrome management with neuromodulation of pudendal nerve. PM R. 2015;7(9 Suppl. 1):S173.
- Martinez-Mas E, Trullenque-Peris R, Lloris-Carsi JM. Experimental study of the pudenal nerve by evoked potentials. A preliminary step to its clinical application. Res Surg. 1989;1(3):148-51.
- Strijers RLM, Felt-Bersma RJF, Visser SL, Meuwissen SGM. Anal sphincter EMG in anorectal disorders. Electromyogr Clin Neurophysiol. 1989;29(7–8):405–8.
- 45. Wexner SD, Jorge JMN. Colorectal physiological tests: use or abuse of technology? Eur J Surg. 1994;160(3):167–74.
- Benson JT, Griffis K. Pudendal neuralgia, a severe pain syndrome. Am J Obstet Gynecol. 2005;192(5):1663–8.
- 47. Ormeci B, Uyanik HU, Tasdelen N, Keles EC, Erdogru T, Oge AE. Dynamic somatosensory evoked potential and magnetic resonance imaging in pudendal neuropathy: a comparative study with respect to the clinical diagnostic criteria. Neurol Sci Neurophysiol. 2022;39(3):119–25.
- Cappellano F, Gelardi C, Fornara C, Salvatore S, Origoni M, Candiani M, et al. Neural blockade under neurophysiological guidance for chronic pelvic pain in 110 women affected by pudendal neuralgia (PN). Neurourol Urodyn. 2013;32(Suppl. 1):S13–S14.
- Lee JC, Yang CC, Kromm BG, Berger RE. Neurophysiologic testing in chronic pelvic pain syndrome: a pilot study. Urology. 2001;58(2):246–50.
- Kogan MI, Belousov II, Shornikov PV, Shangichev AV. Patterns of pelvic floor disorders in CPPS/CP III B. Eur Urol Suppl. 2011;10(2):194.
- 51. Vodusek D. Neurogenic perineal and pelvic pain? An EMG study. J Sex Med. 2012;9(Suppl. 5):326.
- Frasson E, Graziottin A, Priori A, Dall'Ora E, Didone G, Garbin EL, et al. Central nervous system abnormalities in vaginismus. Clin Neurophysiol. 2009;120(1):117–22. https://doi.org/ 10.1016/j.clinph.2008.10.156
- 53. Villot A, Thubert T, Deffieux X, Jousse M, Breton FL, Lacroix P, et al. Perineal neurophysiological assessment in 23 patients suffering from persistent genital arousal disorders: evidence of pudendal neuropathy? Int J Sex Health. 2016;28(1):50–4.
- Amarenco G, Ismael SS, Bayle B, Denys P, Kerdraon J. Electrophysiological analysis of pudendal neuropathy following traction. Muscle Nerve. 2001;24(1):116–9.
- Shafik A. Pudendal canal syndrome as a cause of vulvodynia and its treatment by pudendal nerve decompression. Eur J Obstet Gynecol Reprod Biol. 1998;80(2):215–20.
- Shafik A. Pudendal canal syndrome and proctalgia fugax. Dis Colon Rectum. 1997;40(4):504.
- Gow Ching G, Wexner SD, Jorge JMN, Lee E, Amaranath LA, Heymen S, et al. Evaluation and treatment of chronic intractable rectal pain – a frustrating endeavor. Dis Colon Rectum. 1993;36(2):139–45.
- Wexner SD, Marchetti F, Salanga VD, Corredor C, Jagelman DG. Neurophysiologic assessment of the anal sphincters. Dis Colon Rectum. 1991;34(7):606–12.

- Damphousse M, Jousse M, Verollet D, Guinet A, Le Breton F, Lacroix P, et al. Evidence of pudendal neuropathy in proctalgia Fugax: perineal neurophysiological assessment in 55 patients. Prog Urol. 2012;22(4):220–4.
- 60. Insola A, Granata G, Padua L. Alcock canal syndrome due to obturator internus muscle fibrosis. Muscle Nerve. 2010;42(3):431–2.
- 61. Origo D, Tarantino AG. Osteopathic manipulative treatment in pudendal neuralgia: a case report. J Bodyw Mov Ther. 2019;23(2):247–50.
- Kelly RD, Salcedo DM. Pudendal nerve injury after harnessed parachuting accident: a case report. PM R. 2010;2(9 SUPPL. 1):S129.
- 63. Huertas-Romero NR, Cuenca TT, Diaz-Ruiz J. Neurophysiological study of the pudendal nerve in a patient with chronic genital pain. Muscle Nerve. 2019;60(SUPPL 1):S20.
- 64. Alvarado CG, Reyes DFC, Diaz-Ruiz J, Diaz DOC, Rosas-Jaimes J, Patino JWV. Neurophysiological abnormalities of the pudendal nerve after colpopexy to the right sacrospinous ligament: a case report. Muscle Nerve. 2019;60(Suppl. 1):S99.
- Isik H, Fuglsang-Frederiksen A, Pugdahl K, Tankisi H. Pudendal neuralgia diagnosed by electrophysiological examination. Ugeskr Laeger. 2017;179(21):V08150667.
- Labat JJ, Delavierre D, Sibert L, Rigaud J. Explorations électrophysiologiques des douleurs pelvipérinéales chroniques. Prog Urol. 2010;20(12):905–10.
- 67. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol. 1990;97(9):770–9.

 Gregory W, Lim J, Holland A, Clark AL. Tracking neurophysiologic changes to pelvic floor muscles-before pregnancy and following delivery. Int Urogynecol J Pelvic Floor Dysfunct. 2014;25(1):S23–S24.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Data S1 Data S2 Data S3

> How to cite this article: Wissing MC, van der Wal SEI, Bongarts S, Aarnink J, Notten KJB, van Kuijk SMJ, et al. The clinical value of EMG and SSEP in diagnosing chronic pelvic pain syndrome; a systematic review. Pain Pract. 2025;25:e70028. https://doi.org/10.1111/papr.70028