

Outcomes of Median Nerve Release in Complex Regional Pain Syndrome Type 1 of the Hand: A Prospective Case Series

Francisco del Piñal, MD, Dr Med*

Purpose Pain, allodynia, and stiffness in complex regional pain syndrome (CRPS) greatly affects social, vocational, and community engagement. The aim of this study was to evaluate the effect of isolated median nerve releases on the outcome of CRPS 1 of the hand.

Methods In this prospective case series, people of any age diagnosed with and treated for CRPS 1 of the upper limb attending the author's practice were consecutively recruited. All were self-referrals dissatisfied with the treatment provided previously. Only patients who had been treated with nerve release to control their symptoms were included. Primary outcome measures were pain and Disabilities of the Arm, Shoulder, and Hand score. A secondary outcome was withdrawal from pain medication. Full resolution was defined as 0 pain, on a scale of 0–10, and total withdrawal from pain medication at the latest follow-up.

Results Between January 2018 and December 2022, 82 participants with CRPS 1 of the hand for an average of 20 months were evaluated. Eighty-five nerve releases were performed. As per protocol, carpal tunnel release was performed in all, and three patients also received an endoscopic pronator release. Minor procedures unrelated to the pain were carried out concomitantly in 17 patients. Significant improvements were observed in pain score (mean 8.9 ± 1.2 at baseline vs 0.6 ± 1.8 at 35 months) and in Disabilities of the Arm, Shoulder, and Hand score (82 ± 13 vs 13 ± 20) for the same period. Five patients (6%) were considered failures. In contrast, 65 patients (79%) had full resolution of their symptoms.

Conclusions This study demonstrates that a large percentage of patients diagnosed with and treated for CRPS type 1 can have full resolution of their symptoms with carpal tunnel release. Future research is needed to understand the pathophysiology and the failures. (*J Hand Surg Am.* 2025;50(2):130–137. Copyright © 2025 by the American Society for Surgery of the Hand. All rights are reserved, including those for text and data mining, AI training, and similar technologies.)

Type of study/level of evidence Therapeutic II.

Key words Algodystrophy, CRPS, irritative carpal tunnel syndrome, reflex sympathetic dystrophy, Sudeck.



+ Additional Material
at jhandsurg.org

From *Hand Surgeon, Private Practice, Madrid, Spain.

Received for publication April 15, 2024; accepted in revised form September 10, 2024.

Corresponding author: Francisco del Piñal, MD, Dr Med, Hand Surgeon, Private Practice, Calle Serrano 58-1B, Madrid E-39001, Spain; e-mail: pacopinial@gmail.com.

0363-5023/25/5002-0002\$36.00/0
<https://doi.org/10.1016/j.jhsa.2024.09.024>

COMPLEX REGIONAL PAIN SYNDROME (CRPS) (also known as Sudeck atrophy, reflex sympathetic dystrophy, or algodystrophy) is a well-recognized condition characterized by an abnormal painful response, usually presenting after trauma or surgery, and accompanied by characteristic

vaso- and sudomotor changes. Complex regional pain syndrome is a controversial diagnosis (although one for which there is agreement on the diagnostic criteria) with potentially a variety of pathophysiologic triggers and pathways, all of which have a variable response to existing treatments.^{1–4}

The criteria for diagnosing the condition have evolved over time. The International Association for the Study of Pain presented the so-called “Budapest criteria” to prevent any other painful condition from falling into the CRPS constellation.⁵ Initially, CRPS was divided into type 1 and type 2, the latter reserved for cases where there was a discrete nerve injury. Recently, CRPS 2 has been redefined to prevent common neuropathic pain from inappropriately being given this label.⁶ In the new definition of CRPS 2, the pain should surpass the territory of the involved nerve. A new subtype named “persistent CRPS,” which includes cases with a duration longer than a year, was created to describe a group whose symptoms are thought to be recalcitrant to treatment.^{6–8}

The available protocols for CRPS treatment vary from rehabilitation and drug therapy (opiates, anti-epileptics, bisphosphonates, or combinations thereof) to more invasive treatments such as spinal cord stimulation, ketamine coma, or continuous analgesic infusion of the brachial plexus for recalcitrant cases.^{4,9–16} Although surgery is not commonly considered as treatment, in instances where a nerve injury can be identified (CRPS 2), surgery can be helpful.^{17,18} These cases lie outside the new definition of CRPS type 2.

Recently, del Piñal^{19,20} showed that surgery was mostly effective in a large sample of patients with CRPS. However, in both series, there was a mixture of CRPS 1 and CRPS 2 cases, including some patients wrongly labeled as CRPS, as well as others who had concomitant correction of painful mechanical problems making it unclear the exact role played by the nerve surgery in pain relief in the CRPS 1 patient.

The purpose of this study was to assess the effect of median nerve release on pain and overall function in CRPS 1 patients.

MATERIALS AND METHODS

Study design and participants

This is a prospective case series with minimum 1-year follow-up, reported in accordance with the Preferred Reporting Of Case Series in Surgery guidelines.²¹ For this study, people who attended the author’s practice with a diagnosis of CRPS 1 and who

received simple nerve releases as surgical treatment for pain relief were included. Per protocol all CRPS cases had release of the median nerve in the carpal canal.²⁰ There was no age restriction. Patients whose symptoms developed after previous nerve surgery or where a damaged nerve was the pain generator (CRPS 2) were excluded. Also, people whose nerve release was combined with other procedures to treat an associated mechanical problem (hardware removal, radius malunions, etc.) that could be a pain generator were also excluded. Patients having minor concomitant procedures unrelated to the pain (scar Z-plasty or capsulotomies) were included. Patients who had dystonic-psychogenic hand (also known as causalgia-dystonia) were included.^{22,23}

This study is registered in [Researchregistry.com](https://www.researchregistry.com) (number 10102). Ethics approval was obtained from the Hospital Clínico, Universidad Complutense of Madrid, and all participants provided written, informed consent.

Preoperative assessment

All patients were assessed by the author, a hand surgeon with more than 30 years experience. The author evaluated all patients, did all the measurements, and operations. In addition to complying with the Budapest criteria, the CRPS severity score was calculated.²⁴ Also as part of a broader study, symptoms of irritative carpal tunnel syndrome, the allodynia distribution, and if the finger joints were stiff or supple were also recorded (all data pertinent to the series is included in [Appendix S1](#), available online on the *Journal’s* website at www.jhandsurg.org).

From their previous work-up, 23 patients had undergone a three-phase bone scan. The test was positive in 20 and negative in three. In addition, 75 had electrodiagnostic studies (EDS) performed. In total, 50 had no evidence of any median nerve compression, 20 had mild involvement of the median nerve in the carpal canal, 3 had moderate compression, and 2 had severe compression. Nine of the patients with negative EDS results for compression of the median nerve showed misleading diagnoses, such as signs of root compression (2), ulnar nerve at the cubital tunnel or Guyon canal (6), and radial nerve at the Frohse canal (1). In no case was the median nerve shown to be compressed at the pronator tunnel in EDS.

At the time of the first consultation, all were receiving various combinations of opioids, anticonvulsants, antidepressants, steroids, calcitonin, bisphosphonates, etc. Thirty-one patients (38%) had received multiple invasive anesthetic interventions



FIGURE 1: **A** Active flexion after an 82-month history of CRPS 1. Previous treatments included mutistellate ganglion blocks and a course of ketamine coma, among others. The patient wears a protective glove. **B** Immediate active flexion after carpal tunnel release and **C** at 10 days. The result has remained stable at the 22-month follow-up (patient #64).

(stellate ganglion block, peripheral nerve block, and radiofrequency ablations), including two who also had placement of a spinal cord stimulator.

Intervention

The operation was preferably performed under local anesthesia for the patient to see the improvement in range of motion during the procedure. Surgery was performed with an arm tourniquet in all instances. A 2 cm incision was made in the palm of the hand, and the transverse carpal ligament released. Retraction under the superficial palmar fascia distally and proximally allowed the remainder of the ligament to be exposed and divided under direct visualization. The presence of fat distally and sufficient room to pass the scissors proximally were considered indicative of ample space for all the structures to freely glide in the carpal canal (Fig. 1A–C). In three patients, there were clinical signs and symptoms of irritation of the median nerve at the pronator tunnel, and in those cases, the lacertus fibrosus, the pronator arcade, and the flexor digitorum arcade were released using a modified endoscopic technique under axillary block (apart from the carpal tunnel division).²⁵ In no case was the ulnar nerve nor the radial nerve explored despite the EDS findings suggesting abnormalities of those nerves.

After the operation, acetaminophen was prescribed for 1 or 2 days. Sympatholytic blocks or narcotic medication given were not administered to any patients. However, the drugs that the patients were taking before surgery were gradually decreased to prevent symptoms of withdrawal.

Immediate range of motion was encouraged after the surgery. Only patients with stiffness and/or persistent joint pain attended physical therapy. Aluminum extension splints were prescribed to wear at

night for flexion contractures, and dynamic splints with outriggers for extension contractures of the metacarpophalangeal joints were worn during the day, where indicated.

Outcome assessment

Patients were followed at regular intervals and eventually discharged, but never before 1 year after the surgery. Patients were asked to rate their pain before surgery and at final follow-up on a numerical rating scale, where 0 was nil/minimum and 10 was unbearable/maximum. All patients completed the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire before surgery, at 3 months, and, as a minimum, at final follow-up. The palm to pulp distance was also recorded in centimeters before surgery, at 6 weeks, and at the final visit. All data pertinent to the series are included in [Appendix S1](#).

Data management and analysis

Continuous variables are expressed as means, ranges, and SDs (SD, \pm), and categorical variables are expressed as numbers and percentages. Parametric (two sample *t* and paired *t*) and nonparametric (Mann-Whitney *U* and Wilcoxon) tests were used to analyze differences between outcomes of stiff/non-stiff joints and pain and DASH pre- and post-operation scores, according to whether the data were normally distributed. Fisher exact test was used to determine associations between categorical variables. *P* values less than .05 were considered statistically significant.

RESULTS

In the period from January 2018 to December 2022, the author treated 82 patients who were diagnosed with CRPS 1 and had been under treatment for an

TABLE 1. Outcomes of the Series at 35-Month Follow-up*

Variable	Before Surgery	After Surgery	P Value
Number of patients	82	82	
Pain (NRS) [†]	8.9 (1.2)	0.6 (1.8)	<.05
DASH	82 (13)	13 (20)	<.05
PPD (cm)	4.3 (3)	0.6 (2)	<.05
Drugs for pain	82 (100%)	5 (6%)	<.05
Full resolution [‡]		65 (79%)	

PPD, palm to pulp distance.

*Data are mean (SD), n, or n (%) unless otherwise specified.

[†]NRS: Numerical rating scale where 0 was nil/minimum and 10 was unbearable/maximum.

[‡]Full resolution implies reporting 0 for pain and receiving no drugs for pain.

TABLE 2. Outcomes in the Persistent (Chronic) Type of CRPS 1*

Variable	Before Surgery	After Surgery	P Value
Number of patients	30	30	
Pain (NRS) [†]	8.8 (1.3)	1.1 (2.4)	<.05
DASH	80 (15)	20 (27)	<.05
PPD (cm)	3.7 (3)	0.7 (2.1)	<.05
Drugs for pain	30 (100%)	4 (13%)	<.05
Full resolution [‡]		22 (73%)	

PPD, palm to pulp distance.

*Data are mean (SD), n, or n (%) unless otherwise specified.

[†]NRS: Numerical rating scale where 0 was nil/minimum and 10 was unbearable/maximum.

[‡]Full resolution implies reporting 0 for pain and receiving no drugs for pain.

average of 20.3 ±32.2 months (range, 2–160) at the time of referral. There were 70 females and 12 males with a mean age of 52 ±14.3 years old (range, 13–87). Except for two patients without a clear traumatic mechanism, all had some form of trauma or surgery that was thought to trigger the condition. The most prevalent being distal radius fractures (plated or casted, 30 cases), metacarpal or finger fractures (10 cases), and finger lacerations (10 cases), but also distant trauma (shoulder fractures or arthroscopies, six cases) were quite prevalent (see [Appendix S1](#) for details). They all complied with the Budapest criteria, and their CRPS severity score was 7.7 ±1.8 (range, 4–10; diagnostic for CRPS when ≥ 4).^{4,6,24} Ten patients presented features classified as dystonic CRPS.^{22,23}

Of note, only 13 (16%) had a typical median nerve distribution of the allodynia-dysesthesias in the hand, whereas 22 (27%) reported painful dysesthesias in the ulnar 2 or 3 digits. Nineteen reported (23%) a glove distribution, whereas the rest (34%) had an atypical distribution of only one finger, the knuckles, the wrist, etc. All but three (96%) had proximal shooting pain radiating to the shoulder or back (further details can be found in [Appendix S1](#)).

The operation consisted of division of the transverse carpal ligament in all, with 79 under local infiltration. No visible abnormalities of the median nerve were identified on inspection with loupes (×3.5). In three patients, in addition to the carpal tunnel release, the median nerve was released at the pronator tunnel. This decision was made if patients had a strong Tinel sign in the pronator arcade based on the experience that some failed carpal tunnel release cases respond to additional carpal tunnel

release and pronator release.²⁰ No nerve deformities were noticed in the pronator tunnel. Seventeen patients had minor procedures concomitantly (single or multiple capsulotomies of finger joints, z-plasties in scars, etc.; details in [Appendix S1](#)).

Except for one patient who died after 10 months follow-up, the minimum follow-up was 1 year (average follow-up 35 months, range, 10–70, ±15). The results are summarized in [Table 1](#). Ninety-three percent (77/82) stopped taking medications for pain. One patient (participant #12) stopped taken gabapentin and opioids because her pain disappeared completely, but was still prescribed oral baclofen by a neurologist for spasticity.

There were 30 patients in the group of “persistent” (chronic) type of CRPS; 22 patients (73%) continued to be symptom free at an average 35 months follow-up ([Table 2](#)).

Only 14 patients (17%) were working (with limitations) prior to the surgery. However, after surgery, 76 (93%) returned to their previous work or activities (two patients with limitations).

There were five failures (6%). Two did not improve at all, whereas three improved dramatically for 2–3 months before relapsing. One of the latter patients (patient #9) eventually had a spinal cord stimulator installed elsewhere without relief.

DISCUSSION

The success rate with current treatment protocols for CRPS 1 is low.³ It seems that there is a risk of chronicity, particularly if treatment is delayed.^{2,4} In this study, a large percentage of patients with CRPS

TABLE 3. Outcomes of Nonstiff Finger Joints Versus Stiff Finger Joints*

Variable	Nonstiff Finger Joints	Stiff Finger Joints	P Value
Number of patients	64	18	
Duration in mo (average)			
CRPS	24 (2–160)	8 (2–40)	.08
Follow-up	35 (15)	34 (13)	.77
Pain (NRS) [†]			
Before surgery	8.8 (1.2)	9.3 (0.9)	.18
6 wk	0.8 (1.1)	2.8 (2.7)	<.05
Final	0.5 (1.5)	1.1 (2.7)	.39
(Δ preop/postop)	8.3 (1.9)	8.2 (2.6)	.75
DASH			
Before surgery	81 (13)	83 (13)	.65
3 mo	26 (15)	46 (23)	<.05
Final	11 (18)	22 (26)	.07
(Δ preop/postop)	70 (21)	61 (23)	.12
PPD (cm)			
Before surgery	3.4 (2.6)	7.5 (2.3)	<.05
6 wk	0.6 (1.1)	4.5 (2.7)	<.05
Final	0.1 (0.5)	2.6 (3.2)	<.05
(Δ preop/postop)	3.2 (2.7)	4.9 (2.8)	<.05
Unable to make a full fist	4 (6%)	11 (61%)	<.05
Drugs for pain	3 (5%)	2 (11%)	.30
Full resolution [‡]	46 (72%)	8 (44.4%)	<.05
PPD (cm)			
Number of patients attending	19 (30%)	18 (100%)	<.05
Duration (mo)	4	9	<.05

Δ, difference from preoperative to postoperative values; PPD, palm to pulp distance.

*Data are mean (SD), n, or n (%) unless otherwise specified.

†NRS: Numerical rating scale where 0 was nil/minimum and 10 was unbearable/maximum.

‡Full resolution implies reporting 0 for pain and receiving no drugs for pain.

type 1 experienced a substantial decrease in pain after median nerve decompression. In 65 (79%) patients, pain was completely resolved, including a substantial number (22/30) considered to have chronic symptoms. Comparing these results with the early literature is problematic because many of the early studies were carried out in patients who did not meet the Budapest criteria, having favorable outcome (in the 80%–95% success rate range).^{26–28} Conversely, current studies, with stringent inclusion criteria, show that about two-thirds of patients continue to be symptomatic and meet the Budapest criteria 1 year after the diagnosis despite all efforts with current available treatment.^{29,30} In a Dutch multicenter study, none of the 40 patients meeting CRPS 1 criteria were pain free at the 1-year follow-up.³¹

In this study, not only pain, but patient-reported disability of the upper limb was also assessed with the DASH. Furthermore, as an objective measure of hand function, the palm to pulp distance was also recorded. Although there was a global improvement in the latter (Table 1), there were significant differences in the response among patients with stiff finger joints before surgery versus the rest (Table 3). In the stiff group, early reduction of pain (9.3 preoperative vs 2.8 at 6 weeks) allows them to progress in the rehabilitation, but they should be warned about their worse prognosis compared to patients without digit stiffness before surgery, regarding their ability to make a full fist, despite a much longer time in physical therapy (Table 3). A frozen shoulder also acted as a negative factor and patients spent more time in physical therapy than the average (10

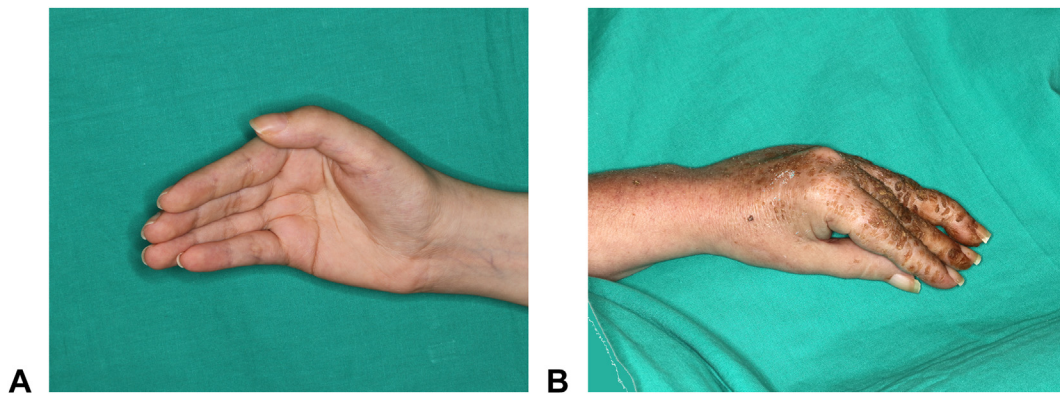


FIGURE 2: The preoperative hands of participants 23 and 32 are shown (left and right). Both are CRPS dystonia cases presenting a fixed stance intrinsic plus for more than one year. The patient in **A** achieved a total cure, whereas the patient in **B** exhibited treatment failure.

months vs 2 months). It is unclear why some patients develop digit stiffness or a frozen shoulder, whereas others do not.

Two patients, both dystonic CRPS, did not improve at all after surgery. Overall, there were 10 dystonic CRPS patients in this series.^{22,23} Of these, five were relieved of their pain symptoms (50%), and one had a painless tremor, but no pain (Fig. 2). Three patients relapsed at about 3 months after the operation. In one patient, there may have been a work-related issue, but I am reluctant to invoke this as the cause of relapse, despite the fact that there are several reports in the literature demonstrating secondary gain behind some CRPS cases.^{32,33} Regarding the failures in this series, it is possible that an unidentified nerve other than the median nerve might have been the trigger. In the future, the development of newer devices to measure small fiber conduction may help to record pain and isolate the exact zone of injury.³⁴

This case series has several limitations. Some may consider that any observational study is flawed; however, randomization was felt to be infeasible in evaluating a new approach to a given condition and of unclear value when the outcomes of the intervention are dramatic.^{35,36} Observational studies are strengthened by single observers; however, this also may have been a cause of bias and poor generalizability. Nevertheless, validated patient-reported questionnaires (DASH) were administered, and objective measurements (palm to pulp distance) taken. Finally, although subjective, pain, rated on a scale of 0–10, is still considered the gold standard for reporting pain and allows comparison with other studies.³⁷ The possibility of potential confounding of the results by concomitant surgery, although improbable, should also be taken into account when

assessing the findings. A major concern is the lack of a control group, and some may consider that the patients may have improved by other treatment modalities or on their own, particularly the early forms. However, recent investigations have shown that with classic approaches there is a high failure rate and a high rate of sequelae in acute CRPS cases at 1 year.^{29–31} By the same token, as discussed, patients in the chronic stage have a low rate of recovery. Conversely, the strengths of this study are the number of cases of this comparatively rare condition, including the number of cases in the chronic stage, and that all patients had a similar treatment protocol.

This study does not clarify the pathophysiology of the condition, although it clearly identifies the carpal tunnel as the trigger site of the majority of CRPS 1 cases of the upper limb, rather than central nervous system, inflammatory, or autoimmune causes.^{1,2} Speculating on the mechanism of the CRPS, nerve compression seems unlikely, as in no case was the median nerve observed to be deformed at surgery, and the EDS were unhelpful. Only two cases showed severe nerve compression. It is possible that in CRPS the nerves are sensitized for unknown reasons and minimal degrees of compression on the median nerve can trigger major symptoms. Alternatively, a mechanical irritation because of the tendons rubbing against the nerve owing to lack of gliding is more likely.³⁸ Lack of nerve gliding has been associated with allodynia which disappears once free gliding has been achieved, improving range of motion of the wrist and even the shoulder.^{39,40} Considering this most plausible mechanism, the condition could be considered as irritative carpal tunnel syndrome (ICTS).

The clinical findings and presentation of irritative CTS and CTS are remarkably different (Table 4).

TABLE 4. Clinical Signs and Symptoms in Carpal Tunnel Syndrome and Irritative Carpal Tunnel Syndrome

Signs and Symptoms	Carpal Tunnel Syndrome	Irritative CTS
Triggering mechanism	None	Trauma/surgery
Main complaint	Numbness	Pain
Sensory symptoms	Paresthesias	Allodynia
Distribution	Median nerve territory	Variable (<20% median nerve territory)
Nocturnal symptoms	Numbness	Pain
Swelling	Rare	Always
Finger flexion	Full	Reduced
Stiffness	Never	Often
Passive finger flexion	Normal	Painful
Tinel	Median nerve distribution	Electroshock in all directions
Phalen	Positive	Impossible often
Durkan	Positive	Positive
EDS	Positive for carpal tunnel	Normal

Few of our patients would comply with any of the CTS-6 items used in the diagnostic process for CTS.⁴¹ The only condition likely to be related to irritative CTS is the one described by del Piñal,⁴⁰ whose patients also presented lack of finger flexion and allodynia as main features. However, none of those patients have sustained previous trauma nor were diagnosed with CRPS.

In conclusion, the current study demonstrates that CRPS 1 may respond to division of the transverse carpal ligament in a large proportion of cases. Further research is needed to understand the pathophysiology, but this work directs future investigations to a local problem at the carpal tunnel.

CONFLICTS OF INTEREST

No benefits in any form have been received or will be received related directly to this article.

REFERENCES

- Marinus J, Moseley GL, Birklein F, et al. Clinical features and pathophysiology of complex regional pain syndrome. *Lancet Neurol*. 2011;10(7):637–648.
- Birklein F, Ajit SK, Goebel A, Perez RSGM, Sommer C. Complex regional pain syndrome-phenotypic characteristics and potential biomarkers. *Nat Rev Neurol*. 2018;14(5):272–284.
- Ferraro MC, Cashin AG, Wand BM, et al. Interventions for treating pain and disability in adults with complex regional pain syndrome- an overview of systematic reviews. *Cochrane Database Syst Rev*. 2023;6(6):CD009416.
- Harden RN, McCabe CS, Goebel A, et al. Complex regional pain syndrome: practical diagnostic and treatment guidelines, 5th edition. *Pain Med*. 2022;23(suppl 1):S1–S53.
- Harden NR, Bruehl S, Perez R, et al. Validation of proposed diagnostic criteria (the “Budapest Criteria”) for Complex Regional Pain Syndrome. *Pain*. 2010;150(2):268–274.
- Goebel A, Birklein F, Brunner F, et al. The Valencia consensus-based adaptation of the IASP complex regional pain syndrome diagnostic criteria. *Pain*. 2021;162(9):2346–2348.
- Schwartzman RJ, Erwin KL, Alexander GM. The natural history of complex regional pain syndrome. *Clin J Pain*. 2009;25(4):273–280.
- De Mos M, Huygen FJ, van der Hoeven-Borgman M, Dieleman JP, Ch Stricker BH, Sturkenboom MCJM. Outcome of the complex regional pain syndrome. *Clin J Pain*. 2009;25(7):590–597.
- Mitchell SW, Morehouse GR, Keen WW. The classic. Gunshot wounds and other injuries of nerves. *Clin Orthop Relat Res*. 2007;458:35–39.
- van de Vusse AC, Stomp-van den Berg SG, Kessels AH, Weber WE. Randomised controlled trial of gabapentin in complex regional pain syndrome type 1 [ISRCTN84121379]. *BMC Neurol*. 2004;29(4):13.
- Varena M, Adami S, Rossini M, et al. Treatment of complex regional pain syndrome type I with neridronate: a randomized, double-blind, placebo-controlled study. *Rheumatology (Oxford)*. 2013;52(3):534–542.
- Patterson RW, Li Z, Smith BP, Smith TL, Koman LA. Complex regional pain syndrome of the upper extremity. *J Hand Surg Am*. 2011;36(9):1553–1562.
- Bruehl S. Complex regional pain syndrome. *BMJ*. 2015 Jul 29;351:h2730.
- Kemler MA, Barendse GA, van Kleef M, et al. Spinal cord stimulation in patients with chronic reflex sympathetic dystrophy. *N Engl J Med*. 2000;343(9):618–624.
- Sigtermans MJ, van Hilten JJ, Bauer MCR, et al. Ketamine produces effective and long-term pain relief in patients with complex regional pain syndrome type I. *Pain*. 2009;145(3):304–311.
- Zyluk A, Puchalski P. Pain control in chronic, refractory CRPS by continuous brachial plexus analgesia. *Handchir Mikročhir Plast Chir*. 2018;50(3):190–195.
- Jupiter JB, Seiler JG III, Zienowicz R. Sympathetic maintained pain (causalgia) associated with a demonstrable peripheral-nerve lesion. Operative treatment. *J Bone Joint Surg Am*. 1994;76(9):1376–1384.
- Dellon AL. Surgical treatment of upper extremity pain. *Hand Clin*. 2016;32(1):71–80.
- del Piñal F. Outcomes of carpal tunnel release in complex regional pain syndrome/reflex sympathetic dystrophy/Sudeck disease patients. *Plast Reconstr Surg*. 2022;150(1):93–101.
- del Piñal F. Diagnosis and outcomes of 225 consecutive cases of complex regional pain syndrome of the hand. *Plast Reconstr Surg*. 2023;152(4):807–816.

21. Agha RA, Borrelli MR, Farwana R, et al. The PROCESS 2018 statement: updating consensus preferred reporting of case series in surgery (PROCESS) guidelines. *Int J Surg*. 2018;60:279–282.
22. Bhatia KP, Bhatt MH, Marsden CD. The causalgia-dystonia syndrome. *Brain*. 1993;116(4):843–851.
23. Schrag A, Trimble M, Quinn N, Bhatia K. The syndrome of fixed dystonia: an evaluation of 103 patients. *Brain*. 2004;127(10):2360–2372.
24. Żyluk A, Mosiejczuk H. A comparison of the accuracy of two sets of diagnostic criteria in the early detection of complex regional pain syndrome following surgical treatment of distal radial fractures. *J Hand Surg Eur Vol*. 2013;38(6):609–615.
25. Hoffmann R, Siemionow M. The endoscopic management of cubital tunnel syndrome. *J Hand Surg Br*. 2006;31(1):23–29.
26. Atkins RM, Duckworth T, Kanis JA. Features of algodystrophy after Colles' fracture. *J Bone Joint Surg Br*. 1990;72(1):105–110.
27. Bickerstaff DR, Kanis JA. Algodystrophy: an under-recognized complication of minor trauma. *Br J Rheumatol*. 1994;33(3):240–248.
28. Żyluk A. The natural history of post-traumatic reflex sympathetic dystrophy. *J Hand Surg Br*. 1998;23(1):20–23.
29. Savaş S, Baloğlu HH, Ay G, Cerçi SS. The effect of sequel symptoms and signs of Complex Regional Pain Syndrome type 1 on upper extremity disability and quality of life. *Rheumatol Int*. 2009;29(5):545–550.
30. Bean DJ, Johnson MH, Heiss-Dunlop W, Kydd RR. Extent of recovery in the first 12 months of complex regional pain syndrome type-1: A prospective study. *Eur J Pain*. 2016;20(6):884–894.
31. Beerthuizen A, Stronks DL, Van't Spijker A, et al. Demographic and medical parameters in the development of complex regional pain syndrome type 1 (CRPS1): prospective study on 596 patients with a fracture. *Pain*. 2012;153(6):1187–1192.
32. Mittenberg W, Patton C, Canyock EM, Condit DC. Base rates of malingering and symptom exaggeration. *J Clin Exp Neuropsychol*. 2002;24(8):1094–1102.
33. Ochoa JL, Verdugo RJ. Neuropathic pain syndrome displayed by malingerers. *J Neuropsychiatry Clin Neurosci*. 2010;22(3):278–286.
34. Terkelsen AJ, Karlsson P, Lauria G, Freeman R, Finnerup NB, Jensen TS. The diagnostic challenge of small fibre neuropathy: clinical presentations, evaluations, and causes. *Lancet Neurol*. 2017;16(11):934–944.
35. Black N. Why we need observational studies to evaluate the effectiveness of health care. *BMJ*. 1996;312(7040):1215–1218.
36. Landewé R, Van Der Heijde D. Primer: challenges in randomized and observational studies. *Nat Clin Pract Rheumatol*. 2007;3(11):661–666.
37. Dworkin RH, Turk DC, Farrar JT, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2005;113(1-2):9–19.
38. McLellan DL. Letter: longitudinal sliding of median nerve during hand movements: a contributory factor in entrapment neuropathy? *Lancet*. 1975;1(7907):633–634.
39. Hagiwara Y, Nakamura T, Sonoki K, et al. “Idiopathic” shoulder pain and dysfunction from carpal tunnel syndrome and cubital tunnel syndrome. *Plast Reconstr Surg Glob Open*. 2022;10(2):e4114.
40. Piñal FD. Hand Allodynia, Lack of finger flexion, and the need for carpal tunnel release. *J Hand Surg Am*. 2023;48(4):370–376.
41. Graham B. The value added by electrodiagnostic testing in the diagnosis of carpal tunnel syndrome. *J Bone Joint Surg Am*. 2008;90(12):2587–2593.