Demystifying CRPS

Early Detection and Multidisciplinary Management in Primary Care

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Learning objectives

01

Recognize clinical signs of CRPS

02

Distinguish Type I and Type II by evidence of nerve injury

03

Build a multimodal plan spanning rehabilitation, medication, psychological care, and interventions

Levels of evidence

Level 1

Meta-analyses and systematic reviews

Level 2

Well-powered randomized trials or validation studies

Level 3

Retrospective studies, open-label trials, small pilots

Level 4

Case reports, clinical experience, empirical observations

What CRPS describes

Continuing regional pain disproportionate to the expected course

Distal sensory, vasomotor, sudomotor, motor, or trophic findings

A changing presentation. No single symptom confirms CRPS

Four clinical domains

01 **Sensory**

Allodynia or hyperalgesia

02 **Vasomotor**

Temperature or color asymmetry

03 **Sudomotor / edema**

Swelling or sweating asymmetry

04 **Motor / trophic**

Motion, strength, movement, skin, hair, or nail changes

Budapest clinical criteria

3 / 4

SYMPTOM DOMAINS

2 / 4

OBSERVED SIGN DOMAINS

Continuing disproportionate pain

No other diagnosis better explains the presentation

Budapest criteria: diagnostic performance

99%

Sensitivity

68%

Specificity

Budapest clinical criteria validation study (Harden et al., 2010)

Clinical assessment and exclusion of better explanations remain essential

Regional pain and a changing phenotype

Pain extends beyond a single nerve territory or dermatome

Abnormal findings usually predominate distally

The pattern can evolve over time

Symptoms and signs are different

Symptoms

At least one reported symptom in 3 of 4 categories

Signs

At least one observed sign in 2 of 4 categories

All four domains apply

Sensory, vasomotor, sudomotor / edema, motor / trophic

Observed now

Count a sign only when present at the diagnostic evaluation

Hyperalgesia and allodynia

Hyperalgesia

An exaggerated response to a stimulus that normally causes pain

Allodynia

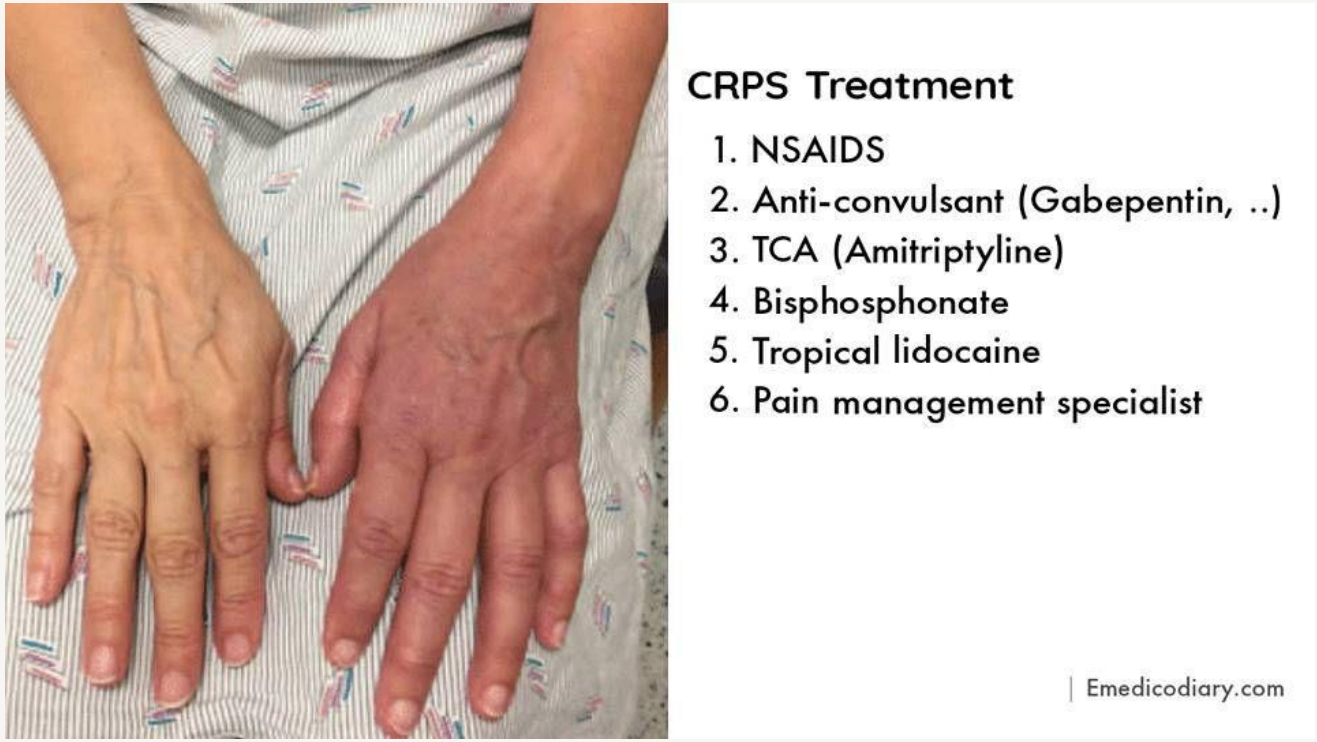
Pain from a stimulus that normally does not cause pain

Vasomotor findings

Color asymmetry

Compare both limbs

Interpret with the full clinical pattern



Trophic changes

Altered skin, hair, or nail appearance

Document changes alongside function and motor findings



Warm and cold CRPS

Warm phenotype

Warm, red, dry, and edematous extremity

Cold phenotype

Cold, blue, sweaty, and less edematous extremity

Informal phenotypes, not a fixed sequence of stages

CRPS with remission of some features

Previously met diagnostic criteria

Improved enough to no longer meet the full criteria

Persistent symptoms may still require ongoing care

Type I and Type II: terminology

Type I

Without established major nerve damage
Historical term: reflex sympathetic dystrophy

Type II

With established major nerve damage
Historical term: causalgia

A focused primary care examination

01 Compare color, temperature, swelling, and sweating

02 Test light touch, pinprick, movement, and range of motion

03 Document strength, motor changes, skin, hair, and nails

04 Record function and repeat the examination over time

CRPS Severity Score

17

CRPS SEVERITY SCORE

8 signs

9 symptoms

Present / absent scoring
Possible total: 0–17

Alternative explanations

01 Infection or inflammatory arthritis

02 Vascular compromise or thrombosis

03 Fracture, nonunion, compartment problem, or focal nerve lesion

04 A new urgent finding changes the workup and referral pathway

Type I and Type II: assessment

Both types	Apply the Budapest clinical criteria
Type II	Establish major nerve injury
Nerve testing	May answer a separate diagnostic question and can be very painful

Early action in primary care

Document the working diagnosis and criteria

Assess urgent alternatives and injury care

Start graded functional use with PT or OT

Follow closely and coordinate pain care when recovery stalls

Functional restoration

Choose a meaningful task: dressing, walking, or hand use

Begin with tolerable activity and advance in measured steps

Address edema, desensitization, motion, and motor control

Track function alongside pain

When movement is difficult

01 Gradual sensory exposure and active motion

02 Consider graded motor imagery or mirror therapy

03 Use pacing to support repeated participation

04 Adjust the team plan to avoid prolonged disuse

Rehabilitation and adjunctive therapies

Functional restoration

PT, OT, recreation therapy, and vocational rehabilitation

Hyperbaric oxygen

One moderate-sized randomized trial reported reduced pain and edema

Acupuncture

Two trials reported mixed findings

Clinical approach

Use a stable, empathetic, interdisciplinary plan

Occupational therapy and daily function

Functional assessment and treatment

A gradual, steady progression of activity

Biopsychosocial principles integrated into rehabilitation

Graded exposure and active motion

Start small	Break a feared movement into manageable steps
Practice function	Reaching, bending, or handling light objects
Progress gradually	Increase range and duration as participation improves

Graded motor imagery

01

Left–right discrimination

Identify right and left body parts

02

Explicit motor imagery

Visualize movement before performing it

03

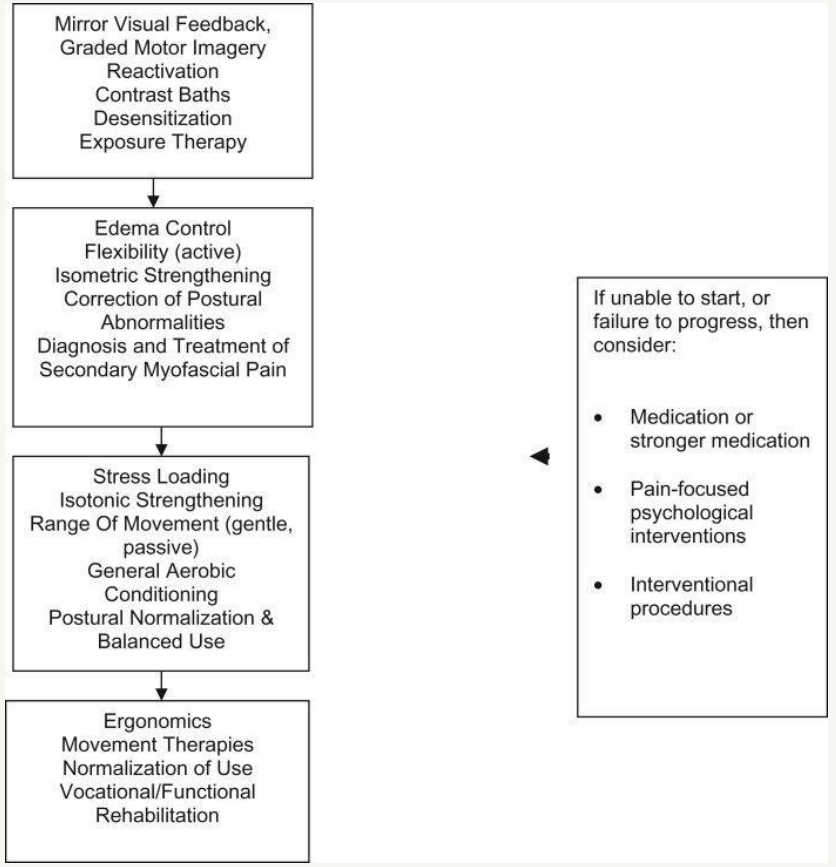
Mirror therapy

Use the unaffected limb’s reflection to support movement practice

Malibu treatment algorithm

Functional restoration remains central

Add supportive treatments when progress stalls



Pharmacotherapy

CRPS-specific trial evidence remains limited

Selected agents may support symptom management

Consider a short steroid course in early inflammatory CRPS

Reassess benefit, adverse effects, and duration



Medication evidence: two contexts

Studied in CRPS

Calcitonin, bisphosphonates, and selected immune-modulating drugs

Studied mainly in neuropathic pain

Antidepressants, anticonvulsants, opioids, and topical agents

Shared treatment goals

01 Set realistic expectations. No drug reliably cures CRPS

02 Choose measurable pain and functional goals before treatment

03 Limit unnecessary medication burden and consider cost

04 Reconsider treatment when benefit is absent or fades

Anti-inflammatory drugs

Options discussed	NSAIDs, COX-2 inhibitors, corticosteroids, free-radical scavengers
Evidence limitation	Research support for NSAIDs in CRPS is lacking
Mechanistic caveat	Neurogenic inflammation may respond differently

Immune modulation

Steroids	Some trial evidence for very early inflammatory presentations
IVIG	A small positive trial was followed by a larger negative trial
Other approaches	Low-dose naltrexone and cetuximab remain investigational in the source literature

Bisphosphonates and anticonvulsants

Bisphosphonates

Some evidence in very early CRPS

Gabapentin

Strong evidence in other neuropathic conditions, limited CRPS-specific evidence

Pregabalin

No CRPS trial data in the cited guideline

Other anticonvulsants

CRPS-specific evidence is sparse

Monoamine reuptake inhibition

Tricyclic / heterocyclic agents	Support descending inhibition and may also address sleep or mood
Evidence context	Strong neuropathic pain evidence does not establish equal benefit in CRPS
Practical considerations	Cost, once-daily dosing, and possible tolerance

Antidepressants: practical considerations

01 Start low and titrate gradually

02 Monitor expected adverse effects and response closely

03 Consider ECG monitoring with tricyclic dose escalation

04 SNRIs have chronic pain indications but lacked CRPS trials in the cited guideline

Opioids: limited benefit and cumulative risk

01 A short morphine trial did not outperform placebo

02 Dose escalation can increase harms without added relief

03 Long-term high doses may worsen pain sensitivity

04 Tramadol and tapentadol also affect monoamine pathways

NMDA receptor antagonists

IV ketamine	Two randomized trials reported benefit with subanesthetic infusions
Limitations	Tolerance, dysphoria, hallucinations, and limited durability
Other routes / agents	No oral ketamine trial evidence in the source. Limited data for amantadine and memantine

Clonidine, nifedipine, and calcitonin

Clonidine	Systematic review found no convincing CRPS benefit
Nifedipine	Uncontrolled case series only
Calcitonin	Multiple trials and meta-analysis, with conflicting results and limited availability

Emerging and topical options

Emerging therapies

Immune modulation, cannabinoids, and botulinum toxin

Topical agents

Lidocaine, capsaicin, DMSO, and selected local anesthetics

Evidence caution

Limited CRPS-specific support. Capsaicin can cause burning

DMSO

Symptoms may improve without a reduction in pain intensity

Treatments when restoration stalls

Reason for inability to begin or progress	Action
Mild to moderate pain	Simple analgesics and/or blocks (see interventional therapy section)
Excruciating, intractable pain	Opioids and/or blocks or later more experimental interventions (see interventional therapy section)
Inflammation/swelling and edema	Steroids, systemic or targeted (acutely) or NSAIDs (chronically); immune modulators
Depression, anxiety, insomnia	Sedative, analgesic antidepressants/anxiolytics and/or psychotherapy (see pharmacotherapy section)
Significant allodynia/hyperalgesia	Anticonvulsants and/or other sodium channel blockers or NMDA-receptor antagonists
Significant osteopenia, immobility and trophic changes[#]	Calcitonin or bisphosphonates
Profound vasomotor disturbance	Calcium channel blockers, sympatholytics and/or blocks (see interventional therapy section)

igure 3. An empiric, consensus-based pharmacotherapy guide (modified by consensus from [3]). The following strategies are sug-ested for patients who have been diagnosed with CRPS but who cannot begin or progress in the functional restoration algorithm level 4)*.
It is important to remember that these general suggestions are overruled by individual patient presentation.
It is important to note that certain drugs (e.g. calcitonin), may be associated with analgesia as well as the more primary action.

Psychological support

Assess fear of movement, sleep, mood, and coping

Use pain education and cognitive behavioral approaches

Include the patient’s goals and barriers

Integrate psychological care with physical and medical care

Interventional therapies

Sympathetic nerve blocks

Brachial plexus blockade

Neurolytic sympathetic procedures

Neurostimulation



Sympathetic nerve blocks

Purpose	May create a less painful opportunity for rehabilitation in selected patients
Evidence	High-quality evidence for a definitive CRPS role is lacking
Assessment	Technical block success and meaningful clinical benefit are distinct questions

Regional blocks and infusion techniques

Brachial plexus infusion

Case-report evidence with catheter and procedural risks

Epidural infusion

Titrated analgesia may support active rehabilitation

Intrathecal therapy

Limited support, particularly without severe dystonia

Risk assessment

Bleeding, infection, and other procedural complications matter

Sympathectomy and neurostimulation

Ablative sympathectomy

Limited evidence, recurrence, and post-sympathectomy neuralgia

SCS and DRG stimulation

Consider when interdisciplinary restoration and noninvasive care fail to progress

DRG and motor cortex stimulation

Dorsal root ganglion

Targets sensory pathways near the spinal cord for focal pain regions

Motor cortex

Neurosurgical electrode placement over the motor cortex. Highly invasive and off-label in the source literature

Evidence and clinical practice

Diagnosis and outcome measurement have advanced

Convincing evidence remains limited for many interventions

Interdisciplinary pain and symptom management remains essential

Three clinical takeaways

01

Use Budapest criteria Document observed signs

02

Verify nerve injury before labeling Type II

03

Begin coordinated functional restoration early

Discussion

What findings prompt you to suspect CRPS?

Which better explanations must you address today?

Who will coordinate the next month of care?

Reference

Harden RN, McCabe CS, Goebel A, et al.

Complex Regional Pain Syndrome: Practical Diagnostic and Treatment Guidelines, 5th Edition

Pain Medicine. 2022;23(Suppl 1):S1–S53

doi:10.1093/pm/pnac046