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REVIEW

# Reflex sympathetic dystrophy—a complex regional pain syndrome

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**Abstract**

Reflex sympathetic dystrophy (RSD) is a complex and poorly-understood condition characterized by: (a) pain and altered sensation; (b) motor disturbance and soft tissue change; (c) vasomotor and autonomic changes; and (d) psychosocial disturbance. Neurological symptoms typically do not conform to any particular pattern of nerve damage. Many different names have been ascribed to this condition and most recently the term 'complex regional pain syndrome' has been coined to emphasize the complex interaction of somatic, psychological and behavioural factors. Diagnostic criteria have been proposed by the International Association for the Study of Pain, but are still subject to debate. This review article describes the clinical features which may present as part of the condition, and the patho-physiology and pre-disposing factors so far identified. The evidence for effectiveness of different interventions is presented and a treatment approach outlined for inter-disciplinary management. While RSD is traditionally associated with pain in the extremities, the possibility is raised that the same process may underlie chronic pain syndromes affecting more central structures, such as testicular or pelvic pain.

## Introduction

Reflex sympathetic dystrophy (RSD) is a complex and often poorly-understood condition characterized by:

(a) pain and altered sensation;
(b) motor disturbance and soft tissue change;
(b) vasomotor and autonomic changes; and
(b) psychosocial disturbance.

RSD may affect either upper or lower limb, may be localized, or may involve the whole extremity. Rarely it affects more than one limb.[1, 2] Neurological symptoms typically do not conform to any particular pattern of nerve damage.

This review article represents an assimilation of the literature to describe the condition, its diagnosis and an approach to rehabilitation.

## Terminology

A number of different names have been given to this condition including:

- Reflex sympathetic dystrophy (RSD);
- Algodystrophy;
- Sudek's atrophy;
- Shoulder-hand syndrome (SHS);
- Sympathetically-maintained pain (SMP);
- Complex regional pain syndrome (CRPS); and
- Causalgia.

The multiple terms are often confusing and make literature searching difficult. Some are applied to specific subgroups (e.g. Sudek's atrophy which describes the condition of RSD in the hand following Colles fracture or causalgia which derives from the Greek '*causos*' or 'heat' and describes the burning pain sometimes associated with neuropathic damage). Others (e.g. Complex regional pain syndrome) attempt to describe the general condition.

Until recently 'Reflex sympathetic dystrophy' was the most popular term. The reflex nature is unclear, however and the role of sympathetic system not well understood, so the term 'Complex regional pain syndrome' has been coined to emphasize the complex interaction of somatic, psychological and behavioural factors and the regional (i.e. non-localized) distribution of symptoms.[3] The International Association for the Study of Pain (IASP) has recently attempted to develop criteria to improve the

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diagnosis of CRPS. However, these have not been fully validated and need further work.

At the current time, 'Reflex sympathetic dystrophy' is the term more widely used in the literature and recognized by clinicians other than pain specialists. It is not entirely clear that all which is described in that huge literature on RSD would conform to the criteria for CRPS. This review leans heavily on the RSD literature, so that term will be used for the current purpose.

## Clinical features

The main clinical features of RSD are listed in table 1.[3–5] The pattern of presentation may alter somewhat with time and the clinical course is commonly divided into three stages:[6, 7]

(1) acute or hyperaemic;
(2) ischaemic; and
(3) atrophic.

In the early stages, vasomotor changes may be very prominent with acute pain, swelling, colour change and a 'hot' bone scan. In the later stages, atrophic changes predominate with pallor, contracture, and osteopenia, which may result in part from learned disuse.[3]

## Pathophysiology

The often-striking response of RSD to sympathetic blockade together with signs of autonomic dysfunction supports the notion that it represents an exaggerated sympathetic response to post-traumatic inflammatory responses, rather than the consequence of sympathetic damage.[8] The concept of sympathetically-maintained pain is supported by human and animal experiments, which demonstrate that catecholamines can activate primary afferent nociceptors, together with the fact that sympathetic blockade is sometimes effective in relieving symptoms. However, the complex interaction of psychological factors and failure to demonstrate increased levels of catecholamines and related factors peripherally[9–11] suggests that the processing problem is central rather than peripheral.[3]

## What triggers RSD?

In 95% of cases there is some history of trauma or surgery.[3] However, there is little relationship between the severity of the condition and of the original insult. Typically it follows minor injury or surgery.[12]

**Table 1** Clinical features of reflex sympathetic dystrophy

*Pain and altered sensation*
(1) Severe and very unpleasant pain
- often graphically described as 'knife-life' or 'burning'

(2) Altered sensation with
- numbness
- paraesthesia (pins and needles)
- hyperalgesia (increased sensitivity to painful stimuli)
- allodynia (normal stimuli perceived as pain)

*Motor disturbance*
(1) Spasms—which may take the form of
- dystonic posturing[4]
- myoclonic type jerks at night—otherwise known as 'restless legs'[5]

(2) Restriction of joint movement
- usually limitation of flexion
- joint tenderness/effusion
- soft tissue contractures—develop within a relatively short space of time

*Vasomotor/autonomic changes*
- Variable skin colour changes from pallor to redness or purple, blotchy discoloration (this may have little relation to the ambient temperature, peripheral pulses are usually maintained)
- Temperature change—may be either warmer or colder than the unaffected limb
- Swelling and oedema—in the affected extremity
- Sweating change—either decrease or increase
- Trophic changes (shiny skin with hair loss)

*Psychosocial*
A complex interaction of psychological and behavioural factors, which may include
- enhanced disability behaviour—reflecting a greater level of dependence than would be explainable by the physical condition alone
- depression
- anger, distress, hostility—not infrequently there may be litigation underway
- physical and vocational inactivity—either preceding or since the onset of symptoms

RSD in the upper limb is reported following carpal tunnel release (whether performed endoscopically[13, 14] or operatively[15]), Dupytrens repair[16, 17] and other tendon release procedures.[18]

RSD in the lower limb is a recognized complication of knee surgery—including tibial osteotomy[19, 20] and arthroplasty[21] where it is associated with a poor prognosis. It is also reported following crush injury to the foot,[22] ankle arthrodesis,[23] amputation[24] and hip arthroplasty.[25]

It is well-described following minor interventions such as arthroscopy, in which there is only minimal trauma.[26, 27] One multi-centre study quotes a frequency of 2.3% following knee arthroscopy.[28] It is also reported following ankle arthroscopy[29, 30] albeit less frequently.

In other reports, RSD has been associated with stroke,[31, 32] mastectomy,[33] pregnancy[34] and ostegenesis imperfecta.[35]

## Predisposing factors

Certain psychological features are reported in association with RSD although it is sometimes difficult to separate cause and effect.

- Patients with industrial injuries have a higher rate of RSD for procedures such as arthroscopy,[36] and a number of studies report high frequencies where 'worker's compensation' insurance is involved.[12]
- There is a strong association with inactivity (including jobless persons and bankrupt self-employed people).[37]
- Patients with RSD report more significant life events in the year preceding onset.[38–40]
- Positive associations are also reported with a psychological history:[41]
  (a) depression;[37, 42–44]
  (c) personality disorders;[38, 45]
  (c) psycho-neuroticism;[38]
  (c) hypochondriasis and hysteria;[46]
  (c) expressed helplessness;[47] and
  (c) 'pain prone-ness'[48] or chronic pain.[49]

RSD is frequently reported in association with an exaggeration of disability or signs, which may vary from simple functional overlay to actual malingering.[50, 51] Results of treatment in compensation cases with pain dysfunction may be poor.[52]

## Diagnosis

The mainstay of diagnosis relies on the presence of clinical features appropriate to the stage of the condition. There have been no clearly defined criteria for the diagnosis of RSD. However, the IASP has recently developed a new set of diagnostic criteria for CRPS types I (without nerve damage) and II (with nerve damage). The criteria specifically avoid mention of the sympathetic nervous system or responsiveness to blockade. They include:

- Presence of an initiating noxious event or cause of immobilization.
- Continuing pain, allodynia or hyperalgesia disproportionate to the precipitating event.
- Evidence at some time of: oedema; changes in skin blood flow; sweating.
- No other condition which can account for the pain or dysfunction.

The criteria were developed by consensus and have not yet been fully validated. A multicentre study[53] to investigate validity, criticized the criteria which failed to include the motor and atrophic changes. The authors have proposed an experimental revision providing more stringent criteria for research purposes. These are:

- Continuing pain, allodynia or hyperalgesia disproportionate to the precipitating event.
- Symptoms in all four of the following areas:
  (a) pain/sensitivity (hyperalgesia or allodynia);
  (d) vasomotor—assymetry of skin temperature or colour;
  (d) sudomotor/fluid—oedema or sweating change; and
  (d) motor/dystrophic—reduced range of movement, motor patterns or atrophic change; and
- Signs in at least 2 of the four areas.

Both sets of criteria fail to describe psycho-social issues adequately and more work is clearly needed before either is fully accepted.[3]

### INVESTIGATIONS

Standard X-rays typically demonstrate patchy demineralization in the affected part, which may be the result of disuse. Three-phase bone scintigraphy shows increased activity in both the blood pool and static phases.[54]

A comparison of X-ray and bone scintigraphy[55] revealed that bone scans are more sensitive than X-rays, especially in the early phase of the condition. Three-phase scans are not necessary, since changes are equally well seen in the static phase. Bone scans appear to be sensitive to treatment and may therefore be used both for initial diagnosis and for monitoring response to treatment.

MRI has more recently gained recognition in diagnosis,[56, 57] especially where radiology is contra-indicated, e.g. in pregnancy.[34] The following features are described:

- periarticular marrow oedema—often involving more than one bone;
- soft tissue swelling;
- joint effusions;[58] and
- a subchondral band of low T1-weighted intensity is a rare finding of questionable significance.

Infra-red telethermography is reported to be sensitive and specific[59] but is rarely used in clinical practice. Dexa scans are not useful for diagnosis, but may have a role in monitoring progress.[60]

## Management

If prevention is the best form of treatment, attention to analgesia and early mobilization post-operative may help to avoid the problem in the first place.[61, 62] Peri-operative guanethideine blockade is not shown to have any protective effect, however.[63]

Once developed, early recognition and proactive treatment are generally acknowledged to be associated with the best outcomes.[64] In the acute phase, sympathetic blockade can be strikingly effective, if short-lived, but may provide a useful window of pain-relief in which to encourage exercise and physiotherapy. Active physiotherapy is shown not only to be effective, but cost-effective.[65–67] Treatment in the second or third stages of the disease is associated with a poorer outcome.[68]

### MEDICAL/SURGICAL INTERVENTIONS

A number of medical interventions have been tried with varying success (see table 2). The evidence for effectiveness has been reviewed by Kingery.[69]

It is recognized, however, that medical management alone is insufficient to sustain benefit.[83] Sympathetic blockade can provide effective relief of symptoms in the short-term, but the effects are not long-lasting and symptoms inevitably recur. Permanent destruction of sympathetic nerves is currently considered inadvisable.[84] Here, too, symptoms recur, but now cannot even be blocked.[3]

There are relatively few properly controlled trials of these techniques, and this is a condition where the placebo effect may be expected to be strong. Low-tech treatments such as sympathetic block or analgesia may provide a useful window of pain relief during which to win the patients' confidence and engage them in a rehabilitation programme.[85] Hi-tech treatments such as spinal cord stimulation should not be applied, other than as part of research trials, until their benefit and cost-effectiveness are proven.[3]

**Table 2** Medical/surgical interventions reported to be of benefit in RSD

*Sympathetic blockade*
- by intravenous injection with various agents[70]
- by infiltration of the symapthetic plexus with local anaesthetic[71]

*Destruction of the local sympathetic supply*
- by dorsal or lumbar sympathectomy[72–74]
- by radiofrequency lumbar sympatholysis[75]

*Bisphosphonates*[76]
*Calcitonin*[77]
*Intra-thecal morphine*[78]
*Corticosteroids, given*
- intravenously[79]
- intra-muscularly[80]
- orally[32]

Spinal cord stimulation[81,82]

## Inter-disciplinary approach to rehabilitation

In this complex condition, the longer symptoms and behaviours persist unchecked, the greater the interaction of psychosocial factors, and the worse the prognosis. A co-ordinated inter-disciplinary approach is needed.[86] Psychological support is reported to have a strongly positive effect on treatment outcomes and a cognitive behavioural approach with clearly functional goals and self-management techniques are recommended to maintain long-term independence.[87]

### TREATMENT GOALS INCLUDE:[3]

- Reduction of pain—if possible;
- reduction of pain behaviour;
- reduction of inappropriate use of health services—many patients go from doctor to doctor in search of a cure;
- increased function of the affected limb;
- increased quality of life and self-esteem; and
- improved psycho-social functioning and relationships with family/friends.

### PROGRAMME STAGES[3]

To understand rehabilitation of the patient with RSD it is necessary to understand the sequence of events which occurs during its development (see figure 1). The critical stages of the programme are:[87, 88]

#### *Engage the patient*

First of all it is necessary to engage the patient—they need to understand that the key to getting better is to use the limb, despite their experience that to do so hurts. Medical interventions can provide a window of pain relief during which the exercise programme gets underway.

#### *Desensitization*

Subject the patient to regular sensory stimulation, gradually building up both duration and intensity of stimulation. The rate of increase should be under the

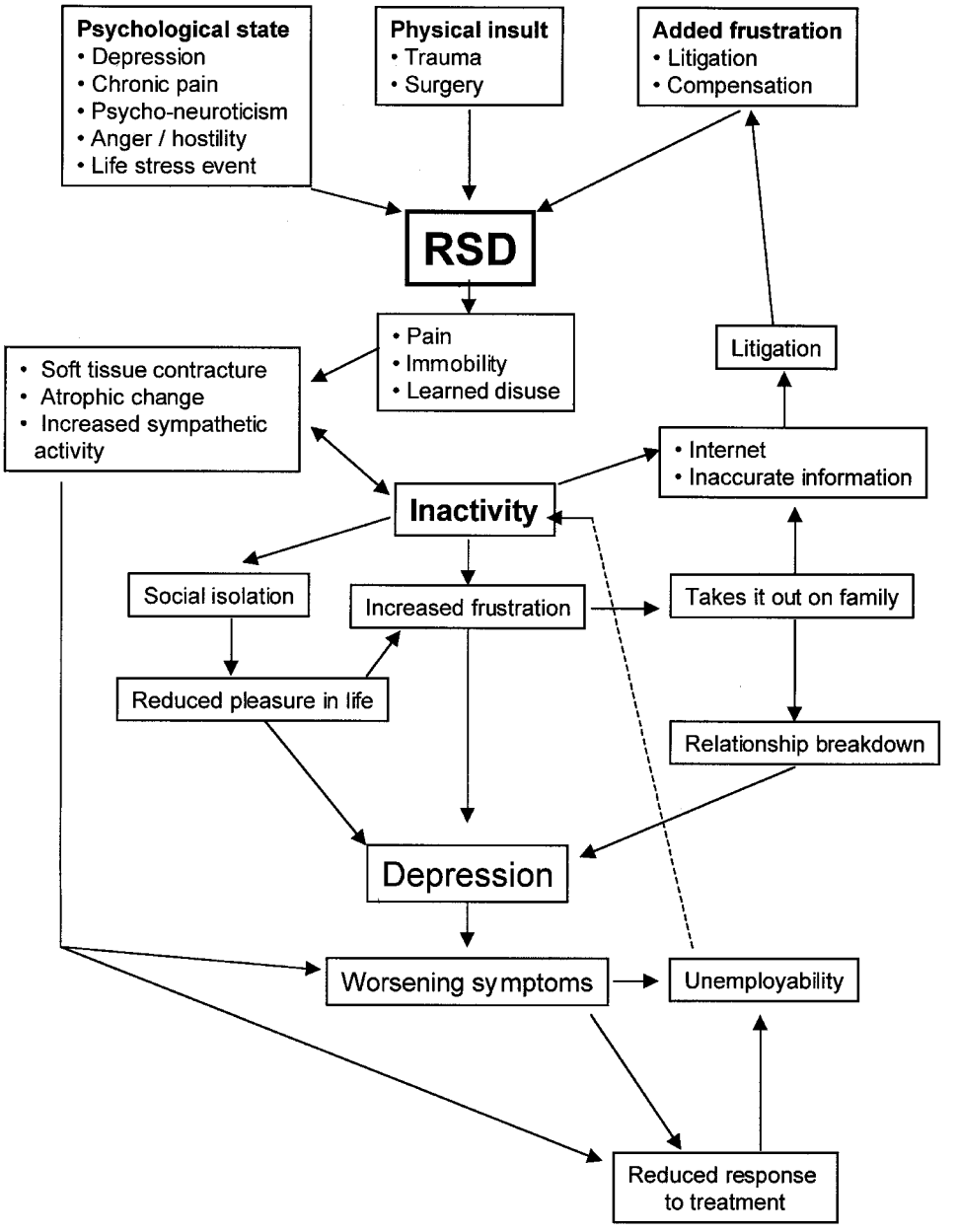


**Figure 1** Complex psychosocial/behavioural interaction deterioration in RSD

patient's control, according to clear goals which they are encouraged to set for themselves.

*Flexibility*

Physical techniques are applied to control oedema, increased range of movement and build up muscle strength with isometric exercises. Initially the programme of stretches and exercises may be passive but, as soon as possible, responsibility is passed over to the patient to do their own programme of stretches and exercises, again with a clear set of goals.

*Postural balance*

Encourage the patient onto their feet for standing and balance control exercises (in lower limb RSD), aerobic conditioning and isotonic exercises.

*Movement*

Goals are now extended towards regaining normal movement patterns of dynamic function such as walking or dextrous movements. Mirrors and biofeedback techniques may be useful to increase awareness of undesired movement patterns and encourage the patients to self-monitor and correct them.

*Vocational rehabilitation*

Finally, once the best possible function is achieved, patients are followed through to apply that function in the workplace, if necessary modifying the job or work environment to enable productive activity. This is particularly important, since inactivity is seen to be a major contributor to the occurrence of RSD. If this stage is ignored, relapse is more likely. If productive work is impossible, efforts should focus on leisure and social activities instead.

*Psycho-social/behavioural rehabilitation*

Also crucial to the success of the rehabilitation programme is education of the patient and their families about the nature of the condition and how their behaviours can either perpetuate or improve it. The psychosocial issue must not be swept under the carpet. The patient needs to understand and accept the part played by these factors, how they affect their symptoms and what they can do about it.

They need to understand that theirs is a normal reaction to abnormal circumstances. They and their families need to be taught techniques for stress management, coping strategies, pacing and variation of activities, and reduced pain behaviour. Their families often need permission to withdraw and let the patient do tasks for themselves.

*Other specific interventions*

- Depression: if the patient is considered to be depressed, this should be treated with SSRIs or tricyclic anti-depressants. Many recommend the use of amitriptyline because of its supposed specific action on pain, but there is no hard research evidence to support this view.[3]
- Law-suits: if the patient is engaged in a law-suit, this should be facilitated to its conclusion as soon as possible. Litigation fuels anger, bitterness, stress and increases sympathetic arousal—rehabilitation is unlikely to be successful until litigation is completed one way or the other.[3]

## RSD affecting other parts of the body

RSD has traditionally been thought of as affecting the limbs only. However, several authors have questioned whether there may be proximal manifestations of the condition.

Muller and colleagues[89] have recently suggested that frozen shoulder may be a manifestation of RSD and Julian and colleagues[90] have asked the same question with regard to vulvar vestibulitis.

Pelvic pain has frequently presented the medical profession with a diagnostic and management conundrum. In some cases there is a clearly identifiable cause such as endometriosis, or chronic pelvic inflammatory disease, but in some there is no clearly identifiable cause despite intensive investigation. Such patients quite frequently demonstrate the following features in common with RSD:

- a disproportionate level of distress and psychosocial dysfunction;[91]
- an initiating surgical procedure—often minor,[92] and around which there may be litigation; and
- a previous psychological background.[93]

It now seems likely that chronic regional pain syndromes may account for at least some cases of pelvic pain, and possibly also some cases of testicular or chronic abdominal pain. In some cases there may also be a sympathetically-maintained element to the pain and this may account for the reported success of testicular denervation (which includes the sympathetic supply) in some cases of intractible testicular pain.[94, 95] Either way, since effective treatment options are lacking, and since the condition may escalate into psycho-social breakdown and catastrophe if not handled effectively, a similar process interdisciplinary rehabilitation with strong cognitive/behavioural elements would appear to be a logical and sensible approach.

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