Introduction

The expected response to injury is an orderly and predictable healing process, with return of function, circulatory dynamics and a gradual cessation of pain. This predictable response to injury does not always occur and injury can instead result in Complex Regional Pain Syndrome (CRPS).

The condition comprises of four cardinal features: pain is the most distressing to the patient and classically it is out of proportion to the degree of injury. The other features are swelling, movement abnormalities (including joint stiffness), and colour, temperature and sudomotor changes collectively known as vasomotor instability.

In 1864 Silas Weir Mitchell first described the condition in soldiers following gunshot injuries sustained in the American Civil War and termed it causalgia (Richards, 1967). Südeck in 1901 described the loss of trabecular bone architecture most seen peripherally in the peripheries which later became Südeck’s atrophy (Nonne, 1901). The lack of full understanding of the aetiology and pathogenesis of the condition has lead to a plethora of names – 67 English language terms! (Veldman, 1996).

Reflex sympathetic dystrophy (RSD) is the most commonly used alternative name because of the symptoms of colour changes and sweating being potentially sympathetically mediated. Early uncontrolled studies indicated that the condition responds to treatment with sympathetic blockade (Hannington-Kiff, 1977, Leriche, 1926), which encouraged the belief that the sympathetic nervous system was involved. Because some patients do not respond to this treatment and it was important to distinguish this condition and its symptoms and signs from other causes of pain (Stanton-Hicks et al., 1995), the International Association for the Study of Pain (IASP) in 1994 coined the term complex regional pain syndrome (CRPS). CRPS is divided into two types: CRPS type I where there is no obvious nerve damage and CRPS type II where there is identifiable nerve damage (causalgia) (Stanton-Hicks et al., 1995).

Using the IASP criteria, patients must have continued disproportionate pain and one symptom in three of four categories (sensory, vasomotor, sudomotor, and motor), and one sign in two of the same categories. There must be no other diagnosis that better explains the symptoms and signs.

Keywords
Complex regional pain syndrome, Reflex sympathetic dystrophy

Date received: 23rd June 2011; revised 15th November 2012; accepted 21st November 2012
Because these criteria were not accurately validated and lacked specificity and thus lead to over diagnosis the criteria were altered, and the 'Budapest criteria' [Harden et al., 2010.] are now recommended for diagnosis [Table 1]. These criteria do not distinguish the severity of the condition nor relate to the prognosis or treatment and it must be noted that these criteria are used in the pain clinic setting only and are sometimes difficult to use in the clinical setting [Atkins, 2010]. As some form of nerve injury can be difficult to exclude in CRPS I, it is questioned whether it is necessary to distinguish between the two types [Oaklander et al., 2006, Harden, 2010]. As the symptoms and signs of both types of CRPS are similar the use of the terms 'high flow' CRPS (or warm CRPS) and 'low flow' (or cold CRPS) are being used more often. High flow CRPS, describing the hot, painful oedematous extremity, and low flow CRPS the cool, atrophic limb.

Incidence

Using the clinical criteria described in Table 2, Sandroni et al. (2003) initially reported an incidence of 5.5 per 10^5 person years at risk in the USA. In the Netherlands this was found to be four times higher: 26 new cases of CRPS per 10^5 population [de Mos et al., 2007]. The difference was attributed to differences in case definition and validation. CRPS is between two and four times more common in women, the median age of onset is between 40–53 years [Allen et al., 1999, de Mos et al., 2007], and post menopausal women appeared to be at the highest risk of developing the condition. The upper limb is most frequently involved [Duman et al., 2007, Veldman et al., 1993]. It has been reported to be rare in black people – but this epidemiological study was performed on an American population that was 80% Caucasian [Sandroni et al., 2003].

Clinical Features

It manifests itself typically within a month of injury, but may be recognised as early as 2 weeks following injury [Field and Atkins, 1997, Zyluk and Puchalski, 2013] with burning or searing pain, early oedema and increased heat and redness ('high flow' or warm CRPS) and later with joint stiffness and contracture ('low flow' or cold CRPS). There is a spectrum of severity in this condition with some cases having milder symptoms and recovering more quickly [Zyluk and Puchalski, 2013], and those with more severe symptoms resolving more slowly or not at all. This latter group may be the chronic refractory group suggested by Zyluk and Puchalski [2013]. There are four cardinal clinical features of CRPS:

1. Pain

Pain is neurogenic in nature being described as burning, nagging, or throbbing. It is the most significant of all the features. There are three clinical terms that are used to describe the pain:

1. hyperalgesia – increased sensitivity to noxious stimuli – i.e. a pin prick causing very severe pain.
2. allodynia – pain provoked by non noxious stimuli – i.e. stroking the skin causing pain.
3. hyperpathia – temporal summation of allodynia – i.e. repeated stroking causing increased pain.

Table 1. Budapest Criteria for CRPS [Harden et al., 2010]

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>Disproportionate to inciting event</td>
</tr>
<tr>
<td>Sensory</td>
<td>Hyperaesthesia/allodynia</td>
</tr>
<tr>
<td>Vasomotor</td>
<td>Temperature/colour changes – asymmetry</td>
</tr>
<tr>
<td>Sudomotor</td>
<td>Oedema/sweating changes – asymmetry</td>
</tr>
<tr>
<td>Motor/Trophic</td>
<td>Decreased ROM, weakness, tremor/dystonia</td>
</tr>
<tr>
<td>Sensory</td>
<td>Hyperaesthesia to pin prick</td>
</tr>
<tr>
<td>Vasomotor</td>
<td>Allodynia to light touch</td>
</tr>
<tr>
<td>Sudomotor</td>
<td>Evidence of temperature/colour asymmetry</td>
</tr>
<tr>
<td>Motor/Trophic</td>
<td>Evidence of oedema/sweating asymmetry</td>
</tr>
<tr>
<td></td>
<td>Evidence of decreased ROM, weakness, tremor/dystonia, trophic changes to skin, hair or nails</td>
</tr>
</tbody>
</table>
By definition the pain is out of proportion to the degree of the initiating injury. It is non dermatomal in distribution. Sleep is often disturbed and the pain can be unresponsive to narcotics. It is often so severe that contact with or active movement of the limb is avoided; this has been termed cognitive neglect by some authors (Galer and Jensen, 1999) and altered perception of the affected limb by others (Lewis et al., 2007). The patient protects the limb with the unaffected one. Non-painful stimuli such as sudden noises, loud noises and unexpected movements can elicit a painful response. The impact on the patient’s life can be devastating and indeed crippling. In most cases the symptoms and signs are confined to the injured area, but not all. Synchiria is when the condition also affects the other limb – touching the affected limb hurts the other limb. Lower limb CRPS can affect the genitals and bladder. Upper limb CRPS can give symptoms in the mouth and face. This is thought to be due to the proximity of the foot and the genitals and the hand and the face in the sensory homunculus of Penfield and Rasmussen (1950).

2. Swelling

Swelling occurs early in the condition and generally involves the whole hand. The fingers appear sausage shaped and the patient is reluctant to move them or have them moved (Figure 1). Later the swelling diminishes, but even when it does there may be a perception that the hand is still swollen and larger than the opposite side, a ‘body perception change’ (Moseley, 2005). It is because of the swelling that tight plasters have been implicated erroneously in the condition (Field et al., 1994).

3. Movement Abnormalities

These can involve joint stiffness and/or motor abnormalities. The term stiffness covers three aspects of joint movement, firstly lack of movement potentially caused by swelling or pain (Watson-Jones, 1940). Secondly a sensation of resistance to movement (a sensation is a feeling felt by the patient or the examining doctor) occurring at all stages of the disease. Lastly stiffness can refer to a fixed joint contracture.
Motor abnormalities may include muscle weakness in the affected limb (prevalence 70–95%, Janig and Baron, 2003), tremor (which is present in half the patients), and even dystonic movements which are rarer but are present in around 10% (Wasner et al., 2003).

Why the stiffness and or motor abnormalities occur remains unclear. The lack of movement may be protective in that movement causes pain thus the patient does not move the affected limb. Movement is known to evoke pain in patients with chronic painful disease presumably because movement activates nociceptors. In CRPS even thinking about moving (motor imagery) can itself cause pain and potentially hinder movement (Moseley, 2004, Moseley et al., 2008).

4. Vasomotor Instability

Early in the condition the limb shows colour changes (often pink or red, but can be blue), temperature changes and alterations in sudomotor activity with one hand sweating more than the other (Figure 1). From a medicolegal perspective it is important to distinguish these alterations of colour and temperature from simple dependency and immobilisation of the hand (Singh and Davis, 2006). In addition, alteration in nail and hair growth can occur, they are often increased on the affected side (Figure 2) but may be reduced (McCabe and Blake, 2008).

Investigations

The diagnosis is clinical; there is no diagnostic test. It can be aided by other investigations such as radiographs where changes involve diffuse osteopaenia which may be peri-articular as described by Südeck. Radiographs are not a sensitive test. 30% of cases show no radiographic change. Bone scanning is not a specific test (19% at best), but is sensitive (96%) early in the condition (Lee and Weeks, 1995, Schurmann et al., 2007). MRI shows bone oedema on T2 weighting but is similar to the bone scan, being specific but not sensitive (Schurmann et al., 2007).

Thermography (measuring temperature differences between the limbs) (Schurmann et al., 2007), isolated cold stress testing (Koman et al., 1984), and sudomotor tests (resting sweat output and thermoregulatory sweat testing) (Chelimsky et al., 1995, Sandroni et al., 1998) are all laboratory techniques that have been used to aid diagnosis. They are all sensitive tests that are not specific for CRPS and thus are not useful diagnostically.

Aetiology

There are various precipitating factors involved in the aetiology of the condition. Trauma is the most common cause (Veldman et al., 1993), with fractures accounting for 46% of cases and sprains for another 10–12% (de Mos et al., 2007). The incidence following distal radius fractures is up to 25% although most of these are mild and self limiting (Atkins et al., 1989, Bickerstaff, 1990, Field, 1993, Zyluk and Puchalski, 2013). Unfortunately there is no evidence that alterations in methods of treatment of those fractures abolish it (Sarangi et al., 1993). Nor does the degree of displacement or fracture type appear to make any difference (Bickerstaff 1990, Field, 1993).
Other factors include strokes, heart attacks, inflammatory diseases, smoking and even hereditary factors (raised HLA-DR13 [Van Hilton et al., 2000]). There are some cases where no cause is found [Veldman et al., 1993]. The injury may be iatrogenic. 16% of 140 CRPS cases at the Mayo institute were post-surgical [Pak et al., 1970]. Carpal tunnel decompression was the cause in up to 5% of cases and between 5 and 25% following Dupuytren’s contracture release (Reuben et al., 2006). Thus informed consent should probably mention CRPS as a complication of all upper limb surgery.

But what does the causative incident actually do to cause CRPS?

Symptoms such as altered limb awareness, opposite limb symptoms, and symptoms in the face or jaw have all promoted the concept of there being some kind of psychological affliction in these patients. That CRPS is primarily psychological in origin is, however, without basis [Bruehl, 2001]. In a prospective study of psychological profiles of patients following Colles’ fracture there was no difference between those that did and did not suffer CRPS. There was a tendency for the psychological profile to worsen once the condition had become established (Field and Gardner, 1997) indicating that having the condition may cause a psychological disturbance. Two further studies of CRPS patients have found no difference in personality profile or depression levels when compared to those not developing the condition [Pulchalski and Zyluk, 2005; van der Laan, 1999].

The true aetiology of the condition is controversial and hotly contested. The evidence favours two theories, however many others abound. The first is that the initiating incident could cause inflammation or secondly it could cause neurological damage.

1. Inflammation

In the early phase of the condition the clinical features of rubor, tumor, calor, dolor and function laesa certainly point towards an inflammatory response. Goris [2009] used an inflammatory score to assess the severity and response to treatment following wrist fracture. Systemic inflammatory mediators such as tumour necrosis factor (TNF- alpha), calcitonin gene related peptide (CGRP), interleukin 2 (IL-2) and substance P have been isolated although the authors admit that this may be as a result of neurogenic dysregulation [Schinkel et al., 2009]. Free radicals have been implicated both in causation [Van der Laan and Goris, 1997] and free radical scavengers in treatment [Goris, 1985]. All of which suggest that CRPS may be an exaggerated local inflammatory response to trauma.

2. Neurological

The theory of a neurological abnormality in CRPS type I is the more compelling for two reasons. Firstly the symptoms experienced are so like those in CRPS type II, where there is a specific nerve injury. Secondly the bizarre symptoms associated with pain being experienced in other parts of the body are best explained by cortical reorganisation.

Neuralgic pain associated with CRPS may be explained by damage to small fibre neurons. These thinly myelinated [A-delta] and unmyelinated [C-fibre and sympathetic axons] transmit afferent information from mechanical, thermal and chemical stimuli. They are also responsible for release of vasoactive peptides such as CGRP and substance P, which are present in CRPS. These small fibres are particularly sensitive to injury, and their degeneration often results in pain [Llewelyn et al., 1991]. Nerve conduction studies are not sensitive enough to pick up these abnormalities. Histological examination has found C-fibre degeneration, but no myelinated axon damage in limbs amputated for CRPS [Van der Laan et al., 1998]. Immunological labelling of axonal markers has confirmed small fibre nerve damage in CRPS [Oaklander et al., 2006]. With this small fibre nerve damage come the trophic changes in the skin seen in CRPS (Figure 2). Very similar skin, nail and hair changes occur in small fibre peripheral neuropathies (SFPN).

Small fibre nerve damage may explain the bone changes seen in CRPS. Bone scan and radiographic changes are attributed to increased osteoclastic activity. Osteoclasts are only bone resorbing at low pH which is when small fibre nociceptors will fire. Small fibre secretions are critical for bone formation and maintenance and bone is significantly influenced by small fibre axonal degeneration [Hukkanen et al., 1993]. The pain in long standing or chronic CRPS could be explained by some form of nerve damage as occurs in the long standing pain experienced in post herpetic neuralgia.

Cortical remapping, the perception of symptoms in cortical areas adjacent to the area represented by the injury, is one of the most fascinating conundrums in this condition. Upper limb CRPS symptoms can be felt in the jaw or face, or even in the contralateral limb. Lower limb affliction can be expressed as bladder or genital pain. These symptoms are common and inadequately recognised [Koltzenburg et al., 1999]. They must implicate central pathways in the aetiology of the condition. This melding of areas within the cerebral cortex explains the symptoms that CRPS patients feel of altered perception of the affected limb.

We know that after amputation of the hand the representation of the limb in the cerebral cortex
alters (Farné et al., 2002). Juottonen and others (2002) used magneto encephalography (MEG) to examine sensory cortical responses in CRPS patients and found them reduced by 40%. Maihofner (2003) performed a longitudinal study finding that there was a direct correlation between the extent of pain in CRPS and the degree of cortical reorganisation. These changes reduced as the level of pain reduced and had not returned to normal even after 60 weeks (Maihofner et al., 2004). Clinical correlation with these sensory MEG findings has been found by Forderreuther et al. (2004): when testing touch in digits of CRPS hands 48% were unable to discriminate which digit had been touched compared to 6% in the normal hand. Maihofner et al. (2007) found changes in the contralateral motor cortex with the representation of the hand increasing (as opposed to the sensory cortex where it diminished in size). This increase correlated with reduced dexterity. This is compounded by the findings of Moseley (2005) who demonstrated that the CRPS patients perceive their affected hands as being significantly larger. There is definitely an alteration of perception of the limb in CRPS. Patients pay less attention to the affected limb, have peculiar desires to amputate, and have a mismatch between sensation of the limb and how it looks (McCabe and Blake, 2008). This may be a reason why patients often feel tightness of their plasters of Paris after Colles’ fracture.

Natural History

Bickerstaff (1990) found that 90% of the symptoms of CRPS had resolved at two years following Colles’ fracture. Sandroni (2003) found that 74% of their 74 cases of CRPS resolved in an average of 12 months. Most cases do resolve (Bickerstaff, 1990, Zyluk and Pulchalski, 2013), but there is a significant percentage that do not and it is likely that these patients are the more severely affected although this is not proven. This is addressed in the paper by Zyluk and Pulchalski in this issue. Features of poor finger function and stiffness following Colles’ fractures at three months predicts morbidity at 10 years (Field et al., 1992).

Does it recur?

It is believed that further trauma such as another operation can reactivate CRPS in a person who has already had a previous episode (Schinkel et al., 1996), although this remains unproven. Veldman and Goris (1996) reported a recurrence rate of 9% in 1183 patients over a 3–20 year period, 3% in the same limb and 6% in another limb. 535 were spontaneous recurrences. Zyluk (2004) recorded spontaneous recurrence in five out of 250 cases over a 15-year period. Eight of those 250 patients required further surgery and none developed CRPS. The evidence therefore suggests that it can recur but that further surgery may not be of concern.

Prevention

There is a paucity of hard evidence for the success of interventions that may be used to prevent CRPS. There are two randomised controlled trials of vitamin C treatment following distal radius fractures, one after wrist fracture (Zollinger et al., 1999) and one following operative treatment of wrist fractures (Zollinger et al., 2007). There was a decrease in the incidence of CRPS by 15% in the first and 8% respectively in the treated groups. The dose was 500mg daily for 50 days after injury. It is cheap and has no morbidity so it seems a sensible option.

It has been suggested that CRPS may be less likely after regional than general anaesthesia (Rocco, 1993, Viel et al., 1994). Reuben et al. (2000) noted a reduction in the recurrence of CRPS following stellate ganglion block in a retrospective series of 100 patients (with CRPS already) undergoing surgical procedures. No study has examined the efficacy of stellate ganglion blocks to patients undergoing surgery without a history of CRPS. Clonidine may be effective as an intravenous regional block for preventing recurrence in those patients with CRPS undergoing surgery. It did cause a reduction in incidence compared to a local anaesthetic block alone but there was no placebo arm to this study (Reuben and Wartier, 2004).

A variety of drugs including calcitonin, carnitine, corticosteroids, ketanserin, and mannitol have not been found to be effective prophylactically (Reuben and Wartier, 2004). Anecdotally post-operative gabapentin and amitriptyline have been suggested but there is no evidence of any efficacy for these administrations.

Treatment

Treatment for this condition is confusing, for two reasons: the medical profession is not sure of the condition’s aetiology and it is unsure what it is treating; and although early treatment is thought to be beneficial it is not known how soon after a precipitating event the condition commences, and despite early treatment some patients do poorly (Patterson et al., 2011, Zyluk 2013).
The treatment options include physical therapies, mirror visual feedback, medication and surgery. A multidisciplinary approach to management is important in this condition including a surgeon, a therapist and the pain clinic staff including doctors and psychologists.

**Physical treatment**

Hand therapy is the mainstay of treatment. It should continue until the symptoms have been resolved. The main aim is to prevent late joint contractures. It also prevents secondary weakness, and minimises pathological cortical remodelling (Maihofner et al., 2004). Unfortunately there is little scientific evidence that physiotherapy is effective. Maintaining active and passive joint range, including stress-loading activities and desensitisation, are the main aims of the therapist’s treatment.

**Mirror visual feedback**

Mirror visual feedback (MVF) involves the patient hiding their affected hand behind a mirror. The patient sees their normal hand and also its mirror image (which the patient sees as if it were the affected limb) (Figure 4). Exercising the normal hand looks to the patient as if he or she is exercising the affected hand. This visualisation of the moving mirror image (which the patient sees as the affected hand) actually encourages the patient to move their real affected hand movement. A recent overview has confirmed the efficacy of MVF in CRPS (Ezendam et al., 2009). Moseley (2004) incorporated this technique with graded visual imagery techniques to good effect.

![Figure 4. Demonstrating Mirror Visual Feedback – the image in the mirror the patient sees as their abnormal hand and as it moves as a normal image the patient moves the abnormal hand accordingly.](image)

**Medication**

**Topical Medication.** Free radical scavengers such as Dimethyl sulphoxide (DMSO) in the form of a cream and N-Acetyl cysteine (orally administered) have been used particularly in the Netherlands (Geertzen et al., 1994, Goris et al., 1987). A very elegant controlled study of the application of cream with and without DMSO found DMSO to be significantly more effective but the numbers were very small and the criteria for diagnosis were vague but it seemed that these were early cases only (Zuurmond et al., 1996). Perez et al. (2003) found DMSO to be more effective in warm CRPS and NAC more effective in cold CRPS, although there was not an arm of the study with no treatment. These treatments – the only medications that have a proven track record – are not available in the UK.

**Oral Medication.** Calcitonin, biphosphonates, capsaicin creams and vitamin C have been found to be useful in acute CRPS but they are untested in chronic cases (Price et al., 1998). Paracetamol, anti-inflammatory agents and amitriptyline have not been proven to be of benefit in CRPS (amitriptyline has only been proven effective in non-CRPS pain) (Collins et al., 2000). Gabapentin has been proven to be effective against pain in the first eight weeks but there is no evidence for its use beyond that (van de Vusse et al., 2004). Other anticonvulsants have not been shown to be helpful.

Evidence-based therapeutic guidelines for treating CRPS type I have recently been produced (Perez et al., 2010), all CRPS patients with chronic pain should be offered analgesics, but as in the US there are no drugs specifically approved for the condition (Oaklander and Fields, 2009), drugs used in post herpetic neuralgia are generally recommended.

**Injections.** We know that intravenous regional guanethidine blockade does not work (Livingstone, 2002). Sympathetic blockade was found to be no more effective than saline in a controlled trial, but the local anaesthetic arm had a longer lasting effect (Price et al., 1998). It may be that sympatholytics are beneficial in the acute phase of the condition but not so in chronic cases (Schattsneider et al., 2006). Meta–analysis does not support the use of local anaesthetic sympathetic blockade (Cepeda et al., 2002). The conclusion stated that it ‘is based on poor quality evidence, uncontrolled studies and personal experience and complications from these procedures may be significant’.

**Surgery.** Surgery to the affected limb is rarely indicated for CRPS. Any temptation to amputate should
be strongly resisted as the pain will not be cured [Dielissen et al., 1995]. However if the CRPS patient has carpal tunnel syndrome it is the author’s belief that surgical release will ease the patient’s passage of recovery however there is no scientific proof. Surgical release of finger contractures has not been successful and has at best a 50% improvement [Patterson et al., 2011]. Spinal cord stimulation is ineffective (Kemler et al., 2008).

Medicolegal Aspects

The major concern is that most of the Budapest diagnostic criteria can be feigned. Pain and all the symptoms and signs (apart from the motor trophic signs) can be contrived if a patient wishes. Unfortunately any physician can be fooled by a good actor with internet access.

Motor/trophic signs such as wasting, nail changes, skin atrophy cannot be faked, but may be misdiagnosed as diabetic neuropathy. Tremors and abnormal movements can be feigned.

Signs are more definite than symptoms, so in a medicolegal assessment it is preferable to look for signs that cannot be mimicked such as:

1. pain, but particularly allodynia and hyperalgesia that can be demonstrated. The temporal summation of hyperalgesia is difficult to feign.
2. trophic changes in skin and nails.
3. wasting of muscles.
4. fixed joint contractures.
5. radiographs showing periarticular osteopaenia – although not diagnostic a radiograph showing changes is significant.

There are some ‘red flags’ that I look for but they do not ensure diagnostic accuracy:

1. excessive swelling – check for excessively tight cuffs or ligature marks further up the limb.
2. wrapped-up limbs – these may be to accentuate temperature or colour differences, and bad cases of CRPS would never let anything touch the hand.
3. flexed posture of fingers with no fixed contracture.
4. Alterations of colour and temperature can be achieved from dependency and immobilisation of the hand [Singh and Davis, 2006].
5. Inconsistency of timing and degree of tremors.

Conclusion

CRPS is a well recognised condition and is diagnosed much more frequently. As part of informed consent, surgeons are probably beholden to mention that their surgery may be involved in the causation of the condition.

Look for it early, particularly suspect if a patient with a fracture is in pain in a cast or has had to have the plaster changed because it is too tight. The diagnosis is still often delayed due to physicians not recognising it. The patient can become confused and frustrated, which results in multiple clinic visits and disproportionate use of healthcare resources [Allen et al., 1999]. As there are now clear and validated diagnostic criteria it should be recognised and treated early and proactively by a multidisciplinary team. If the condition is recognised early make sure they are given enough analgesia soon enough and allow them to undergo hand therapy. A good therapist will recognise when and if it is necessary to involve the pain clinic as they see the patient more often than the treating surgeon. This will not cure the patient but in my experience eases their passage through the condition.

Further research into the neurological cortical representation and its alteration in CRPS will hopefully yield greater insight into the understanding of the aetiology of this bizarre but fascinating disease.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of interest

None declared

References

Hannington-Kiff JG. Relief of Südeck's atrophy by regional
Goris RJA. Delayed recovery of reflex sympathetic dystrophy
Goris RJA, Dongen LM, Winters HA. Are toxic oxygen radi-
Geertzen JH, de Bruijn H, de Bruijn-Kofman AT, et al.
Galer BS, Jensen M. Neglect-like symptoms in complex
Forderreuther S, Sailer U, Straube A. Impaired self per-
Field J, Warwick D, Bannister GC. Features of algodystro-
Field J, Gardner FV. Psychological distress associated with
Field J, Atkins RM. Algodystrophy is an early complication
Field J, Prothero DL, Atkins RM. Algodystrophy after Colles’
Hannington-Kiff JG. Relief of Südeck’s atrophy by regional intravenous guanethidine. Lancet. 1977, 1:1132–33.
Rocco AG. Sympathetically mediated pain may be rekindled by surgery under general anaesthesia. Anaesthesia. 1993, 665–79.
Watson-Jones R. Fractures and other bone injuries. ES Livingstone, Edinburgh. 1940; Chapter 3.