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A review of physiotherapy management of complex regional pain syndrome

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Introduction

The physiotherapy management of CRPS mirrors that of most conditions, i.e. although widely advocated there is little in the way of strong scientific evidence to support the use of our interventions. Nearly all the major medical experts in this field advocate the use of physiotherapy but seldom offer much more than a few passing words or a list of techniques. While physiotherapy is supported, a key question is—do we really know what we are doing, or should be doing, with this condition?

Reviewing the physiotherapy literature leaves one realising that there is very little that hasn't been tried. Though not strongly supported by treatment trials, it is likely that modality based therapies as well as activation approaches are of some benefit in this complex and disabling condition. What has been encouraging in the last few years is the recognition of the multifaceted nature of the condition and hence the need for a multifaceted, multidimensional and multidisciplinary management approach. As will be discussed, more rational and better guidelines for therapy are evolving and being published (see Stanton-Hicks et al 1998).

This chapter will review the literature that relates to the management of CRPS in the adult population. There is also a detailed discussion of the implications of the review findings for the formulation of better management strategies.

Historical perspective

The management of this painful condition has been characterised by an approach that was at best aggressive and at worst torture! My (Thacker)

own earliest clinical guidance for treatment of this condition was to be told that I must move the affected hand or foot no matter what the patient felt or complained of. Although one could object to what is basically a semantic discussion, the word *aggressive* is noted repeatedly in the literature in relation to the prescription for physiotherapy. We much prefer the term *proactive*. This aims to develop a forward thinking, patient driven and centered, active approach to therapy which attempts to overcome the negative and frightening physical connotations that are the undoubted legacy of the word aggressive.

Other noticeable aspects of therapy have been the general acceptance that there is some form of sympathetic over-activity/abnormality dominating the presentation of the patient. Many of the interventions proposed over the years have been specifically targeted at such 'over-activity.' Apparent successes reported by advocates of such approaches are often used as evidence to lend support to the concept that the sympathetic nervous system (SNS) is faulty/overactive/blameworthy, thus perpetuating the outdated concept of abnormally increased sympathetic tone (see discussion in previous chapters.)

A further observation arising from a survey of the literature is that the management of pain in CRPS patients is often dealt with medically and within a strict medical model. Also, as a generalisation, most physiotherapy approaches to pain relief focus on TENS and/or acupuncture (but see below.) Is it possible that we have forgotten the therapeutic value of carefully prescribed active movement as an analgesic in these patients? (The reader is advised to consult an excellent paper by Harding, 1999.)

Lastly many accounts prescribe therapy dependent on the stage of the disorder. This is an outdated concept (see Stanton Hicks et al 1995 and Chapter 2.) Current thinking, and the advice suggested here, is that treatment selection should be patient and problem specific rather than reliant on challengeable pathological or theoretical constructs that many now believe to be floored (Stanton Hicks et al 1998).

Review of modalities reported for the treatment of RSD/Causalgia (CRPS Types I and II)

Electrotherapy

There is a lack of detailed information in the majority of studies looking at specific electrotherapy modalities. The following is therefore a necessarily brief discussion of relevant material.

TENS

TENS is the most quoted non-pharmacological modality for pain relief in this condition. Unfortunately, there is little in the way of high quality evidence to support its use. Papers detailing successful pain relief from TENS in CRPS

are almost exclusively single case studies with little guidance on dosage (Fitzpatrick 2000, Thomas 1996). A literature search in April, 2002, revealed only three papers when combining the terms reflex sympathetic dystrophy, complex regional pain syndrome, and TENS.

This leads to obvious problems for the practising clinician who is seeking guidance over treatment selection and application. Extrapolation from successful regimens used for other neuropathic and ongoing pain states may be reasonable, since many of the clinical presentations and underlying mechanisms are similar.

Since the mature literature on CRPS is now more questioning and cautious regarding cause, a degree of care is required where treatment selection and dosage is based on rigid pathological models or dogmatic statements in texts and papers. Some knowledge of underlying pain mechanisms can help in decision making, though. For instance, we now know that pain mechanisms alter and change as time goes on (see previous chapters.) This may help give better explanations for some of the difficulties encountered in treatment choice as well as provide reasons why some patients may respond better than others. Also, TENS settings that excite large myelinated Aβ fibres (i.e. high frequency settings but often low settings too), may, in individuals with mechanically evoked allodynia (extreme hypersensitivity to touch or movement), cause extreme exacerbation of symptoms. Since it is known that central sensitisation (Chapter 3) allows Aβ fibre input to access nociceptor pathways and hence cause pain, this phenomenon can be understood easily. One requirement for successful TENS application in patients exhibiting this high sensitivity response might be for the therapist and patient to search for areas of electrode placement that are beneficial, or better still that can be used in an incremental way to bring about progressive desensitisation. This implies things like graded increases of current as well as progression of electrode placement into areas of heightened sensitivity and reactivity if at all possible.

It is often the case that this type of TENS hypersensitivity is a dominant feature in one patient whilst completely absent in another. Although Walker and Cousins (1996) have warned of the potential for negative effects of TENS in CRPS patients, this should not deter therapists from using it if changes in application can be made so that progress in sensitivity control or pain management are achieved.

There are several references to the effects of electrostimulatory modalities on blood flow with a proposal of some effect on vasomotor activity (e.g. Low 1994). This has been suggested to be of potential benefit to patients with CRPS. A warning is that while TENS may alter vasomotor activity, the reasoning to support its use relies on an exclusively peripheral mechanism based model that assumes sympathetic nervous system dysfunction requiring correction.

Hardy and Hardy (1997) attempt a more objective approach to the use of TENS effects. They highlight its potential influence on temperature control (generally accepted as an indicator of vasomotor activity) and suggest that, with careful monitoring of temperature together with the patient's subjective

responses, it may be possible for clinicians to bias the TENS effect towards stimulation on blood flow if required.

It is important to recognise that successful use of TENS does not reliably indicate that any *specific* analgesic mechanism is operating. Within a patient we know that all forms and applications of TENS have widespread modes of action at many levels. For example, as in all therapies, there are likely to be effects determined via psychophysiologically activated pathways (see the Lawes chapter in Volume 4 of this series.)

Barbara Headley supported the use of TENS for CRPS patients back in the late 1980s (Headly 1987). She described how the exact mode of action of TENS in general and more specifically in CRPS was unknown. She intimated that a better understanding of the mechanism of action of TENS would result in a more rational decision over the settings used and the overall treatment protocol. The evidence available to date suggests that this has not yet occurred!

Taylor-Mullins (1989) stated that TENS (high frequency) would not by itself alleviate all the pain associated with CRPS but proposed that it may produce enough relief to facilitate other interventions, a theme supported by others (Headley 1987, Hardy and Hardy 1997, Thomas 1996, Stanton Hicks 1998).

Fredorczyk (1997) reported that there was a paucity of studies to support the use of TENS in painful conditions of the upper extremity.

Withrington and Wynn Parry (1984) offer a useful observation on TENS: 'This is an extremely valuable, but much abused modality of treatment. Far too often pain clinics hand the patient a stimulator with only the briefest instructions of how to use it, and not surprisingly the treatment proves ineffective.' They suggest that before discarding the modality several parameters including pulse width, repetition and pattern, electrode placement and duration of use should be experimented with.

An important aspect to the use of TENS is that whilst it is seemingly a passive therapy, it can be viewed as an active therapy in that it involves patients in their own management and as such offers a means of their having at least some control. This effect may be intensified if the individual understands its principles and is allowed to be actively engaged in a trial and error approach to find the most effective settings and mode of application.

Although Hareau (1996) strongly advocates the use of TENS in the management of pain in patients with CPRS, she suggests that it should be discontinued if it appears that the patient is becoming dependent on the modality. Unfortunately she fails to offer any help in how to identify potential 'TENS junkies' or a pathway of management to help patients reduce their dependency.

Interferential therapy

Some common texts claim that the autonomic nervous system can be manipulated using this modality (Low & Reed 1994, Savage 1984). It is interesting to note that the more scientific the text, the less likely it is that

such claims are found (Martin 1996). There is no convincing evidence in the literature searched that this therapy can alter either sympathetic or parasympathetic activity. However, it is worth reasoning that the way in which an individual reacts to the treatment stimulus may have a bearing on their autonomic tone.

Ultrasound

In discussing the use of ultrasound in the management of CRPS most authors refer to a paper by Portwood et al (1987). The paper offers three case studies to support tenuous claims of positive outcomes noted in their patients. There are no controls and all three patients received other interventions, too.

Harden (2000) stated that ultrasound was less effective in his clinic compared with the results of Portwood et al (1987). Unfortunately Harden's work has some methodological flaws; for example he fails to give the number of subjects from which he obtained his data.

At a mechanistic and purely peripheral level of reasoning, the use of ultrasound can be questioned since its effects are reported to be 'pro' inflammatory. As CRPS is thought to involve exaggeration/recruitment of the inflammatory and/or immune responses (see Chapters 2 & 3) this type of effect may be provocative.

Biofeedback

Grunert et al (1990) reported positive results when using thermal biofeedback (combined with relaxation and psychotherapy) in a group of individuals with residual RSD (CRPS Type I). They studied a group of 20 patients who had histories of repeated poor responses to other interventions. They used both visual and audible biofeedback to teach patients how to control their peripheral blood flow. They allowed patients to use the device during activities of daily living and relaxation. Their outcomes showed that the patients were able to learn how to control their blood flow as well as demonstrate a significant reduction in pain which was still present at a follow up 12 months later.

This study is limited by the usual criticisms of no control group and multiple interventions. However, the total package of care does demonstrate the desirable effects which occur *when patients are given some form of control and involvement in their own management*. This in itself could account for the alterations in blood flow noted as there would be a change in the SNS due to alterations in psychological driving mechanisms.

Hardy and Hardy (1997) advocated the use of thermal biofeedback although the only source they quoted to support the inclusion of this modality into their therapeutic regimen was the Grunert et al (1990) paper.

Earlier Headley (1987) discussed biofeedback as a potential intervention in the management of RSD (CRPS Type I). She cited the work from two papers (Blanchard 1979, Fischer-Williams et al 1981) that reported positive preliminary results in patients with RSD. What is interesting to observe is

that the case studies presented in these papers show a degree of commonality with the work of Grunert et al (1990) in that the responders had proved refractory to other therapies. Apart from the important 'control' aspect mentioned, it may be that newly introduced therapies, enthusiastically offered, have a 'novelty factor' effect that in some way kick starts positive biological responses. Here again, psychologically initiated and recruited mechanisms may underpin the positive changes observed.

Muscle stimulation

There are some advocates of the use of electrical stimulation of muscles in patients with CRPS Type I (Hareau 1996, Thomas 1996). As with other modalities there is no good trial evidence offered to support the use of these interventions. The suggested aims for such interventions include increasing blood flow, mobilising stiff joints, and increasing the strength of the musculotendinous apparatus (Hareau 1996, Thomas 1996).

Taylor-Mullins (1989) proposed the use of electrical muscle stimulation combined with compression bandaging to reduce the oedema associated with CRPS.

Iontophoresis

The transdermal application of guanethidine was first suggested by Hannington-Kiff in the mid-1980s. The treatment application followed observed successes with the drug when injected to produce a regional blockade (Hannington-Kiff 1984, see Chapter 4). The delivery of a drug using iontophoresis is dependent on its charge when in solution. Fortunately guanethidine has a high positive charge and therefore is relatively easy to deliver in this way.

Hannington-Kiff (1984) like most others, used a few non-controlled case studies to support the positive role of this blocking procedure. He reported that the individuals receiving the therapy gained immediate pain relief from the intervention. This observation raises interest since guanethidine blocks normally evoke an immediate *increase* in sympathetically maintained pain due to the massive initial noradrenaline release that it produces in the tissues (see Chapter 4.) It is of course possible that the individuals treated lacked adrenosensitivity in the tissues that the drug reached, or that transdermal application of the drug has a different mode of action. Sound reasoning for the effect should always include non-specific therapeutic effects (see placebo chapters in Volume 4) as well as the possibility that the current generated to produce the iontophoresis may have had a role to play too!

Heating

Warming the tissues has been advocated in the management of CRPS both to desensitise the tissues and to induce some pain relief (Charlton 1991,

Hareau 1996, Lampen-Smith 1997, Fredorczyk 1997, Stanton Hicks et al 1998, Harden 2000). The majority of these authors do not recommend any particular method of producing therapeutic heating. Whilst there is little 'scientific' evidence to support the use of heat, as we all know, many patients report that it is effective in providing a temporary reduction in pain.

Acupuncture

Ernst et al's (1995) paper is one of the few reports of this modality in the management of CPRS Type I. They demonstrated a reduction in pain, measured using a visual analogue scale, between a group receiving traditional Chinese acupuncture and a group receiving a sham procedure. Unfortunately the title of this article is somewhat deceptive as it leads one to think that they conducted a large scale randomised control trial (RCT—see Chapters 9 & 10). In fact they investigated only 14 patients and failed to use any statistical tests to analyse their results. The sham procedure, far from being physiologically inactive, involved the needling of the tissue at 'non acupuncture' points. It is important for clinicians to be aware that a great many studies have shown equal therapeutic effects whether needling is performed on recognised acupuncture points or not (see Melzack 1994).

Scrutiny of the results of Ernst et al (1995) also revealed a large degree of variance at each data point (indicted by large overlapping error bars on the graphs). Results like these, that lack a proper statistical analysis, should leave the reader with serious doubts about the validity of the claim that traditional acupuncture is a globally effective modality in the relief of pain in CRPS Type I.

There are a couple of dated studies (Chan & Chow 1981, Leo 1983) that have attempted to ascertain the benefit of electro-acupuncture in the management of this condition, but they have serious methodological flaws and are therefore of dubious value in terms of clarifying the situation. (See also Lesley Smith's analysis in Chapter 10.)

Splinting

Splinting was widely advocated in the older literature on CRPS Type I (Headley 1987, Taylor-Mullins 1989). Unsurprisingly, there has been little evidence to support its use. Even so, this modality still has some protagonists. For example, Thomas (1996) advocates the use of splinting (both resting and functional) in the management of those with upper limb CRPS Type I. His support is based solely on his own clinical observations. Most specialist hand and plastic surgery departments still suggest that splinting may be useful if used in conjunction with an active rehabilitation program (personal communication J. Leathwood, London.)

A review of the discussion on immobility in Chapter 2 highlights the potential harm that may be caused by injudicious splinting and casting that reinforces the patients' unwillingness to move.

Desensitisation

Most authors include some sort of desensitisation program as part of overall patient management (Headley 1987, Hareau 1996, Fitzpatrick 2000). Hardy and Hardy (1997) give sensible advice on desensitisation of hypersensitivities relating to vibration, touch, heat and cold . This is one approach that has some evidence to support its use and the reader is referred to consult their work as a resource (see also Chapter 6.)

Harden (2000) supports the use of desensitisation strategies and suggests that as well as specific techniques like those above, general exercise itself may act as a desensitiser of the person as a whole, perhaps reducing the maladaptive high 'gain' of their nervous system.

Movement and CRPS

The literature suggesting that movement is an essential component in the management of CRPS would pretty much include the whole of the reference list cited at the end of this chapter. This overwhelming support is based almost exclusively on clinical observation or relates to the simple fact that these patients are so often not moving and need to in order to have some sort of life.

Although there are no RCTs that look specifically at the use of movement based therapies in the management of this condition, there remains a firm consensus of opinion that movement based therapies should be *aggressive or vigorous*. Thankfully, Thomas (1996) rightly points out that such an approach may lead to a breakdown in patient confidence and reluctance on the part of patients to participate fully in their therapy. Overcoming the common chronic pain scenario, whereby the patient lurches from periods of overactivity and flare up to prolonged underactivity/rest, by careful planning, pacing and graded increases in activity are now well established and should be taken on here (see other management chapters in these volumes; also, Main & Spanswick 2000).

The movement based therapies that have been advocated range from passive mobilisations right through to self directed techniques aimed at compressing the joint surfaces. A frequent suggestion is for progress to move from passive through to active movement being often linked to the determined staging of the disorder. There are however more structured approaches to therapy such as the 'Scrub and Carry' regimen of Carlson and Watson (1988) (see below.)

In a recent highly recommended article, Stanton Hicks et al (1998) proposed guidelines for CRPS management that included various movement based strategies. Whilst unsubstantiated by any large clinical trial the progressive multilevel and multidisciplinary management approach is one which is now universally accepted for any ongoing pain state. A key focus of the approach is that of functional restoration. Here, all interventions, as far as possible, work together and are geared to promoting the achievement of time dependent goals.

The term kinesiaphobic has been used (Boas 1999; Vlaeyen & Linton 2000) to describe the observation that most patients with severe and long standing pain disorders have a dislike of movement if not an intense fear of it (see the section on fear avoidance, Volume 1 of this series.) Therapists are advised to respect such feelings. Patient fear of movement may be based on the experience of the provocation of intense pain or the belief that they may experience pain with a given movement. They may also fear further damage. The use of overtly aggressive or rigorous techniques and approaches is likely not only to be highly unproductive but also to promote further fear and anxiety of movement and result in increased disability (Vlaeyen & Linton 2000).

For this review, movement techniques have been split into the following categories: passive mobilisations, massage, and active exercises. Functional restoration and progressive loading are also discussed at the end of this chapter.

Passive mobilisations

All types of passive movements have been advocated to maintain/increase range of movement in CRPS patients. Hardy and Hardy (1997) advocated the use of continuous passive movement with the suggestion that it would allow pain free movement when hands-on approaches increase pain. This is a commonly recommended but un-researched modality (Taylor-Mullins 1989, Hareau 1996, Harden 2000).

A mechanistic and mechanism-based manual therapy reasoning approach has led to the application of 'targeted' passive mobilisation techniques for the management of 'sympathetic components' of disorders like CRPS. The techniques include, cervical side gliding, sympathetic slumping, and thoracic mobilisation.

Through the 1990s Wright, Slater and Vincenzino have been responsible for much of the work and interest in the effect of passive manual therapies on peripheral SNS activity (Slater 1995; Slater et al 1993,1994; Vincenzino et al 1994, 1998, 1999). Recently, Slater (2001) has pointed out that reproduction of symptoms by the sympathetic slump does not indicate that the SNS is involved in the condition, but more likely that the nervous system as a whole is sensitive to movement.

The work of Wright, Slater and Vincenzino noted above, suggests that there is a period of sympathetic excitation following the manoeuvers listed above and it is this finding that has led them to speculate on the mechanisms behind post manipulative pain relief. Others however have voiced caution and offered different interpretations and opinions (Zusman 1995, Thacker 1995). This work will be discussed in more detail in a later section.

The variable response of the SNS when measured following a high velocity thrust manipulation has been studied (Larson et al 1980, Kappler & Kelso 1984, Harris & Wagnon 1987). Both inhibitory and excitatory effects have been shown in normal subjects and those with symptoms/pathologies. Closer inspection of this work highlights several methodological flaws, most notably

the lack of suitable controls and the methods of monitoring changes in blood flow and skin conductance. It is interesting to note that the osteopathic and chiropractic professions seem to have moved away from using sympathetically mediated mechanisms to explain the effects of manipulation.

Clinicians need to be alert to the fact that touch alone has been shown to alter sympathetic activity (Appenzeller 1990). This suggests that there is an important need for an appropriate control group if the true effect of different manual interventions are to be fully described and understood.

As far as the SNS is concerned it may be well worth considering touch, manual therapy, mobilisations, or manipulations (or any therapy for that matter) on a spectrum of 'perceived threat' for a given patient. Thus, it would be predicted that a technique rated as a high 'threat' would strongly activate the SNS whilst those who perceive a technique more positively/of low threat, are likely to have only a modest sympathetic activation. Consider also that patients' perception of what is being done to them is likely to change from one moment to the next and therefore may be reflected in their sympathetic activity (Gifford 1998). If this is indeed the case, and the desire is to avoid a 'sympathetic' response, it supports the need for adequate preparation/advice/explanation of an intervention or technique before it is performed. On the other hand, and not recommended, if a high sympathetic response is deemed desirable, then it may be best to maintain an air of mystery, novelty, fear and the unexpected!

Massage

Although not strictly a movement based therapy, massage is often discussed with other manual therapies. Various massage techniques have been proposed as useful interventions in the management of CRPS Type I. Lampen-Smith (1997) has suggested that the main aim of massage interventions is to increase blood flow/venous return, encourage lymphatic drainage and to aid in the relief of pain. She offers a reasonable level of support for the use of massage in CRPS Type I patients. This is of interest when compared with the lack of references to support other interventions.

Frazer (1978) published a single case study demonstrating a positive response to connective tissue massage with a CRPS Type I patient. Unfortunately the presentation suffers from many problems associated with case studies of its era and so does not allow us to extrapolate.

One observation is that therapies such as massage may be, on the one hand, just as effective as other passive manual therapies, and on the other far less complicated and far easier and quicker to learn. Massage can also be easily done by the patient or spouses/relations and be seen as an active part of a graded, desensitising programme along side other management processes.

Comment on passive therapy approaches

The difficulties in decision making faced by hands-on passive management is highlighted when approaches to patients with upper limb CRPS by manual

therapists are compared to those advocated by specialist hand therapists. For 'peripheral' symptoms manual therapists might prioritise treatments that attend to spinal sources, or 'comparable spinal signs' found via physical assessment. Findings may in turn be linked to constructs of sympathetic involvement relating to issues like pathomechanics of the sympathetic chain or to 'facilitated segments' at appropriate levels. One hypothesis seems to be that therapy directed towards the spine and/or the sympathetic trunk may impact sympathetic activity in a beneficial direction (see discussion in Section 1). The hand therapist meanwhile concentrates efforts in the peripheral tissues, integrating mobilisation techniques of selected hypersenstive and/or hypomobile tissues along side progressive functional challenge.

Luckily, therapists have a certain freedom to operate in a trial and error fashion in order to bring about progress that is very often governed by the patient's pain response. What is evident is that constructs that relate the clinical presentation and the treatment approach to a particular mechanism are often very useful in justifying a novel approach. This is particularly so when a problem is well known for being difficult to help, combined with a form of therapy which might appear a little far fetched.

It is always worth considering what the patient might be thinking. Thus, justification is particularly important for the patient who could well find treatments of the spine, in sometimes very contorted positions, quite difficult to accept or to see as relevant. After all, it is hard to *believe* in something (an important requirement of the placebo response) if we do not understand it in the first place! Therapists using techniques whose focus of approach is on the spine for distal symptoms might be well advised to spend time explaining their underlying rationale (see placebo chapters in Volume 4).

Perhaps the most important point is to advise that therapists try to avoid getting carried away with the apparent power behind complicated manual therapy techniques and consider the technique as just one possibility, or more bluntly, a modest and questionable 'modality' in a management programme whose major aim is functional restoration and physical confidence.

The idea that pain relief has to be achieved before functional recovery is likely to be unproductive and unfulfilling. Time and again predominantly passive pain focused approaches are being criticised in chapters and essays, like those found in this and the other volumes in this series, as well as in the mature literature on chronic pain prevention and management. Readers are particularly alerted to the frequent statement that *passive therapy* with *high expectations* of pain relief/cure/fix, whether provided by hands, machines or tablets, may be making the patient become *reliant* on the therapy, pain fixated, fearful of movement and unnecessarily shifted towards a passive style of coping. Factors like these are seen as significant factors in causing rather than preventing chronic incapacity.

Active exercise

It makes logical sense to promote active movement and function in an individual who has limited function, limited ranges of movement and

significant fear of movement and its consequences (see Volume 1 of this series.) It is therefore unsurprising that the use of active movement as a part of management programs is now so widely advocated (Charlton 1991, Hareau 1996, Lampen-Smith 1997, Stanton Hicks 1998, Stanton Hicks et al 1998, Harding 1999, Fitzpatrick 2000). All these authors suggest the biggest obstacle to the restoration of movement is the presence and impact of pain.

Various exercises and concepts have been proposed to help restore active movement but perhaps the most widely advocated for CRPS is the 'Scrub and Carry' regimen of Watson and Carlson (Watson & Carlson 1987, Carlson & Watson 1988, Charlton 1991, Hardy & Hardy 1997, Stanton Hicks 1998). This involves a progressive pattern of axial loading (compression) and distraction. This program has received support as it is thought not only to encourage movement but also to help desensitise the affected extremity. The name comes from the original authors use of a scrubbing brush to facilitate vertical compression of the limb and the use of a brief case/bag to provide a load to provide axial distraction! In line with other desensitising and activating programmes they advocated a build up (graded exposure) of these forces over time. However, even though the regimen is now quite old there is as yet no good evidence for its widespread support.

Hareau (1996) advocates an exercise approach that works from proximal to distal which has obvious parallels with the management of neurological conditions. Hareau (1996) suggests that the approach minimises the chance of exacerbation of the patient's symptoms.

Discussion 1: Manual therapy, sympathetic effects and the sympathetic slump

It is the opinion here that we are at a cross-roads in the professional development of physiotherapy. On the one hand we desire better and more scientific evidence and explanations for our interventions, while on the other we are uncomfortable and uneasy with what we may find.

Mention was made earlier of the work that has focused on manual therapy induced SNS change and an extended discussion of this work is presented here.

This work, and its clinical interpretations, seems to have had a powerful, almost global, impact on manual therapy approaches to pain and its relation to conditions that traditionally contain 'sympathetically labelled' phenomena. Since closer scrutiny of the work reveals some potential challenges and flaws with the results and their interpretations, I (Thacker) feel it important and appropriate to outline them here. It is desirable that this discussion is seen not as unnecessary intellectual niggling but as part of the important process of mature scientific appraisal whose goal is ultimately the good of the patient and the standing of the physiotherapy profession.

Slater (1991) hypothesised that a modified version of the slump test could be used to investigate the response of the sympathetic trunk to physical

loading. Based on analysis of the anatomy and relations of the sympathetic trunk (see Chapter 1) in relation to spinal axes of movement and known nervous system dynamics, she proposed that a combination of movements could physically tension the ipsilateral sympathetic chain and ganglia. (To tension the right sympathetic chain the movements required are: in a long sitting position, add, thoracic spine flexion, side flexion and rotation to the left with cervical extension and left side flexion.)

Together with co-workers, Slater (Slater et al 1993, 1994) went on to investigate the effects of the 'sympathetic slump' combined with a rib mobilisation technique on a group of normal subjects. They were able to show that there was a slightly greater increase in skin conductance (due to increased sweating, see below), in the upper limbs in their experimental group, than in either a placebo condition or control group. They showed that although this effect was bilateral it was greater on the side of the loaded sympathetic chain. They also measured skin temperature and although the placebo and test results were significant versus the control [temperature was seen to decrease] they were not different from one another. They concluded that the sympathetic slump had excitatory physiological effects on the peripheral SNS function.

Further studies were performed using a neural tension test to load the upper limb nerve trunks and similar results were obtained. Again skin conductance was increased with no change in temperature versus placebo and control. Slater (1995) also published a study into the effects of the 'sympathetic slump' on a group of patients with frozen shoulder. Her results showed similar measurable effects to those of normal subjects quoted above, that is, an apparent sympathetic excitation. She concluded, with notable caution, that sympathetic slump alone was not likely to help in the treatment of patients with this condition.

Peterson et al (1993) showed that a grade III passive mobilisation technique applied to the C5 vertebra increased sympathetic activity in the upper limbs of normal subjects. They monitored both temperature (as a measure of vasomotor activity) and skin conductance (sudomotor function) and included a placebo condition in the trial. They suggested that these responses might underlie the analgesic effect of mobilisation techniques.

The results and conclusions of these investigations are open to alternative interpretations.

All of the above studies used direct measurement of skin conductance in the upper limb as a determinant of the sympathetic response to the physical testing procedures. (Skin conductance is a measure of sweat production/sudomotor function: increased moisture on the skin leads to a lower impedance of the skin.) They all reported significant increases in their experimental and placebo groups with increases also noted between these groups in a pattern in favour of the treatment groups.

One criticism is that they tested sudomotor function (skin conductance) in each case over the palmar aspect of the thumb and/or index fingers. While this seems straight forward, it is known that sudomotor activity of the sweat glands on the palmar aspect of the hand are controlled solely by the psycho-

emotional centres (personal experience of sweaty palms associated with nerves supports this!) and though reflexogenically mediated via the sympathetic efferents to the palm, may really represent a measure of the psycho-emotional drive (Appenzeller 1990, Uncini et al 1988, Scerbo et al 1992, Kunimoto et al 1992). As argued by Gifford (Gifford 1998, p. 84), the individual undergoing a manual therapy procedure may well be processing the event as a relative 'threat', feel a bit uncertain or uncomfortable, and hence mediate an increase in SNS activation.

Thus an additional proposal for these results is that they may well reflect the variable but possibly powerful psychological impact of the manoeuver in addition to the tissue, segmental and brain stem feedback and feedforward mechanisms that the authors suggest. The point is that the activity of the dedicated brain stem sympathetic compartments and those relating to nociceptor modulation are powerfully influenced by higher centres. The way we perceive the situation we are in and the sensory information we 'receive' is a primary driver of these systems' activity.

In the studies cited that investigate the effects of various techniques on the SNS activity, what needs to be appreciated is the physical differences between the 'treatment' condition, the 'placebo' condition, and the 'control' conditions. The treatment condition involves the well controlled application of the technique under investigation; the 'placebo' condition involves the exact same physical interaction as the treatment condition (physical touch, as well as interaction and communication) except that the actual movement of the technique is not done. In other words, the therapist applies their hands to the patient for the same amount of time as the technique takes, but does not push and let go to produce the treatment oscillation effect. The 'control' condition involves putting the patient in the same position as the treatment condition and the placebo condition, but no hands are applied (for example, see Vincenzino et al 1996). The difficulties of producing a 'placebo' and 'control' group for this type of research are acknowledged. However, to an observer with a bias towards a psychological interpretation, what appears to be being analysed is the psychological impact of three very novel and odd situations. The psychological impact is being reflected in the activity and outward response of the SNS. One would imagine that all three conditions are open to quite varied individual responses that would be determined by such things as feeling awkward, feeling psychologically or physically uncomfortable, or feelings of threat or pleasure. Thus one person might feel far more uncomfortable being left alone in the long sit slump (sympathetic slump) position for the control condition than they might in the placebo condition where they are being held. A quite opposite reaction might occur in another individual who perhaps is not comfortable with the person who is touching and moving them. Different reactions might also be expected relating to the subjects previous experiences, too. For example, if some of the subjects were physiotherapists they might feel comfortable with being physically undressed and handled, whereas others might find the situation distressing.

Another consideration is that in all of the slump investigations the contact with the trunk by the operator was on the opposite side of the body to the sympathetic chain being 'tensioned' and focused on. It is known that manual contact on one side of the body tends to increase the sudomotor activity on the contralateral side and inhibit it on the ipsilateral side in normal subjects (Appenzeller 1990). This offers a further level of complexity to interpreting the results of these procedures.

Caution is indicated when accepting results from studies that use skin conductance as a measure of sympathetic activity as they are known to be fraught with technical difficulties and inaccuracies. For example, the responses are known to habituate as well as being influenced by many factors that are independent of the SNS (Uncini et al 1988, Scerbo et al 1992, Kunimoto et al 1992, Janig 1993, Mathias & Bannister 1993). Mathias and Bannister (1993) have even gone so far as to suggest that for these reasons it is unsuitable to use these measures in clinical trials.

The other measurement recorded throughout the experiments was temperature change, used by the researchers as an indicator of alterations in vasomotor activity. Variable responses were noted in all the studies under scrutiny, with only one showing significant reductions indicating vasoconstriction (see Chiu & Wright 1996, Peterson et al 1993). The results seem to be at variance with the overview of the work provided by Vincenzino and Wright (2002). Here, the authors claim that substantial increases in cutaneous vasomotor activity occur. There appears to be a degree of ambiguity with the interpretation of the results and readers are urged to consult the individual studies cited.

Even so, the validity of this measurement has been questioned. For example, Janig (1993) stated that in experimental conditions, 'the skin temperature is not necessarily correlated with the activity in sympathetic vasoconstrictor neurons. This most probably also applies to equivalent clinical situations but is generally ignored.' He concludes with the important clinical caveat that, 'it is not correct to conclude on the basis of the cold feet or hands of patients that there is a high sympathetic tone or hypersympathetic activity.'

It is important to note that in all the studies the temperature monitoring probe was placed on the dorsum of the hand, a position which is likely to produce confounding results due to the well established fact that the dorsum of the hand has both an active vasodilation and vasoconstriction system under the direct control of the SNS (Bell 1983, Lindblad et al 1990, Johnson 1995). Simply swapping the recording probes around so that conductance was measured posteriorly and temperature anteriorly may have improved the validity of the tests but would still be fraught with confounding variables. Slater (2001) recently indicated caution when making interpretations about sympathetic functioning from these or similar studies.

Vicenzino and Wright (see Vincenzino et al 1998 & 1999) have investigated and proposed a hypothesis to explain the pain relieving effects of mobilisation techniques used in manual therapy. They have used data from their many studies to suggest that the apparent sympathetic excitation noted may be

linked with an increase in activity of the dorsal portion of the periaqueductal grey matter in the midbrain with consequent descending inhibition of the spinal cord. They suggest that this involves the non-opioid predominantly noradrenergic lateral column pathways (reviewed by Wright 2002). Whilst a feasible and important suggestion it remains an untested theory and in view of the above criticisms may be based in part on work over which there has to remain a few questions. What is certain is that any single modality is unlikely to exert its analgesic effects via a single line labeled specific pathway such as this (personal communication, P. D. Wall , London, 2001.) What has begun is excellent, but clearly more work is needed to further clarify the situation.

Discussion 2: Models of care—plea for an ordered approach

The most striking observation from this review of the literature is the lack of depth and breadth. There is a lot of work that needs to be done and the hole left by the lack of controlled clinical trials is significantly large to leave little in the way of informed guidance.

CRPS, as its name suggests, is indeed complex and multifactorial. Little wonder that most of us feel a sense of dread and even inadequacy when faced by patients with such extreme sensitivity and disability. Pleasingly, the CRPS Guidelines for Therapy, Consensus Report written by Stanton-Hicks and colleagues (1998) offers a way forward and we re-iterate an earlier suggestion that it is essential reading (see Fig. 5.1.) There are several key points worth highlighting:

- The report summarises current thinking and the consensus position of the International Association for the Study of Pain. It offers clear diagnostic criteria and explains many of the common symptoms of the condition as well as therapy guidance.
- The report provides a multidisciplinary algorithm for management with a central physical therapy based core whose primary goal is reactivation and return of function (see Chapters 6 & 7.)
- Supported on either side of this central core of physical therapy/ reactivation is a requirement for appropriately timed 'medical' and 'psychological' interventions.
- A stepped approach to management that is time dependent is viewed as essential.

The medical interventions listed include the use of:

- *Medications* early on i.e. NSAIDS, opioids, tricyclic antidepressants, alpha-2 agonists, sodium ion channel blocking agents. If these prove inadequate or there is no response the algorithm moves on to:
- '*Blocking procedures*' (+/- medications), listed as: focal, sympathetic, regional, epidural, and pumps. The last tier of intervention then shifts to:

- *'Neurostimulation'* (+/- medications), being either peripheral or epidural.

The supporting psychological interventions proposed include: *Counselling* that deals with expectations, motivation, control, family and 'diary'; *Behaviour*; *Relaxation*; *Imagery*; *Hypnosis;* and *Coping Skills*.

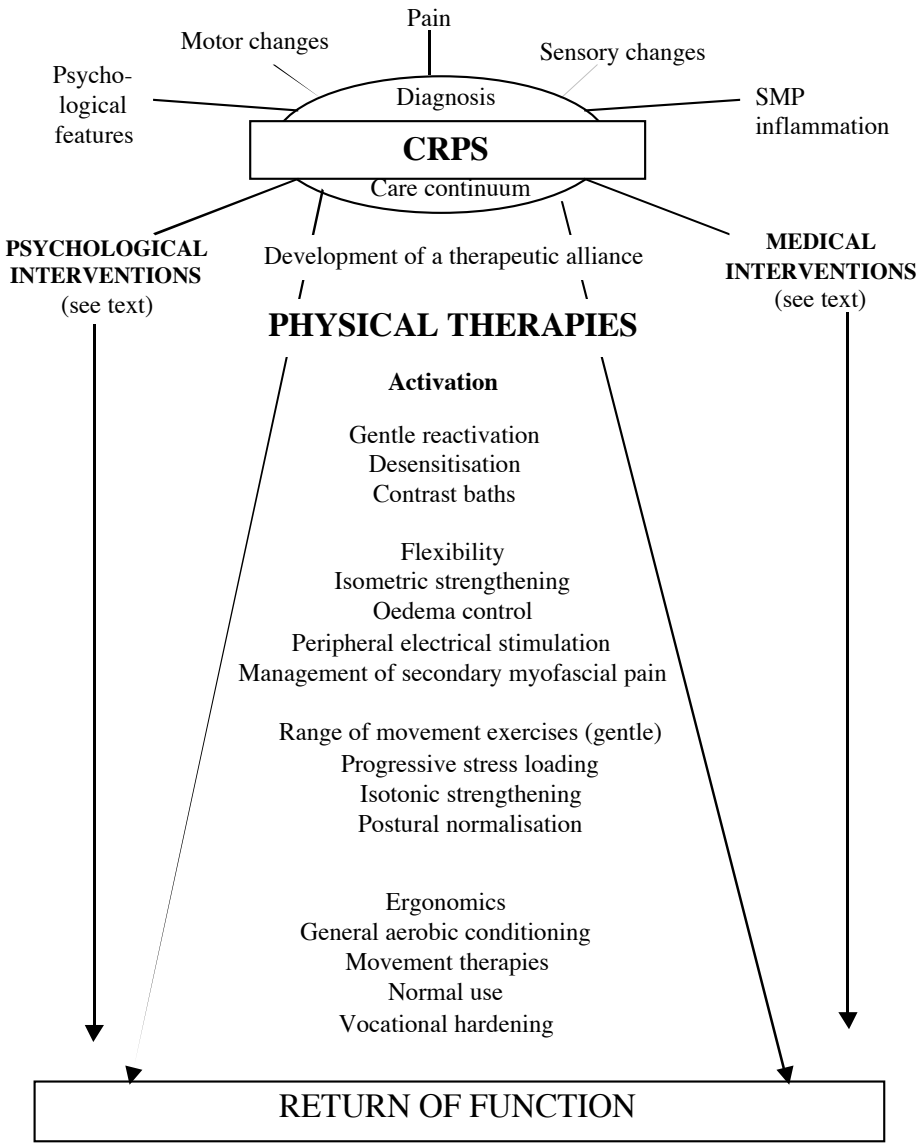

Fig. 5.1 A slightly modified version of the algorithm of management suggested by Stanton-Hicks et al 1998.

Adapted from: Stanton-Hicks et al 1998 Complex regional pain syndromes: guidelines for therapy. The Clinical Journal of Pain 14:155–166.

The authors suggest that 'failure' to achieve improvement after three weeks of the commencement of each stage (see Fig. 5.1) indicates the need for involvement of these other specialties in the management process. Importantly, they point out that patients require maximum support throughout and that any person refractory to therapy should not feel as though they have failed.

Interestingly, they indicate the first and most important stage of the activation process is the development of a good rapport between the patient and the therapist and the development of a 'therapeutic alliance.' Once achieved, the next step is to move onto the desensitisation, mobilisation and motivation phase (see Chapters 6 & 7.) They state 'it is essential that movement phobia be overcome and the patient begin to actually move and allow the limbs to be touched.'

The report suggests that concomitant conditions that continue to 'input' nociceptive information into the CNS such as myofascial problems, may require appropriate attention.

The next phase involves strengthening, stress loading and general aerobic conditioning to support the general and continued reactivation achieved.

The statement that 'It is particularly important to avoid aggressive or passive range of motion (ROM) tests, especially in an extremity that is insensate after regional anaesthetic blockade' is noteworthy since it is in stark contrast to traditional approaches that promote 'aggressive' and insensitive attitudes to this condition. It still seems common for therapists, clinicians and anaesthetists to be under the impression that therapy is best started as soon as someone has received a nerve block. This may be inappropriate practice and is certainly not based on any credible evidence available. It can be argued that the benefits of movement on this condition involve and require normal proprioceptive feedback to the nervous system. This would be impossible during or immediately after the blocking procedure, particularly where there has been a tourniquet applied since ischaemia, produced by a tourniquet, primarily blocks the activity of large myelinated nerve fibres (Thacker 1996). Other dangers that might apply to this approach involve the exercising of dysfunctional tissues in potentially ischaemic conditions. This is known to have a deleterious effect on soft tissue healing (Tim Watson, personal communication, London 2001).

The final step of the treatment algorithm aims at full functional restoration. This stage may include vocational rehabilitation, functional capacity measurement and other interventions geared at normal use of the affected limb. The report emphasises the need to include the role of employee education to allow early, albeit modified, return to work. As already outlined there is also a focus of attention on the use of psychological management and the role of cognitive behavioural management strategies.

The authors state clearly that the biggest obstacle to patient progression is pain and subsequent fear avoidance (see Part 2 in Volume 1 of this series.) They make a clear statement that unacceptable pain requires effective treatment and this is a key indicator for the need for pharmocotherapy,

regional blocks, neurostimulation etc. Thus, while physical function is the driver for the algorithm, interventions beyond those outlined in Figure 5.1 are only implemented to allow continued progression. The report is important and unique in that it sets out a broad based multidisciplinary rationale for appropriate treatment selection and progression.

The report usefully outlines in moderate detail the pharmacological and invasive procedures listed above. It also gives some evidence for and against their use and their known efficacy. Mechanisms of action and dosage are briefly outlined and a 'quick guide' is provided to this massive topic. Special attention is given to the topic of CRPS in children, not only with respect to direct management but also to the support and involvement of parents. Specific references are included offering a good first stop for the interested reader.

The recommendations of this report are as yet unproven. However, it is an exciting piece of work for physiotherapists whose input forms the central core of the reactivation process. It is also a rational, well structured and staged approach. In so doing, it takes physiotherapy away from the haphazard trial and error approaches discussed earlier and for which we seem best known. An excellent start for physiotherapy would be to implement the approach and, at some stage, audit and publish outcomes. If proven to be of value it would serve to challenge trends towards unstructured multi-interventions. Here of course, outcomes are impossible to ascribe accurately to one aspect, one intervention or one modality that might be involved in the care. The advice here is to not be charmed by those who advocate easily achieved outcomes using single modalities as core components of their approaches (see Muramatsu et al 1998, as a typical example). Rather, see CRPS, defined by its very nature, as a complex and multifactorial problem that requires a multifactorial, open minded and compassionate approach.

Conclusion

Three main issues arise with CRPS:

1. Proof of the efficacy of physiotherapy modalities and skills in treatment and management is clearly lacking.
2. Until relatively recently, there has not been a clear framework on which to base any intervention. Whether a particular modality works, whatever the discipline, may be largely down to hope, faith, and luck. Patients with CRPS and many patients with other chronic pains, if medicine is honest, are dumped onto physiotherapy. We are the option that takes a bit of time up and moves the difficult patient out of the consulting room hopefully never to return. The proposals of Stanton-Hicks et al (1998) that were reviewed above provide a constructive, multidisciplinary and multidimensional approach that is bathed in strong mutual cross-disciplinary respect. The hope is that all those involved will be interested in playing a part in patient outcomes and as a result work together for

this common goal. The guidelines are clear, graded, and have long term objectives that seem sound and exciting and are a logical option to adopt. What is pleasing is that the right sort of physiotherapy provides the core component of therapy.

3. If the approach proposed by Stanton-Hicks et al (1998) is agreed and adopted it means that mechanistic, tissue targeted and modality dominated therapies, that are the mainstay of most current physiotherapy treatments, and still form a dominant part of training, will have to take a significant back seat in the future management of patients with conditions like CRPS. There are a great many new skills to learn, but most challenging of all is the appreciation and application of important new models of care (see Chapter 8 this volume and Muncey chapters in Volumes 4 and 2).

REFERENCES

Appenzeller O 1990 The Autonomic Nervous System. An Introduction to the Basic and Clinical Concepts, 4th edn. Elsevier, Amsterdam

Bell C 1983 Vasodilator neurons supplying skin and skeletal muscle of the limbs. Journal of the Autonomic Nervous System 7:257–262

Boas R 1999 Workshop Presentation, 9th World Congress on Pain. International Association for the Study of Pain. Austria

Carlson LR, Watson HK 1988 Treatment of reflex sympathetic dystrophy using the stress loading program. Journal of Hand Therapy 1:149–154

Chan CS, Chow SP 1981 Electroacupuncture in the treatment of post-traumatic dystrophy (Sudeck's atrophy). British Journal of Anaesthesia 53:899–902

Charlton JE 1991 Managagement of sympathetic pain. In: Wells JCD, Woolf CJ (eds) Pain Mechanisms and Management. British Medical Bulletin 47 (3):601–618

Chiu TW, Wright A 1996 To compare the effects of different rates of application of a cervical mobilisation technique on sympathetic outflow to the upper limb in normal subjects. Manual Therapy 1:4:198–203

Ernst E, Resch L, Fialka V, Ritter-Dittrich D, Alcamioglu Y, Chen O, Leitha T, Kluger R 1995 Traditional acupuncture for reflex sympathetic dystrophy: A randomised sham-controlled, double blind trial. Acupuncture in Medicine 13(2):78–81

Fitzpatrick AF 2000 Clinical Practice Guidelines. Reflex sympathetic dystroph/complex regional pain syndrome. Reflex Sympathetic Dystrophy Association of America

Frazer FW 1978 Persistent post sympathetic pain treated by connective tissue massage. Physiotherapy 64(7):211–212

Fredorczyk J 1997 The role of physical agents in modulating pain. Journal of Hand Therapy 10:110–121

Gifford LS 1998 Output mechanisms. In: Gifford LS (ed) Topical Issues in Pain 1. Whiplash—science and management. Fear-avoidance beliefs and behaviour. CNS Press, Falmouth 81–91

Grunert BK, Devine CA, Sanger JR, Matloub HS, Green D 1990 Thermal self-regulation for pain control in reflex sympathetic dystrophy syndrome. Journal of Hand Surgery 15A (4):615–618

Hannington-Kiff JG 1984 Pharmacological target blocks in hand surgery and rehabilitation. Journal of Hand Surgery 9B(1):29–36

Harden RN 2000 A clinical approach in complex regional pain syndrome. Clinical Journal of Pain 16:S26–S32

Harding V 1999 The role of movement in acute pain. In: Max M (ed) Pain—an updated review. Refresher course syllabus. IASP Press, Seattle 159–169

Hardy MA, Hardy SGP 1997 Reflex sympathetic dystrophy: The clinicians perspective. Journal of Hand Therapy 10:137–150

Hareau J 1996 What makes treatment for reflex sympathetic dystrophy successful? Journal of Hand Therapy 9:367–370

Harris W, Wagnon RJ 1987 The effects of chiropractic adjustments on distal skin temperature. Journal of Manipulative and Physiological Therapeutics 12 (2):57–60

Headley B 1987 Historical perspective of causalgia. Management of sympathetically maintained pain. Physical Therapy 67 (9):1370–1374

Hooshmand H 1993 Chronic pain: Reflex sympathetic dystrophy prevention and management. CRC Press. Boca Raton

Janig W 1993 Pain and the autonomic nervous system: Pathophysiological mechanisms. In: Bannister R, Mathias C J (eds) Autonomic Failure, 3rd edn. Oxford University Press, Oxford

Johnson JM, Pergola PE, Liao FK, Kellog DL, Crandall CG 1995 Skin of the dorsal aspect of the human hands and fingers possess an active vasodilator system. American Journal of Physiology 267:948–954

Kappler RE, Kelso AF 1984 Thermographic studies of skin temperature in patients receiving osteopathic manipulation treatment for peripheral nerve problems. Journal of the American Osteopathic Association 84 (1):126–127

Kunimoto M, Kirno K, Karlson T, Wallin BG 1992 Non linearity of skin resistance to intraneural electrical stimulation of sudomotor nerves. Acta Physiologie Scandinavia 146 (3):385–392

Lampen-Smith RA 1997 Complex regional pain syndrome I (RSD) and the physiotherapeutic intervention. New Zealand Journal of Physiotherapy 4:19–23

Larson NL, Watson MW, Kelso AF 1980 Effectiveness of manipulative treatments for parasthesias with peripheral nerve involvement. Journal of the American Osteopathic Association 80 (3):114(Abs)

Leo KC 1983 Use of electrical stimulation at acupuncture points for the treatment of reflex sympathetic dystrophy in a child. Physical Therapy 63:957–959

Lindblad LE, Ekenvall L, Klingstedt C 1990 Neural regulation of vascular tone and cold induced vasoconstriction in the human finger skin. Journal of the Autonomic Nervous System 30:169–174

Low A, Reed A 1994 Electrotherapy Explained: Principles and practice 2nd edn. Butterworth-Hienemann, Oxford

Low J 1994 Electrotherapeutic modalities. In: Wells PE, Frampton V, Bowsher D (eds) Pain Management by Physiotherapy. Butterworth Heinemann, Oxford 140–176

Main CJ, Spanswick CC 2000 Pain Management. An interdisciplinary approach. Churchill Livingstone, Edinburgh

Martin D (1996) Interferential therapy. In: Kitchen S, Bazin S (eds) Clayton's Electrotherapy 10th edn. Saunders, London

Mathias CJ, Bannister R 1993 Investigation of autonomic disorders. In : Bannister R, Mathias CJ (eds) Autonomic Failure 3rd edn. Oxford University Press, Oxford

Melzack R 1994 Folk medicine and the sensory modulation of pain. In: Wall PD, Melzack R (eds) Textbook of Pain 3rd edn. Churchill Livingstone, Edinburgh

Muramatsu K, Kawai S, Akino T, Sunago K, Doi K 1998 Treatment of chronic regional pain syndrome using manipulation therapy. Regional Anaesthesia 44(1):189–192

Peterson N, Vincenzino W, Wright A 1993 The effects of a cervical mobilisation technique on sympathetic outflow to the upper limb in normal subjects. Physiotherapy Theory and Practice 9:149–156

Portwood MM, Lieberman JS, Taylor R 1987 Ultrasound treatment of reflex sympathetic dystrophy. Archives of Physical Medicine and Rehabilitation 68:116–118

Savage B 1984 Interferential Therapy. Faber and Faber, London

Scerbo AS, Freedman LW, Raine A, Dawson ME, Venables PH 1992 A major effect of recording site on measurement of electrodermal activity. Psychophysiology 29(2):241–246

Slater H 1991 Adverse neural tension in the sympathetic trunk and sympathetic maintained pain syndromes. In. Proceedings of the Seventh Biennial Conference of the Manipulative Physiotherapist Association of Australia

Slater H 1995 An investigation of the physiological effects of the sympathetic slump on peripheral sympathetic nervous system function in patients with frozen shoulder. In: Shacklock M (ed) Moving in on pain. Butterworth Heinemann, Sydney

Slater H 2001 Vegetatives nervensystem In: Van den Berg F (ed) Angewandte Physiologie: 3 Therapie, Training, Tests. Georg Thieme Verlag, Stuttgart

Slater H, Vincenzino G, Wright A 1994 'Sympathetic slump': The effects of a novel manual therapy technique on peripheral sympathetic nervous system dysfunction. The Journal of Manual and Manipulative Therapy 2 (4):156–162

Slater H, Wright A, Vincenzino G 1993 Physiological effects of the sympathetic slump on peripheral sympathetic nervous system function. In: Integrating Approaches. Proceedings of the Eighth Biennial Conference of the Manipulative Physiotherapist Association of Australia

Stanton-Hicks M 1998 Management of patients with complex regional pain syndromes. PSNS SIG Newsletter. IASP Press, Seattle

Stanton-Hicks M, Baron R, Boas R, Gordh T, Harden RN, Hendler N, Koltzenberg M, Raj P, Wilder R 1998 Complex regional pain syndrome: Guidelines for therapy. Clinical Journal of Pain 14:155–166

Stanton-Hicks M, Janig W, Hassenbusch S, Haddox J D, Boas R, Wilson P 1995 Reflex sympathetic dystrophy: Changing concepts and taxonomy. Pain 63:127–133

Taylor-Mullins PA 1989 Management of common chronic pain problems of the hand. Physical Therapy. 69 (12):1050–1058

Thacker MA 1995 The effect of 'self sympathetic slumping' on peripheral sympathetic nervous system activity. MSc Thesis. University College London. London University

Thacker MA 1996 Early intervention in the management of complex regional pain syndrome I. Does it make a difference? Abstract, Pain Society of Great Britain Annual Scientific Meeting

Thomas D 1996 Physiotherapy and rehabilitation of upper extremity reflex sympathetic dystrophy. Clinical Orthopaedics 1 (2):339–360

Uncini A, Pulman SL, Lovelace RE, Gambi D 1988 The sympathetic skin response: Normal values, elucidation of afferent components and application limits. Journal of Neurological Science 87 (2-3):299–306

Vincenzino B, Collins D, Wright A 1996 The initial effects of a cervical spine manipulative physiotherapy treatment on the pain and dysfunction of lateral epicondylalgia. Pain 68: 69–74

Vincenzino G, Cartwright T, Collins DM, Wright A 1999 An investigation of stress and pain perception during manual therapy in asymptomatic subjects. European Pain Journal 3:13–18

Vincenzino G, Collins D, Wright A 1994 Sudomotor changes influenced by neural mobilisation techniques in asymptomatic subjects. The Journal of Manual and Manipulative Therapy 2(2):66–74

Vincenzino G, Collins DM, Benson HAE, Wright A 1998 The interrelationship between manipulation induced analgesia and sympathoexcitation. Journal of Manipulative and Physiological Therapeutics 7(12): 448–453

Vlaeyen JWS, Linton S J 2000 Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. Pain 85(3):317–332

Walker SM, Cousins MJ 1996 Complex regional pain syndromes: Including 'Reflex sympathetic dystrophy' and 'Causalgia'. Anaesthesia and Intensive Care 25:113–125

Watson HK, Carlson LR 1987 Treatment of RSD of the hand with an 'active stress-loading' program. Journal of Hand Surgery 12A:779–785

Withrington RH, Wynn Parry CB 1984 The management of painful peripheral nerve disorders. Journal of Hand Surgery. 9B (12):24–28

Wright A 2002 Neurophysiology of pain and pain modulation. In: Strong J, Unruh A, Wright A et al (eds) Pain: A textbook for therapists. Churchill Livingstone, Edinburgh 43–64

Zusman M 1995 Sympathetic responses to passive movement manoeuvres. Proceedings of the Ninth Biennial Conference of the Manipulative Physiotherapists Association of Australia