

CHRONIC HAND PAIN

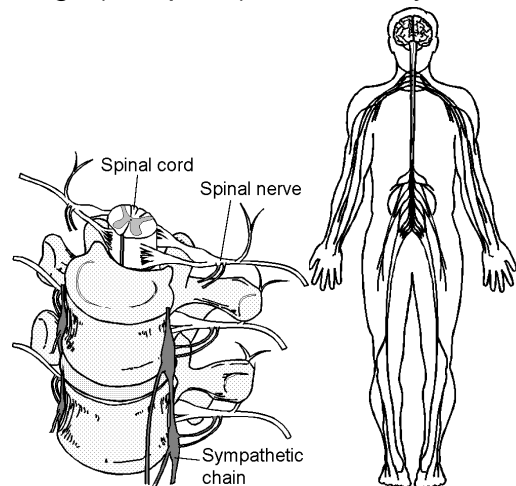
Introduction

Pain is transmitted to the brain from specific nerve endings (receptors) via a variety of nerve fibre (axon) types. The information is ultimately relayed up the spinal cord to the brain where the information is processed by specific centres.

There are two nerve systems operating within the body, both of which are involved with the appreciation and responses to pain.

Spinal nerves run to and from the spinal cord. They are for conscious functions such as touch and muscle movement

Sympathetic nerve fibres run down blood vessels and with the spinal nerves to and from the sympathetic chain and are concerned with subconscious automatic activities such as temperature regulation.

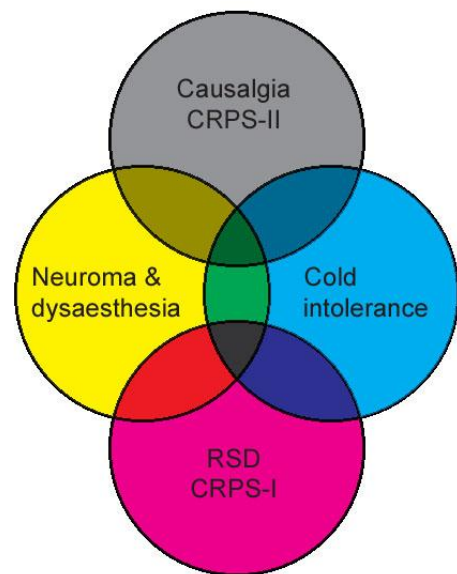


Pain

Chronic pain is a poorly understood problem. That originating in nerve has confusing names based on historical theories as to causation. These have been reclassified as “Chronic Regional Pain Syndrome” (CRPS).

The two principle types of pain, other than that caused by acute injury and compression, are **reflex sympathetic dystrophy** and **causalgia**. We also see patients who have **neuroma tenderness** and **dysaesthesiae**. These all can co-exist and all are often associated with **cold intolerance** which in its own right is a very common sequel to hand injuries.

Reflex sympathetic dystrophy (syn. algodystrophy, CRPS-I) is a poorly understood phenomenon, which has been thought to be due to a local disturbance of sympathetic nerve activity. Its occurrence is not specific to type or severity of injury and it is well recognised to occur after relatively trivial injuries. The early stages of this syndrome are characterised by pain, hyper-sensitivity to touch, stiffness and “dystrophic” signs such as vasomotor instability as indicated by swelling (oedema), colour, temperature change and altered sweating. The symptoms and signs extend beyond the area of injury or surgery. Other changes seen include increased hair growth and finger-nail ridging. The severity of the condition can vary considerably. Milder examples are self limiting and merely slows progress after injury or surgery. Severe forms are fortunately very rare but can cause significant dysfunction in the whole limb. The overall prognosis for these cases is often poor and recent studies show that many patients never fully recover, often experiencing permanent symptoms, which are often triggered either by exposure to extremes of temperature or by heavy activities. The chronic stage is characterised by chronic intractable pain, trophic changes and joint stiffness



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Causalgia (syn. CRPS-II) describes a burning-type pain that follows nerve injury. This comprises a background pain, which is exacerbated by touch, temperature and emotion. The problem is confined to the nerve distribution. There are no external signs as it is not associated with “dystrophic” changes (see RSD). It is related to the “phantom” sensation and pain experienced after amputation. Although initiated by nerve injury, the pain is centrally mediated, perhaps at the level of the spinal cord. Yet uncharacterised pathways or chemical changes probably occur at a synaptic level. Attempts to modulate it surgically at a peripheral nerve level are invariably unsuccessful.

Neuroma tenderness follows nerve injury and results from the effects of nerve regeneration. Axons grow from the cut end of a nerve and attempt to find the other end of the nerve. If the nerve was not repaired, the gap is usually too large for the fibres to find the other end. After amputation, where there is no nerve for the axons to grow towards. In these situations, the axons therefore collect as a tender swelling that is called a neuroma. In general, there is an inverse relationship between sensory recovery and the extent of neuroma formation. Patients are free of pain at rest and when not using the hand. However, if the neuroma area is tapped or knocked an “electric” type pain shoots into the original distribution of the nerve.

Dysaesthesia also follows a nerve injury where regeneration has been imperfect. Nerves contain many types of fibre which serve different purposes, both for movement (motor) and feeling (sensory). Recovery from injury will be associated with mismatching of fibre types between nerve ends. The result is some quantitative (loss of feeling) and qualitative (altered feeling) change in the area of nerve supply. Paradoxically although sensation is actually reduced, patients describe the area affected as “sensitive” because touch can be perceived as being “unpleasant”, “pins & needles” and smooth can feel rough.

Cold intolerance is not specific to type of injury, but it is very commonly observed after digital replantation and fingertip injuries. It is a poorly understood phenomenon, which has been defined as an exaggerated or abnormal reaction to cold exposure of the injured part causing discomfort or the avoidance of cold. There are four components to its presentation; pain/discomfort, stiffness, altered sensibility and colour change, which may appear in isolation or in any combination. The reported incidence after hand injuries varies between 50 and 100 per cent, but is probably in the region of 70-85%. It tends to develop over the first three months after injury and to remain fairly constant in the first year after injury. Some improvement is then observed over the subsequent two years, but it resolves in few patients. Its severity can vary widely and there is some suggestion that this is affected by the presence of a fracture and the type of injury. Its presence causes difficulty with working in cold or exposed conditions, unless the hands are kept well protected.

Treatment

The assessment and treatment of chronic hand pain and tenderness is difficult. Diagnosis is very dependant on the history of events, the symptoms and signs detected on examination. Investigations such as x-rays, bone-scans and nerve conduction may also assist in some situations. The management of the problem will often involve a number of clinical staff including general practitioner, surgeon, pain specialist (often an anaesthetist), hand therapist and psychologists. Treatment will include one or more modalities.

Physical treatment: is an important part of the treatment of pain syndromes. This is to improve comfort, prevent or relieve stiffness and to resolve specific issues. This can range from simple measures such as massage, the application of heat and exercises to more sophisticated forms of hand therapy such as sensory re-education and mirror therapy.

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Adjunctive treatment: examples include trans-cutaneous nerve stimulation (TENS) and acupuncture.

Psychological treatment: the presence of pain can increase stress levels and make a person feel more anxious. Both of these altered states of emotion can heighten a person's experience of a pain. There are a range of techniques that can be helpful in reducing the experience of pain e.g. guided imagery, relaxation strategies and talking therapies.

Analgesic drugs (painkillers): aspirin, paracetamol or combined preparations such as co-dydromol.

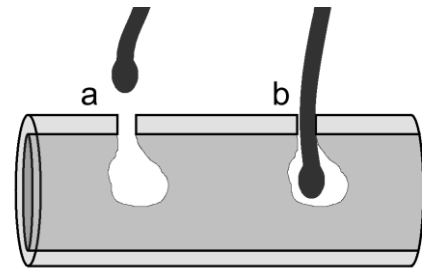
Neuropathic drugs: certain antidepressants (e.g. Amitriptyline) and anti-epileptic drugs (e.g. Carbamazepine, Gabapentin) have specific actions on nerve pain.

Counter-irritant drugs: (e.g. Capsaicin) can be massaged directly onto the injured area and have a similar effect to "deep-heat" preparations.

Sympathetic blockers: are agents that specifically act on the sympathetic pathways. Because of their potential general effects on the body, they have to be injected into and limited to the affected limb (Guanethidine block) or injected directly around the sympathetic chain in the neck (Stellate ganglion block).

Surgery: has a role in the management of some problems but has little or no role in others such as causalgia.

Neuroma burial deeply in soft tissues or bone can significantly improve comfort merely by removing them from points of contact and preventing them being knocked. This also can resolve any associated dysaesthesia, albeit at the cost of making the affected area numb (anaesthetic).



Nerve reconstruction may be considered in areas where nerve function is important. However, the surgery is more complex and recurrent symptoms are possible (see nerve grafting information sheet).

Nerve decompression may be indicated in some cases of RSD. Studies have shown that a significant number of patients have nerve compression (e.g. carpal tunnel syndrome) that certainly aggravates their symptoms and which may possibly be driving the disease process.