Complex Regional Pain Syndrome (CRPS)

Dr. Jean Mooney, PhD,
FChS, FCPodS, FCPodMed, FFPM RCPS (Glas), FHEA
Pain is regrettable but normal

- **Unpleasant but normal** sensory **and** emotional experience
  - **Sensory** awareness, via afferent neural input to thalamus
    - Modified by GABA inhibitory descending control mechanisms
  - **Emotional**, via afferent input from thalamus to the limbic system
    - Modified by GABA and other inhibitory neurotransmitters

- Associated with **actual, or the threat of, tissue damage**
  - Described in terms of ‘damage’

- **Normally resolves:**
  - trauma triggers release of inflammatory mediators + pain
  - inflammation triggers tissue repair
  - Inflammation abates (no more inflammatory mediators)
  - Pain resolves
Persistent Pain (1)

- Pain that **persists**
  - for >3/12
  - lasts beyond the normal healing time
- Pain impulses **generated independently** of the initial trigger event
- Pain becomes a **self-perpetuating** entity that continues after resolution of the initiating condition
  - Abnormal synapses form within the dorsal horn of the spinal cord
  - Release inflammatory neurotransmitters in dorsal horn
  - GABA-dependent descending control mechanisms no longer effective
Persistent Pain (2)

- Damage to nerve causes peripheral sensitization and hyperalgesia
  - Damage to peripheral nervous system
  - → ↑ activity of peripheral nerves and neurons within the dorsal root ganglia of spinal cord
  - → ↑ Na\(^+\) and Ca\(^{2+}\) channel activity in peripheral nerves = more action potentials generated
  - ↑ tissue sensitization to non-noxious stimuli
  - = Causalgia
    - Pain generates pain
Persistent Pain (3)

Injury / trauma to somatic tissue induces prolonged and continuing autonomic dysfunction

- SC-DRG neurones sprout links which stimulate sympathetic efferents
  - i.e.: peripheral injury causes central effect
- → Pain, hypersensitivity and vascular effects at injury site
  - i.e.: CNS activity causes peripheral effect
- = *Allodynia (CRPS)*
Proposed Mechanism of CRPS:

- **↑ sensory input from peripheral tissues**
  - (i.e.: actual or threatened tissue trauma)
  - → ↑ firing of dorsal horn cells and ↑ release of NMDA
  - N-methyl-D-aspartate in CNS triggered by release of arachidonic acid by damaged peripheral cells
  - ongoing pain

- → ↑ Ca\(^{2+}\) - generates formation intracellular second messengers → altered gene transcription both in CNS and periphery
  - ongoing release of inflammatory mediators
  - → peripheral vasodilatation and increased sudomotor activity

- → ↑ NO release - ↓ GABA release
  - loss of inhibitory effect of GABA in DH
  - pain impulses transmitted to thalamus and cerebral sensory cortex of
  - ongoing pain

- NO → ↑ neurotrophins → ↑ sprouting of sympathetic efferents
  - vasoconstriction and atrophic tissue changes
Peripheral events trigger local inflammation

- Initially:
  - Peripheral inflammation triggers CNS activity
  - → Release of pain-inducing neurotransmitters in CNS
  - = pain and hypersensitivity at the periphery

- Medium term:
  - Loss of pain inhibiting substances in CNS
  - = pain continues unabated

- Long term:
  - Increased activity of sympathetic nervous system
  - = peripheral vasoconstriction, pain, oedema and atrophic changes in peripheral tissues
CRPS

- Poorly understood condition
- Limb pain +
  - neurological (S, M, A) effects,
  - skin effects
  - bone abnormalities
- Sx: Severe and disabling
- Hx: trauma (or no trauma)
- Outcome: Effects disproportionate to severity of injury
- Other limbs may become involved (7%)
Epidemiology of CRPS

- Incidence ~ 1 : 2,500
- Age: 40-60 yrs
  - Mean Age of Onset ~ 46 yrs)
  - Can occur at any age
  - Becoming more common in adolescents and younger adults
- May complicate 5% (1:20) of all injuries
  - Follows 15% of peripheral nerve injuries
  - Follows 10-30% of limb fractures
Essential features of CRPS

- **PAIN**
  - Disproportionate, neurogenic, persistent

- +/- wide variety of symptoms!
  - Symptom onset within ~1/12 of exciting event (or immobilization)
    - Skin, nerve, bone Sx
  - Symptom variability → variable consultations
    - Orthopaedics; Neurology; Dermatology; Rheumatology
  - Symptom variability makes Dx difficult and often delays Dx

- Cause becomes evident after very thorough Hx
Main Symptom = PAIN!

- Burning limb pain
- Disproportionate to severity / extent of injury
  - Trigger event may be minor and / or forgotten
- Chronic in nature
- Spreads beyond original injury area
- Gets worse (rather than better) over time
Definition: CRPS = 2 presentations

- **CRPS 1**
  - AKA: *Allodynia*, Reflex Sympathetic Dystrophy (RSD); Sudek’s atrophy
  - Complex (painful neuropathy) of the limbs, arising after a minor or major injury.
  - Associated with
    - Severe ongoing, unremitting pain
    - Trophic changes in the nails, bone, and skin
    - Hypersensitivity of the affected limb
    - Permanent tissue dystrophy e.g.: Limb oedema.

- **CRPS 2**
  - AKA: *Causalgia*
  - S+S as CRPS 1, occurring as the result of an identified nerve injury
Criteria for Differential Diagnosis:

- **Is it CRPS Type 1?**
  - RSD / Sudek’s / **Allodynia**
  - No clear / obvious nerve injury

- **OR**

- **Is it CRPS Type 2?**
  - **Causalgia**
  - Easily identified nerve injury
Classification of CRPS

■ CRPS I:
  ■ **Alldynia**
  ■ Pain which develops in the absence of an *identifiable* nerve injury

■ CRPS II:
  ■ **Causalgia**
  ■ Pain in the presence of damage to a major nerve or nerve trunk

■ Marked and characteristic stigmata develop with both classifications
Signs of developing CRPS

- **Skin temperature changes**
  - 80% show unequal soft tissue temperature
  - Affected area warmer or cooler
  - Soft tissue temperature may fluctuate
  - Skin colour varies with temperature change (red / cyanotic)

- **Skin texture changes**
  - Dry and scaly or hyperhydrotic
  - Increased or absent hair growth
  - Nails – grow faster (+ brittle) or slower
  - Areas of skin break down
  - Diffuse woody (hard) or pitting oedema with clear demarcation line

- **Movement problems**
  - Movement limitation, especially initiating movement
  - Painful and stiff joints
  - Stiffness goes with sympathetic nerve bock
  - Disuse atrophy
  - Sudden / severe muscle spasms and / or involuntary limb jerks (dystonia)
Local Neurological Effects of Early CRPS

- Hypersensitivity especially to touch
- Allodynia (*pain from a non-painful stimulus*)
- Swelling
- Abnormal vasomotor activity
  - Spontaneous temperature changes
  - Hot ↔️ cold
- Abnormal sudomotor activity
  - Spontaneous sweating
- Abnormal pilomotor activity
  - Goosebumps
CRPS Type 1 - Allodynia

1: Initiating noxious event / imposed immobilization.
2: Continuing pain (hyperalgesia)
   - the pain is disproportionate to the inciting event.
3: History or evidence of local
   - Oedema
   - Changes in skin blood flow
   - Abnormal sudomotor (sweat gland) activity
   - Patchy peri-articular osteoaenia (shows at 3+ weeks)
4: No other likely cause for these symptoms

Note:
Criteria 2, 3, and 4 are essential to the diagnosis
Criterion 1 is not always present / recalled
No Lab-based diagnostic criteria
CRPS Type 2 - Causalgia

1: Continuing pain / hyperalgesia following a nerve injury,
   - Pain not necessarily limited to the distribution of the injured nerve.

2: History or evidence of local
   - Oedema
   - Changes in skin blood flow
   - Abnormal sudomotor activity

3: No other likely cause for these symptoms

Note: all 3 criteria must be satisfied.
CRPS: Causes, Risk factors, Incidence

- Limb injury, minor injury or infection (e.g. shingles)
  - It can also occur after heart attacks and strokes.
  - Sometimes may occur spontaneously (10% of cases)
- The cause of CRPS is imperfectly understood
  - Dysfunction of the sympathetic nervous system and CNS DH ‘wind up’
    - Autonomic dysfunction causes abnormal blood flow control / skin temperature changes within the affected area
    - CNS DH ‘wind up’ causes altered peripheral sensation
  - Leads to nerve, blood vessel, skin, fascia, bone, and muscle pathology.
- More common in people between the ages of 40-60 years
  - 1-5% incidence
  - 3:1 M:F incidence
  - 1:5 arise following surgery; 1:25 arise following an injection
  - Less common in younger people
Many triggers may induce CRPS

- Minor trauma (cut finger; nail surgery; injection)
- Head injury or Stroke
- Neuropathology: Poliomyelitis; ALS (ice bucket challenge!)
- Heart attack – myocardial infarction
- Surgery – carpal tunnel release
- Brachial plexus pathology
- Immobility – bed rest; immobilization in cast
CRPS: Onset and Progression

Most cases CRPS progresses through 3 stages.
- Stage 1: Acute phase
- Stage 2: Dystrophic phase
- Stage 3: Atrophic phase

May not always follow this progression.
- Some people progress to Stage 3 almost immediately
- Others remain in Stage 1 indefinitely.

Early diagnosis is essential for best prognosis and outcome
Progression

- **Continuity spread**
  - Gradual spread outward from the initial source

- **Mirror image spread**
  - Gradual symptom onset in other limb

- **Independent spread**
  - Occurs spontaneously (without any apparent trigger) or after secondary trauma

- **Psychological effects**
CRPS **Staging**

- Used to be classed as a 3-stage disease
  - Stage I: Mild (reversible),
  - Stage II: Moderate (reversible),
  - Stage III: Severe (irreversible changes)

- **Convenient but not accurate classification**
  - Stage I may progress to Stage III very rapidly
  - Some cases never progress beyond Stage 1 but do not recover
CRPS: Assessment of severity

- **Stage 1: Mild CRPS**
  - Few signs of pain, disability or distress
  - Pain managed with simple analgesia

- **Stage II and Stage III CRPS**
  - Moderate / severe S+S at presentation
  - Dystonia
  - Failure to respond to treatment modalities
  - Short lived improvements +/- continuing and ongoing deterioration
CRPS: Stage 1 – Acute phase

- Duration: 1-3 months from onset of pain
- Initially hot / warm; later cold / cool
- Hyperaesthesia / Pain, exacerbated by the slightest stimulus
- Tissue oedema (non-pitting)
- Skin becomes dry and thin
- Skin initially red / mottled; later cyanotic
- Nail and hair growth increases in early phase / slows later
- Hyperhidrosis in early phase, anhidrosis later
- S+S initially localised, but later tends to spread beyond the original affected area
- Symptoms are reversible
CRPS: Stage 2 - Dystrophic phase

- Persists for 3-6 months (i.e.: 4 - 6/12 post onset)
- Pain, hyperalgesia and hypersensitivity persists
- Oedema increases and spreads
- Area is cold
- Skin becomes thin, shiny, pale and dry
- Loss of sub-cuticular tissues (fat pad atrophy)
- Decreased nail and hair growth
- Loss of hair
- Bone changes noted on X-rays or bone scan: e.g.: patchy osteopaenia
- Laser Doppler shows reduced blood flow
- Stiff muscles and joints

*Symptoms are reversible*
CRPS: Stage 3 - Atrophic phase

- Begins 4/12 – 9/12 post pain onset
- Pain may affect the entire limb
- Permanent tissue changes develop
  - Muscle wasting
  - Extensive woody oedema
  - Limited limb or limb segment mobility
  - Muscle contractures (unequal pull)
  - Digital and foot deformities and pathologies II° to soft tissue contractures
- Depression and / or mood changes
  - Central (CNS limbic-system) effects
- *Symptoms become irreversible = permanent tissue change*
Diagnosis of CRPS

- Clinical diagnosis
- Difficult, especially in the early stages of CRPS (physical symptoms take longer to show)
- RCP recommends use of the ‘Budapest Criteria’ to make a definitive diagnosis, i.e. increasing local and spreading pain +
  - Local sensory changes
  - Local vasomotor changes
  - Local sudomotor changes +/- oedema
  - Motor / trophic changes
Budapest Criteria for Dx of CRPS

- **Pain**
  - Disproportionate to the initiating or causative event
  - Radiating beyond involved / affected tissues
- **2 signs of change from within the 4 categories**
  - sensory, vasomotor, sudomotor/oedema and / or motor/trophic changes
- **OR 1 sign of change within of 3 these categories**
  - sensory, vasomotor, sudomotor/oedema and / or motor/trophic changes
- **No other diagnosis would better fit** the signs and symptoms
Budapest Criteria of CRPS Diagnosis: Sensory (Category 1)

- **Allodynia** = hypersensitivity to
  - light touch
  - temperature changes
  - deep somatic pressure
  - joint movement
- **Hyperalgesia** = excessive pain awareness to
  - to pinprick
- Allodynia and hyperalgesia both count towards the diagnosis, whether they are
  - reported as symptoms
  - or discovered by test (signs)
Budapest Criteria of CRPS Diagnosis: Vasomotor (Category 2)

- Temperature differences
  - Sign: tissue temperature difference >1°C
- Skin colour changes
  - Red / mottled /cyanotic / palor
- Skin colour asymmetry
Budapest Criteria of CRPS Diagnosis: Sudomotor (Category 3)

- Oedema and/or sweating
- Asymmetric sweating
  - Hyperhidrosis
  - Anhidrosis
- Inappropriate sweating / lack of sweating
Budapest Criteria of CRPS Diagnosis: Motor / Trophic (Category 4)

- Reduced joint ROM
- Motor dysfunction
  - Weakness
  - Tremor
  - Dystonia
- Trophic changes
  - Change in hair growth (loss, coarseness)
  - Change in nail growth (brittleness; increased or decreased growth)
  - Skin changes (atrophy, colour and texture changes)
Table. Budapest Criteria for CRPS

All of the following statements must be met:

- The patient has continuing pain that is disproportionate to any inciting event
- The patient has at least 1 sign in 2 or more of the categories below
- The patient reports at least 1 symptom in 3 or more of the categories below.
- No other diagnosis can better explain the signs and symptoms.

<table>
<thead>
<tr>
<th>No.</th>
<th>Category</th>
<th>Signs/Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sensory</td>
<td>Allodynia (pain to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement) and/or hyperalgesia (to pinprick)</td>
</tr>
<tr>
<td>2</td>
<td>Vasomotor</td>
<td>Temperature asymmetry and/or skin color changes and/or skin color asymmetry</td>
</tr>
<tr>
<td>3</td>
<td>Sudomotor/edema</td>
<td>Edema and/or sweating changes and/or sweating asymmetry</td>
</tr>
<tr>
<td>4</td>
<td>Motor/trophic</td>
<td>Decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nail/skin)</td>
</tr>
</tbody>
</table>

Based on reference 3.
So, Budapest Criteria mean that…..

- CRPS is **classified by changes**
  - CRPS: Observed changes (oedema, sweating changes) may abate over time
  - CRPS-NOS (not otherwise specified) – suspect CRPS if nothing else fits the presenting symptoms

- CRPS diagnosis should be **suspected without delay**
  - In order to initiate early treatment.
  - Early treatment tends to prevent or reduce late stage complications
Tests used in the diagnosis of CRPS (1)

- **Bone imaging** – patchy osteopaenia
  - Radiographs
  - 3-phase bone scan

- **Skin temperature** (thermography)
  - Hyperaemia and warmth in early stage
  - Cyanosis and coolness in later stage

- **Sudomotor function**
  - Resting sweat output (98% specificity)
  - Stimulated sweat output test
Tests used in the diagnosis of CRPS (2)

- **Nerve function**
  - Sensory function tests should be normal in Type 1 (A), but abnormal in Type 2 (C)
  - Single fibre EMG may be intolerable to patient with CRPS
  - C-fibre dysfunction difficult to detect

- **Laser Doppler**
  - Baseline and stressor tests (e.g.: inspirational response)

- **Blockade of the sympathetic ganglia**
  - Unreliable as other pain syndromes give a similar response
CRPS - Investigations

- *Clinical* diagnosis – based on presenting signs and symptoms (Budapest Criteria)
- Investigations may or may not be helpful
  - Blood tests do not support or exclude diagnosis of CRPS
  - Radiographs, EMG, Nerve conduction studies, CT scans, MRI scans may be entirely normal
- X-ray may show patchy osteoporosis in up to 70% of cases, in due course
- Technetium $[^{99\text{m}}\text{Tc}]$ bone scan may show increased or decreased up-take
- Thermography may show changes in tissue temperature
CRPS: Problems of Diagnosis

- Symptoms change with stage of the problem
- Nerve function tests not much used
- Sympathetic blockade
  - Does not differentiate CRPS from other nerve disorders
- Laser Doppler
  - Loss of autonomic-mediated inspiration-related skin blood flow changes
- Sweat tests
  - Loss of normal sweat response to imposed stress

Bottom line – if you suspect CRPS, it probably is
- Presume CRPS in patient with persistent pain after minor trauma or surgery until you are proved to be a pessimist
CRPS: Differential Diagnoses (1)

- **Musculoskeletal**
  - Bursitis
  - Myofascial pain syndrome
  - Undiagnosed local pathology e.g.: fracture / sprain

- **Neurologic**
  - Post-stroke pain syndrome; Peripheral neuropathy Postherpetic neuralgia Radiculopathy

- **Infectious**
  - Cellulitis
  - Infectious arthritis (NB: TB)
CRPS: Differential Diagnoses (2)

- **Vascular**
  - Raynaud's disease
  - Thromboangiitis obliterans (Buerger's disease)
  - Venous thrombosis
  - Traumatic vasospasm

- **Rheumatoid**
  - Rheumatoid arthritis
  - Systemic lupus

- **Psychiatric**
  - Factitious disorder
  - Hysterical conversion reaction
CRPS: Therapeutic Approaches

- **Education**
  - Patient, family, carers

- **Pain reduction**
  - Mainstream drugs, alternative therapies, CBT

- **Physical rehabilitation**
  - Physio and mechanical therapies

- **QoL improvement**
  - Taking care of the inner man/woman
Treatment modalities:

- Pharmacological management
  - Pain / Depression

- Physical therapies:
  - Gradual weight bearing (essential to reduce disuse osteoporosis) via e.g.:
    - gliding exercises
  - Desensitization techniques

- Occupational therapy:
  - Stress loading (to achieve decreasing sensitivity to gradually increasing stimuli) e.g.: via skin massage

- Recreational therapy:
  - Socialisation; Support groups

- Vocational therapy:
  - Return to work

_i.e. Focus on functional restoration through interventional therapies and pharmacological management_
CRPS: Management Principles

- **Multidisciplinary** approach (centred on GP +/- Pain Team)
- **Continuity** of care
  - Personnel
  - Management regime
- Begin treatment **ASAP**
  - <3/12 of onset of first S+S
- Consider **early referral**
  - Early treatment = early remission
- Pain flares (**exacerbations**) occur from time to time
  - Usually settle in a few weeks
  - Maintain meds but reduce intensity of physical therapies
Pharmacological management

- Wide range of pharmacological treatment options = no one ‘magic bullet’
  - Varying medications used to gain the best outcome for the individual patient
- Pharmacological treatment alone is not enough
- Early pharmacological intervention necessary
CRPS: Medications regimes

Initially:
- Simple analgesics: low dose, titrated up as necessary
- E.g.: NSAIDs

If pain non-responsive after 3-4/52
- Medication for neuropathic pain
- TCAs; Gaba-Pentin

Plus: encourage limb use; gentle exercise, desensitization techniques
- Medication for any associated depression

Pamidronate as a 1-off in first 6/12 of non-responsive CRPS
CRPS: Pharmacological Management (1)

- Analgesics
  - NSAIDs
  - Corticosteroids (AIs)
  - Tramadol (narcotic analgesic)

- Antidepressants
  - Amitriptyline (anti-depressant)
  - Doxepin (tri-cyclic AD)
  - Nortriptyline (major AD)
  - Trazodone (sedative AD)

- Anticonvulsants
  - Carbamazepine (*Tegretol*)
  - Phenytoin (*Dilantin*)
  - Gabapentin (*Neurontin*) – good for neurogenic pain
## CRPS: Pharmacological Management (2)

<table>
<thead>
<tr>
<th>Category</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-arrhythmics</td>
<td>Anti-arrhythmics</td>
</tr>
<tr>
<td></td>
<td>Mexiletine (similar effect on smooth muscle as lidocaine)</td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Calcitonin injections – reduce blood levels of Ca(^{2+})</td>
</tr>
<tr>
<td></td>
<td>Pamidronate</td>
</tr>
<tr>
<td>Oral opioids (controversial)</td>
<td>Hydromorphone (opioid analgesic)</td>
</tr>
<tr>
<td></td>
<td>Morphine</td>
</tr>
<tr>
<td></td>
<td>Oxycodone (codeine derivative)</td>
</tr>
<tr>
<td>Muscle relaxants</td>
<td>Clonazepam (sedative)</td>
</tr>
<tr>
<td></td>
<td>Baclofen (GABA agonist)</td>
</tr>
</tbody>
</table>
CRPS: Pharmacological Management (3)

- **Sympathomimetics**
  - Clonidine (reduces sympathetic tone) patch
  - Phentolamine (alpha blocker) IV
  - Epidural blocks
    - Guanethididine (prevents release of nor-adrenalin)
    - IV (Bier’s) block
  - Bretylium (ditto)
  - Ca\(^{2+}\) channel blockers,
  - Beta / Alpha blockers

- **Topical analgesics**
  - Capsaicin cream (counter-irritant)
  - Lidocaine transdermal 5% patches
CRPS: Referral onward from primary care

- **Confirmed cases** may be managed in primary care
  - GP; Pain Clinic
  - So long as Sx are mild and are responding to treatment
- **Non-responsive, severe, and rapidly progressing cases** should be referred to a specialist unit to ensure
- **Accurate diagnosis of CRPS essential**
  - Exclusion / identification of all other causes of presenting S+S
  - Control of difficult S+S (pain, disability, distress)
  - Correct / appropriate functional rehabilitation
CRPS: Rehabilitation

- Considered for all cases
- Started early
- Multi-disciplinary approach
- Group pain management programmes
  - Identify and address psychosocial factors that might reduce patient’s response
  - Identify and address previous –ve experiences, poor coping strategies, depression
CRPS: Treatment approaches

- Education and support
- Desensitization techniques
- Postural control
- Oedema control

- Specialist techniques to reduce intractable pain
  - Mirror visual feedback
  - Graded motor imagery
  - Spinal stimulation
  - Epidural Clonidine + LA
  - Sympathetic chain LA
  - IV regional sympathetic blocks
  - Inter-scalene indwelling catheters
  - Intra-thecal Baclofen and / or serial splinting for dystonia
Plus, as necessary:

- **Referral back to orthopaedic / podiatric surgeons**
  - 25% CRPS follow limb fracture / orthopaedic surgery
  - Rule out another related pathology
  - Scars may cause nerve compression
  - Further surgery is contra-indicated as it may cause further CRPS
  - Never resort to amputation

- **Referral to other specialisms**
  - Rheumatology, Neurology, Neurosurgery
    - Rule out / treat other possible causes of severe pain
  - Dermatology
    - For identification of other causes: post-herpetic neuralgia, vasculitis, ulcers
    - For treatment of CRPS-related skin problems:
      - Skin atrophy, hyper-trichosis, bullae, leukonychia
CRPS: Patient education and support

- **Written ‘contract’ / protocol** of suggested approaches within the care plan
  - Nerve blocks, medications, physical / occupational therapy, psycho-social issues, lab tests, consultations etc
  - Patient commitment to treatment approaches

- **Psychological assessment** from an expert in chronic pain
  - To outline and address other contributory factors
    - Relaxation problems, low self-esteem, inappropriate coping strategies, social support issues, suicidal ideation
  - To initiate interventions
    - Cognitive behavioural therapy
    - Relaxation techniques
CRPS: Complications

- **Most recover** during the 6-18/12 post trauma
  - Others never recover

- **Ongoing problems** may include
  - Depression
  - Pain and stiffness due to immobilization
  - Skin infections
CRPS: Prognosis

- **Duration varies**
  - Mild cases: few weeks followed by full remission
  - Many cases: ongoing pain for months – years
  - Some cases: indefinite pain
- **50% have pain for >6/12**
  - 15% have unrelenting pain and disability for up to 2 years
- Some patients show a *remission / exacerbation* pattern
  - Exacerbations may last for weeks, months or years
- May *spread to involve all limbs* if left untreated
  - Significant morbidity
  - Costly in terms of management and support for chronic pain and associated deformities
  - Significant psycho-social and psychiatric issues
  - Potential drug dependency
  - Total incapacity
- Early diagnosis and intervention permits the very best outcome
CRPS: Prevention

- The mechanism of spontaneous pain in CRPS is still not fully understood
  - But once decoded it could allow new means to block / prevent CRPS pain
- Early ‘aggressive’ intervention improves prognosis in CRPS
  - Don’t wait for the condition to come established….be alert to increasing pain levels post trauma
- High levels of Vit C seem to link to a lower incidence of post-fracture CRPS
  - Fracture patients should be advised to maintain Vit C levels during bone healing
Complex regional pain syndrome

- Is the most complicated neuropathic pain syndrome
- Causes autonomic, sensory and motor effects that are at least very distressing and painful, and at worst – permanent
- Patients undergoing surgery should be warned of the possibility of developing CRPS
- The pathophysiology of the disease is poorly understood
- Treatment modalities are wide and various and many are not research (RCT) based
- Early referral to a Pain Clinic gets the best results
CRPS: **Summary**

- **Chronic pain syndrome**
  - Poorly understood mechanism

- **Linked to (incipient) trauma**
  - CNS and PNS changes
  - Skin, soft tissue, blood flow, bone changes

- **Best response to early treatment**
  - Simple PKs + physical therapies
  - Compound PKs + supportive interventions prn
Just wondering.......... 

- There are many similarities between CRPS and the painful and non-painful neuropathies of DM
  - Pain (in CRPS and painful neuropathy) limits movement, so osteoporotic bones tend not to fracture
  - Loss of pain (in non-painful neuropathy) allows movement and Charcot neuroarthropathy occurs

- If this is true, then what we class as peripheral sensory neuropathy in DM should show the same autonomic and CNS effects as CRPS.....
  - Volunteer needed to do the PhD.................!!