

## **Oxford Textbook of Rheumatology**

### **Pain and fatigue**

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## **Abstracts**

### **Chapter and Book**

Pain and fatigue are the prominent problems for those with a Rheumatic disease, and are often underestimated by clinicians. Symptoms may fluctuate in quality and intensity over time and commonly will vary over the course of a day. For pain, clinical signs and symptoms will be dependent on the source of the pain and whether causative underlying pathology is identifiable or not. Fatigue may range from mild effects to total exhaustion and may include cognitive and emotional elements, with a complex, probably multi-causal pathway. Theoretical knowledge of potential mechanistic pathways for pain and fatigue should be used to inform assessment and treatment approaches. Best practice recommends a multi-disciplinary and holistic treatment approach with the patient an active participant in the planning of their care, and self-management. Many patients with chronic musculoskeletal conditions will not achieve a pain-free or fatigue-free status. Medication use must therefore balance potential benefit against short and long-term side effects. Rheumatology centres should offer specific fatigue and pain self-management support as part of routine care. Emphasis should be given to facilitating self-management strategies for both pain and

fatigue to help the patient optimise their quality of life over years or a lifetime of symptoms. Interventions should include behaviour change and cognitive-restructuring of pain/fatigue beliefs, as well as access to relevant self-help groups and charitable organisations. Referral for specialist advice from regional or national clinics on pain relief and management should be considered if pain interferes significantly with function or quality of life despite local interventions.

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## **1. Introduction**

Pain and fatigue are cardinal symptoms of the Rheumatic diseases. The majority of patients will experience them over the course of their disease though the intensity and quality of them may vary over the time course. This chapter will focus on the identification and management of pain and fatigue in the clinical setting and provide a broad overview of the mechanisms that drive these symptoms and their incidence. In each section pain is discussed first followed by fatigue.

## **2. Epidemiology**

### **2.1 Pain**

Between 25-30% of the total adult population, and approximately 8% of children, in both developed and developing countries may be affected by musculoskeletal pain<sup>1,2</sup>. As the mean age of the global population rises, it is likely that these data will increase further. In those over 85 years of age, joint pain is estimated to effect between 12.9% and 57.0% with the lower limb most commonly involved.

The financial burden of musculoskeletal pain is also high. UK data for chronic low back pain alone has estimated direct health care costs of £1632 million per year with indirect costs of £10668 million<sup>3</sup>. In the US, common pain conditions account for a loss of 13% of the total workforce at any one time and 6.2% of this group report some form of arthritis or musculoskeletal pain<sup>4</sup>.

## **2.2 Fatigue**

Fatigue is experienced by over 90% of people with a rheumatic condition, who often rate their fatigue as more severe than their pain<sup>5</sup>, with severity levels as severe as those seen in Chronic Fatigue Syndrome<sup>6</sup>. People without a long term condition report mean fatigue scores of 20.5 on a Visual Analogue Scale of 0-100, while in rheumatic conditions fatigue ranges from 40.8 (Psoriatic Arthritis) to 74.4 (Primary Sjorgren's Syndrome), with fatigue scores for people with Rheumatoid Arthritis Osteoarthritis, Lupus, Ankylosing Spondylitis and Fibromyalgia lying between those two extremes<sup>7</sup>. The financial burden of fatigue in rheumatic disease is not readily identifiable, but people with inflammatory arthritis consider fatigue the primary cause of days lost from paid work<sup>8</sup>.

## **3. Definitions of pain and fatigue**

### **3.1 Pain**

Pain is defined by the International Association for the Study of Pain as:

“An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”

For those with a rheumatic disease pain is usually experienced in its chronic form ( $\geq 12$  weeks duration) and most commonly falls within one or more of the following classifications: musculoskeletal, inflammatory, neuropathic or a dysfunctional state.

#### ***3.1.1 Musculoskeletal pain***

Musculoskeletal pain is pain that arises from any structures within the musculoskeletal system such as bone, muscles, tendons and/or ligaments. Pain may arise from mechanical dysfunction within a joint, such as in advanced Osteoarthritis, or from the bone, such as in Paget's disease or malignancy. Pain that arises from the muscles alone is commonly referred to as '**myofascial**' pain.

### ***3.1.2 Inflammatory pain***

Inflammatory pain arises from tissue-damaged associated inflammation that is usually accompanied by oedema, redness, heat and a possible reduction in function. When present in the joints it is commonly associated with a perception of stiffness.

### ***3.1.3 Neuropathic pain***

Neuropathic pain may be central or peripheral in origin and is defined as 'pain caused by a lesion or disease of the (central/peripheral) somatosensory system'<sup>9</sup>. Clinical signs of neuropathic pain may include evidence of **allodynia** (pain to a stimulus that is not normally painful) and **hyperalgesia** (increased pain to a stimulus that normally evokes pain) amongst others (see IASP Taxonomy<sup>10</sup>).

### ***3.1.4 Dysfunctional state***

This is the term given to conditions where there is a lack of structural cause for the persistent pain experienced, such as Fibromyalgia and Complex Regional Pain Syndrome<sup>11</sup>. People with these conditions may present with the clinical signs and symptoms of other pain states, such as neuropathic and myofascial pain but there is no evidence of an underlying pathology.

## **3.2 Fatigue**

Fatigue has been defined and classified in a number of ways, yet there is no overall consensus. In terms of the fatigue that is related to musculoskeletal conditions, a broad conceptual definition appears the most appropriate:

“Fatigue is a subjective, unpleasant symptom which incorporates total body feelings ranging from tiredness to exhaustion, creating an unrelenting overall condition which interferes with individuals’ ability to function in their normal capacity”<sup>12</sup>.

Here, the term ‘function’ should be assumed to include physical and/or cognitive function. The term ‘tiredness’ might be disputed, as people with rheumatoid arthritis consider tiredness to be the phenomenon experienced by healthy people, and believe the terms ‘fatigue’ or ‘exhaustion’ are necessary to encapsulate the intensity of the symptoms experienced within chronic illness<sup>13,14</sup>.

#### **4. Aetiopathogenesis**

##### **4.1 Pain mechanisms**

The pain pathway describes the anatomical route that a peripheral stimulus takes to the brain, via peripheral nerves, dorsal horn, and spinal cord, to register as a painful perception. Central projections of the pain pathway from the thalamic nuclei include somatosensory cortex and the limbic system. Although the pain pathway is a credible neuroanatomical concept, multiple brain areas are activated during a pain experience in addition to the classic lateral sensory–discriminative and medial emotional–affective areas involved in pain processing. Melzack and others have proposed that this extensive network of brain regions activated during pain perception, represents a specific cerebral signature, a pain Neuromatrix<sup>15</sup>.

A pain experience can be modified by dysfunction at multiple levels along this pathway, ranging from changes in the molecular constituents of the peripheral inflammatory milieu to alterations in the complex circuitry of cortical representation. Disease states, psychosocial factors plus medical interventions can influence the activity of the pathway.

Despite the elaborate multifaceted system of pain perception, broad types can be identified which correlate with known pain mechanisms (see **3.1**). It should be noted

that much of the research that underpins our knowledge of the neurobiology of pain is undertaken in animal models.

Nociceptive pain is caused by stimulation of peripheral nerve fibres (A $\delta$  and C fibres) via tissue pathology such as inflammation, ischaemia and mechanical stimuli such as pressure and heat. C fibres can release local peptides to induce 'neurogenic inflammation' via neuropeptides such as substance P and calcitonin gene-related peptide (CGRP), which induce vasodilatation, plasma extravasation, and interaction with immune cells.

Neuropathic pain (see **3.1**) arises via nerve signalling from ectopic discharges of damaged neurones or those induced by adjacent healthy nerves, including sympathetic system fibres. Both nociceptive and neuropathic signals can be modified along the pain pathway, so that a given stimulus is amplified - sensitisation. At the site of tissue damage and inflammation, a large number of mediators can cause the reduction in excitation thresholds of peripheral nociceptors, thereby increasing signalling to the spinal cord dorsal horn. These alterations to the properties of ion channels can be short lived as in the case of prostaglandins or bradykinins, or much longer lasting such as cytokines (including TNF $\alpha$ ) and Nerve Growth Factor. There are also important inhibitory mechanisms present in peripheral tissues, and the balance between pro-inflammatory /excitatory mechanisms and the inhibitory cytokines, cannabinoid and opioid systems play an important role. The peripheral nerve message can be amplified on a molecular level by complex intracellular mechanisms which via second messenger systems, modulate gene transcription.

Central sensitization is considered the main pathophysiological mechanism responsible for neuropathic pain and contributes significantly to nociceptive pain.

Peripheral nociceptor hyperactivity causes major secondary changes in the spinal cord dorsal horn, such as enhanced AMP acid (AMPA) NMDA receptor activation in the

dorsal horn, mediated by protein kinases, and upregulation of the signalling cascade that modulates gene transcription (i.e., *c-fos*, *c-jun*). In addition, changes in the descending modulatory systems, and functional changes in spinal cord microglial cells and astrocytes contribute to the enhanced signal transmission of central sensitisation.

#### ***4.1.1 Specific joint & musculoskeletal pain mechanisms***

Features that reflect local joint pathology and characteristics such as mood, social circumstances and central pain processing mechanisms determine pain report in arthritis. Both peripheral and central mechanisms contribute to chronic arthritis pain<sup>16</sup>. Peripheral mechanisms such as inflammation, oxidative stress and ischaemia may be more important in the early disease, with central mechanisms having a greater influence on pain report and disability in later stages. Patients with arthritis often describe a “neuropathic pain” pattern, but whether this reflects true neuropathic pathology or the complex pain experience derived of multiple dysfunctional systems is unclear.

Joints are well innervated by sensory, sympathetic and nociceptive fibres, though large proportions of them are usually insensitive to normal stimuli. These normally quiescent nerve fibres, the ‘silent nociceptors’, can be sensitised by peripheral joint pathology and respond to mechanical stress such as (normal) movement. Joint effusion, especially in chronic arthritis where the synovium and capsule are less compliant, causes significant increases in intra-articular pressure that can activate joint nociceptors.

##### ***4.1.1.1 Rheumatoid arthritis***

Despite modern treatment approaches a majority of patients with RA still describe chronic pain. Some of the abnormal pain processing can revert to normal after prolonged periods, as evidenced by restoration of pain threshold and descending pain



analgesic pathways, following joint replacement. There may be differences in chronic pain due to OA and RA, for example RA patients have lower pain thresholds than controls<sup>17</sup>. This may reflect subtle differences in joint innervation in chronic synovitis, combined with altered pain processing, endogenous opioid systems and descending control.

#### ***4.1.1.2 Osteoarthritis***

Why an osteoarthritic joint is painful is not clear. Multiple joint structures are abnormal in established OA. It is likely that combinations of peripheral inflammation, synovitis, bone marrow lesions (seen with MRI scanning), mechanical stress on periarticular structures along with peripheral and central sensitisation are responsible. Whether clinical examination and readily available imaging can predict which of these is the dominant pathway and therefore guide treatment is debatable. Poor outcome from joint replacement may be related to preoperative pain experience, such as rest pain, night pain and low pain threshold which may be construed as neuropathic.

#### ***4.1.1.3 Fibromyalgia***

The chronic widespread pain of fibromyalgia is a common presentation to rheumatologists. Extensive clinical observation, quantitative sensory testing, and functional neuroimaging studies all point towards significant abnormalities in pain perception; widespread multisystem pain report, diffusely lower pressure pain thresholds, temporal summation, deficiencies in descending pain modulation and cognitive disturbance. This evidence suggests a global amplification of central pain processing. The relative contributions of aberrant descending analgesic serotonergic-noradrenergic system activity, peripheral and central sensitization is unclear. Further imaging studies have suggested that spontaneous pain is related to enhanced activity

in multiple brain networks, additional evidence of a disseminated pain processing problem<sup>18</sup>.

## **4.2 Fatigue mechanisms**

The causal mechanism for fatigue in musculoskeletal disorders remains an issue of debate fuelled by poor study design and conflicting results from studies that are largely cross-sectional using small or biased samples (eg patients in flare). Whilst some studies show fatigue may be causally linked to inflammatory processes, mood, disability, sleep, pain, behaviours and beliefs, the evidence is inconsistent<sup>19</sup> and associations vary across musculoskeletal conditions<sup>20</sup>. In inflammatory musculoskeletal conditions, it has been suggested that IL-1 and IL-6 might be a partial explanatory mechanism<sup>21</sup>.

It is proposed there are three main elements driving fatigue in musculoskeletal conditions (Fig 1)<sup>22</sup>. Disease processes including inflammation and pain may affect fatigue directly, or indirectly through poor sleep, disability and de-conditioning. Cognitions, feelings and behaviours may interact to produce over or under physical activities such as boom and bust behaviours. These three elements interact to form a multi-causal pathway, where for each patient, different factors are present in different strengths and interact to produce fatigue, with different combinations on each occasion<sup>22</sup>. Such an aetiology would account for a symptom that occurs across the whole spectrum of musculoskeletal conditions, disease durations and levels of inflammation, and also allows for fatigue as both cause and effect (eg on occasions fatigue may be driven by low mood, and on others, low mood may be driven by fatigue).

## **5. Clinical features**

### **5.1 Pain**

Pain may be intermittent or continuous and vary in nature depending on the cause and course of the disease.

Pain arising from inflammation, such as in RA, will be accompanied by the other classical signs (see **3.1.2**) and is commonly described as throbbing in nature, at rest and on movement, and exacerbated by use of the affected part. The descriptions that patients with RA use to describe their pain may alter depending on the time of day, the duration of their disease, the joints that are involved and whether those joints are moving or at rest<sup>23</sup>. Diurnal variation of pain and stiffness is common with patients describing an increase in intensity of these symptoms at the start and end of the day. Myofascial pain may be diffuse and poorly localized with reports of a cramplike, dull aching pain either at rest or in motion<sup>24</sup>. Other musculoskeletal pains, such as Osteoarthritic joint pain, is typified by the sufferer describing stiffness on initial movement, increasing pain with prolonged movement and disturbed sleep due to pain<sup>25</sup>. There is a reduction in the range of movement in the affected joint and associated muscle weakness.

Neuropathic pain is typically burning in quality and commonly associated with allodynia (see **3.1.3**). Decreased (hypoesthesia) and increased (hyperaesthesia) sensitivity to stimulation may co-exist in the affected region and itchiness may also be present. The patient may avoid touch to the affected part by themselves and others. They may prefer to keep allodynic areas free from clothing during the day and the bedclothes at night.

For those with Fibromyalgia they report more widespread chronic pain that is commonly associated with fatigue and psychological distress<sup>26</sup>. Although hyperalgesia and allodynia are commonly reported at specific trigger points these sensations often spread far beyond these areas with sufferers describing generalised sensitivity<sup>27</sup>.

## **5.2 Fatigue**

Fatigue has a very similar picture across RA, lupus, OA and fibromyalgia, with numerous qualitative research studies evidencing common perceptions of the nature, consequences and attitudes toward fatigue (eg<sup>13,28,29</sup>). Fatigue is the physical feeling of a heavy body that seems hard to move ('paralysed') and has run out of energy. Fatigue also has a cognitive element, with descriptions of 'foggy-headedness', with difficulty concentrating or focussing, a feeling of an 'absent presence'. The extremes of this physical and mental fatigue are akin to being 'wiped out' with exhaustion or 'overwhelmed'. Fatigue occurs on most days for many people and varies in intensity, with duration ranging from hours to weeks.

Fatigue threatens people's normal roles. It reduces or even stops people's ability to perform everyday activities and chores, social and leisure activities, voluntary and paid work, either because of physical or cognitive exhaustion. It has emotional consequences from frustration, leading to anger and depression, and may cause difficulties with relationships when social arrangements are cancelled. This is compounded by a reluctance to discuss fatigue as it is invisible, therefore people feel disbelieved or fraudulent, and struggle with the society's general perception that everybody gets tired. People with musculoskeletal conditions differentiate fatigue from the tiredness experienced by healthy people by its unpredictability, lack of causality ('unearned'), intensity (tired vs fatigue or exhaustion), constancy, and the fact that rest does not lead to recovery.

## **6. Management of pain and fatigue**

### **6.1 Pain**

To manage pain effectively, it is necessary to recognise the differing components that contribute to the subjective and personal experience termed 'pain'. Therefore the

approach to the management of chronic musculoskeletal pain should be multidisciplinary<sup>30-38</sup>.

### **6.1.1 Pharmacologic (see Fig.2)**

The use of oral analgesics should balance the beneficial effects on pain against the risk profile of the medication in that specific patient<sup>39</sup>. Many patients with chronic musculoskeletal pain will not achieve a pain-free state. Referral to a pain clinician should be considered when; a patient is gaining medications on increasing doses for little further impact on pain but accruing short and long-term adverse effects, or is on high dose strong opioids. Patients with concurrent chronic pain states or neuropathic pain may also need neuromodulatory agents and specialist pain advice.

### **6.1.2 Non-pharmacologic treatment- multidisciplinary approach in chronic pain**

Exercise should be a core treatment irrespective of age, comorbidity, pain severity and disability, and should include local muscle strengthening and general aerobic fitness<sup>30</sup>. Physiotherapy will play a key role in initiating and advising how a patient can safely exercise. Weight reduction is beneficial, and patients may need encouragement and advice. Many patients may have significant psychosocial comorbidity including depression and anxiety, which untreated will hamper pain management. Psychological support such as counselling and cognitive behavioural approaches should be considered<sup>30,31</sup>. Orthotics, podiatry, physiotherapy and occupational therapy can provide advice on appropriate braces, splints, assistive devices and walking aids. Assessment of footwear and provision of insoles may relieve pain, improve gait and posture and help to reduce falls.

### **6.1.3 Pain management approach**

Patients with chronic musculoskeletal pain who have exhausted medical and surgical management options and continue to struggle coping with their symptoms should be considered for a multidisciplinary pain management approach<sup>40,41</sup>. Pain management

programmes can be out-patient or in-patient based, and are usually accessed through the pain clinic<sup>41</sup>.

## **6.2 Fatigue**

Guidelines by the British Society for Rheumatology and NICE on the management of RA recommend that fatigue is addressed, but do not provide guidance on management. Assessment could be by Visual Analogue Scale or Numerical Rating Scale, or by multi-dimensional questionnaires, which yield a more complete picture of different dimensions of fatigue. Such PROMs need to be well-validated, and can be selected from a comprehensive review of fatigue PROMs in musculoskeletal conditions<sup>7</sup>.

### **6.2.1 Pharmacologic**

In RA, Cochrane reviews of biologic, and non-biologic pharmacological (disease-modifying) therapies on fatigue are underway (2011). A systematic review of biological therapies suggests small effects on RA fatigue, as does an overview of disease-modifying therapies<sup>42,43</sup>. This small effect maybe because inflammation is not always main driver for fatigue. The presence of fatigue in the absence of inflammation, is currently not an indication for changing or starting biologic or disease-modifying therapies. In Fibromyalgia, meta-analyses show no effect for gabapentin or pregabalin on fatigue but there is evidence that anti-depressants may reduce fatigue in the short-term<sup>44</sup>. No reviews of pharmacological interventions could be found for fatigue in other musculoskeletal conditions.

### **6.2.2 Multidisciplinary team interventions to support self-management of fatigue**

Given the presumed multi-causal pathway for fatigue, and the consequent limited evidence for pharmacological interventions, the multidisciplinary team should aim to support the patient in their self-management of fatigue. A Cochrane review of non-pharmacological fatigue interventions in RA is underway and currently, meta-analysis

shows small effects from psychological interventions<sup>45</sup>. However, none of the interventions had fatigue reduction as the main aim: fatigue was a secondary outcome and thus patients were not recruited with fatigue, and the studies were not powered for fatigue. A review of arthritis self-management programmes shows courses that aim to enhance self-efficacy (confidence in personal ability to undertake a task) result in better overall outcomes<sup>46</sup>. Self-efficacy can be enhanced by approaches such as cognitive behavioural therapy (CBT, linking thoughts, feelings and behaviours) or social cognition (groups and peer role models) and include teaching patients skills such as problem-solving and goal-setting. Subsequently, a single RCT of group CBT that specifically aimed to reduce the impact of fatigue in RA has shown benefit, and this utilized high/low energy activity monitoring, pacing, sleep and stress management and assertiveness skills, using a goal-setting approach over 6 weeks<sup>47</sup>. However, the intervention was facilitated by a clinical psychologist, which most rheumatology departments do not have, therefore clinical teams need to find ways of developing skills to deliver interventions using these approaches. In Fibromyalgia, reviews showed no effects on fatigue from either CBT or acupuncture; whilst a short term effect was seen with educational/psychological interventions that included exercise<sup>48</sup>.

For exercise interventions alone, a meta-analysis in fibromyalgia showed a small effect on fatigue<sup>49</sup>. A Cochrane review of fibromyalgia exercise interventions in which fatigue was reported concluded that effects on fatigue were unknown. Meta-analysis of exercise interventions in RA showed small effects on fatigue, but none of the interventions had fatigue as their main aim<sup>45</sup>. Few exercise interventions have been performed in other musculoskeletal conditions that either specifically target or report fatigue, but single studies suggest exercise may be useful (eg in lupus)<sup>50</sup>.

## **7. Summary**

Pain and fatigue are significant and common symptoms in the Rheumatic diseases and commonly co-exist. The clinician should be cognoscente of these problems and regularly provide an opportunity for the patient to discuss these at routine appointments. Treatment strategies should be informed by clinical presentation and knowledge of mechanistic pathways. A multi-disciplinary approach to care is important for optimum outcome. Regular review of pain and fatigue throughout the disease trajectory is required to accommodate changes driven by disease activity and patient lifestyle.

**Key points:**

1. Pain and fatigue are the predominant problems for those with a rheumatic disease.
2. The approach to a patient with chronic musculoskeletal pain or fatigue should be holistic and multidisciplinary.
3. Many patients with chronic musculoskeletal conditions will not achieve a pain-free or fatigue-free status. Medication use must therefore balance potential benefit against short and long-term side effects.
4. Patients with chronic musculoskeletal pain or fatigue who continue to struggle with their symptoms should be offered support for behaviour change and adaptation, and/or referred to a Pain clinic, a fatigue specialist within their rheumatology department, or pain or fatigue management programmes.

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### **Further Reading**

Guidelines for the management of musculoskeletal pain, notably osteoarthritis, have been produced by several professional bodies including EULAR, ACR, APS and NICE<sup>27-35</sup>. A recent topical review on osteoarthritis provides a broad overview<sup>1a</sup>. Further information on the use of opioids in chronic pain is available from the British Pain Society<sup>2a</sup>.

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### **Sources of patient information**

There are several professional websites providing good information for patients with chronic musculoskeletal pain or fatigue. The following links to the British Pain Society and Arthritis Research UK provide some examples.

### **Opioid medications for chronic pain.**

Available at: [http://www.britishpainsociety.org/book\\_opioid\\_patient.pdf](http://www.britishpainsociety.org/book_opioid_patient.pdf) (accessed 27 November 2011).

### **Understanding chronic pain.**

Available at: [http://www.britishpainsociety.org/book\\_understanding\\_pain.pdf](http://www.britishpainsociety.org/book_understanding_pain.pdf) (accessed 27 November 2011).

### **Pain management programmes for adults: Information for patients**

Available at: [http://www.britishpainsociety.org/book\\_pmp\\_patients.pdf](http://www.britishpainsociety.org/book_pmp_patients.pdf) (accessed 27 November 2011).

### **Pain and arthritis**

Available at: <http://www.arthritisresearchuk.org/Files/2030-Pain-and-arthritis.pdf> (accessed 27 November 2011).

### **Fatigue and arthritis**



Available at: <http://www.arthritisresearchuk.org/Files/2030-Pain-and-arthritis.pdf>  
(accessed 27 November 2011). [note to editor, revised version in press]

### **Brief clinical vignette-Pain**

A 53 year old lady presents requesting 'stronger pain killers' for her 'rheumatic' pains. She has a past medical history of osteoarthritis (worst in the fingers, and hips) and lumbar spondylosis with an L4/5 disc prolapse and left L4 root impingement, obesity and angina. Her sciatic pain and arthritic pains are not adequately controlled using paracetamol on an 'as required' basis, with marked sleep disturbance.

### **Treatment options:**

#### **A. Non-Pharmacologic**

1. Physiotherapy review: She needs education and information on how to begin an exercise programme suitable for her needs. It will need to allow for her back problems and include advice on core stability. She is likely to have poor hip girdle strength which will also need addressing.
2. Weight reduction advice: complimented by a dieticians input if necessary.
3. Occupational therapy (OT): She has marked carpometacarpal (CMC) thumb osteoarthritis and reduced hand dexterity. OT can review her hand function, provide advice and exercises to improve dexterity, and thumb splints to support the CMC joints.
4. Review of aids and footwear: She is using one walking stick which is too short and with an uncomfortable handle for her osteoarthritic hands. Her shoes are fashionable rather than practical with moderate heels and tight over the forefoot compressing her 'bunions'. Standing barefoot, she has pes planus. A physiotherapy review will assess her walking aids and change the walking stick to a Fischer stick of the correct height

with a moulded handle suitable for her hand problems. She requires orthotic/podiatry review for advice on appropriate insoles and shoes. By making her feet more comfortable, her balance will improve and risk of falls reduce.

### **B. Pharmacologic**

1. Suggest use of Paracetamol 1g qds.
2. Add in use of topical NSAID gel, sparingly bd - tds for osteoarthritic fingers (1).
3. If topical NSAID gel is ineffective, consider topical capsaicin cream 0.025 – 0.075% (1)
4. In view of the history of ischaemic heart disease, avoid use of oral NSAID's.
5. Consider adding a weak opioid – substitute codeine / paracetamol combination for the paracetamol alone (1).
6. As she has neuropathic pain and sleep disturbance, consider use of amitriptyline 10mg nocte (2).

### **C. Pain management**

1. If the lumbar nerve root pain is most symptomatic and difficult to control with oral analgesics, consider a referral to a pain clinic. Targeted nerve root blocks combined with further neuromodulatory medications may be needed.
2. If despite optimisation of non-pharmacologic, pharmacologic and (where appropriate) interventional and surgical treatment, she continues to struggle to cope with her pain, she may be suitable for a pain management programme (2).

### **D. Surgical**

1. She may eventually need consideration for arthroplasty for the hip OA (1).
2. If she has completed an optimal package of care including a combined physical and psychological treatment programme and still has severe low back pain amenable to surgical intervention for which she would consider surgery, she may need referral to a spinal surgeon (2).

### **Brief Clinical Vignette - Fatigue**

A 35 year old woman has had rheumatoid arthritis for 10 years. She requests a review of her biologic medication because of fatigue and increasing pain. Whilst her fatigue has improved since starting biologic therapy the previous year, it has returned and is causing major impact.

### **Assessment**

On examination by the rheumatologist, no evidence of synovitis can be found and the patient's inflammatory indices are not raised. There is no obvious other cause for her fatigue (eg thyroid insufficiency, diabetes, significant anaemia). She is referred to a member of the rheumatology team who has developed an expertise in fatigue self-management (often a nurse or an occupational therapist). Their assessment begins with inviting the patient to describe the nature of the fatigue (probing for physical, cognitive and emotional elements) and its consequences. This process helps validate this invisible symptom, as the patient has experienced disbelief from others, leading to self-doubt about the validity of her fatigue. The patient is asked what she believes contributes to her fatigue (eg poor sleep, boom and bust behaviour, low mood) and how she has tried to manage it. Any indication of small successes in self-management (eg it's better if I don't do quite so much) gives the professional the opportunity to introduce the idea that improving self-management will be a useful way forward. Information on perceived causes, gives topics for the coming weekly sessions (eg sleep). .

### **Self-management support**

Daily activity monitoring of high and low energy activities, leisure activities and fatigue episodes (by colouring in an hourly chart) shows, at the end of the week, that the patient works long days with no breaks. As a consequence she spends Sundays in

bed, trying to recover and prepare for the week ahead. Discussion reveals that the patient believes she must continue to run the household perfectly in order to be a 'proper' mother to her three young children, and that not to do this would mean she had 'given in' to her arthritis. She forgoes all socializing in order to spend her energy on chores. The professional gently challenges her concept of 'being a proper mother' and helps the woman consider the difference between managing her arthritis fatigue by pacing, versus trying rigidly to 'not give in' and consequently being overwhelmed by fatigue. The patient is helped to make a realistic goal for the coming week around pacing and planning, with the reward of achieving something important to her (eg finding time to meet up with a friend). Over 3-4 weekly sessions, daily activity diaries and pacing strategies are reviewed, self-management of stress and sleep are tackled, with further weekly goals being agreed. Mood is discussed and if appropriate the General Practitioner is asked to provide relevant support. The patient is referred to physiotherapy for graded exercises to help her work toward her rewarding goal of getting back to the gym or go swimming. She is referred to the Occupational Therapist for help with reducing some of the effort needed for activities of daily living. Where possible the team's intervention would have been delivered in a group setting, as the use of peers as role models is particularly effective in enhancing self-efficacy. Approximately one month after the end of the fatigue intervention, a final appointment to consolidate the skills learnt to deal with fatigue (and recurrent setbacks) is helpful.