

# Pain Neuroscience and the Role of Exercise

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350 years ago, French philosopher/scientist Rene Descartes put forward what became known as the Cartesian model of pain, which contained the basic truth that nerves send messages to the brain, which interprets the messages as pain. Over the years there has been a range of definitions of pain, but all recognise that pain is an output of the brain 100% of the time.

According to Australian pain specialist Lorimer Moseley, "The pain system is activated when the brain concludes that the body tissues are in danger and action is required." Pain messages are however not a sufficient measurement of pain as the degree of pain felt by the patient depends on the brain's evaluation of the pain signals it receives. This evaluation will depend on elements such as genetics, meaning ascribed to pain by the individual, unconscious thoughts, previous experience, learning, or spinal cord and brain problems.

There could for example be a far greater pain response to a sudden pain in the back if the patient has previously suffered back pain and thought it cured. While some people's brains may interpret a particular pain as not dangerous enough to take action, others may interpret it as extremely dangerous, and will consequently feel more severe pain.

Pain itself therefore is not good at providing an accurate indication of tissue state, but it is a behaviour modifier, in that it makes us take action to get out of a situation that the brain interprets as a threat.

Coping with chronic pain calls for brain responses very different to the pain responses our brains may use for injury pain, but these responses are less instinctive and therefore our brains need to be trained to interpret them in a way that enables us to live a good life despite our arthritis.

- With chronic pain, it is vital to remember:
  - Chronic pain can be managed, but not cured
  - 'Hurting' doesn't necessarily mean 'harm'
  - There are physiological reasons for your pain
  - It does not imply tissue damage is happening
  - Things you do that wouldn't otherwise be regarded as painful can cause you pain when you are already in pain
  - An increase in pain sensation does NOT mean there is new or more damage happening.

## Pain and Disability

There is a relationship between pain and disability but the relationship is variable depending on the degree of pain felt by the individual. Disability however robs people of their most significant life experiences. It is not enough to focus on the physical impairment and pain – we must also focus on preventing disability.

## Pain and Osteoarthritis

Like many other pain conditions, OA is associated with numerous misconceptions and erroneous beliefs about both causes and effective management. OA is however not a linear condition caused strictly by degeneration as it was regarded in the past. It is now known to be a specific disease with a complex pathophysiology including neuropathic peripheral and central abnormalities in addition to local inflammation.

However, patients with objectively determined degrees of damage report very different amounts of pain. Some people with tissue damage will report no pain at all, as seen in a research project in the 1990s where 60% of grade 2 – 3 radiologically diagnosed OA joints, and 40% of grade 4 OA joints, were painless. At the other end of the pain spectrum, it was found that surgical procedures to cut neural pathways to the painful area may fail to remove the pain.

## Gate control theory

In 1965 Melzack and Wall introduced what they called the gate theory, which introduced the concept of psycho-physical interactions by recognising that pain is not a simple one-way system from the periphery to the central nervous system. They believed that while the transmission of pain is affected by the intensity of the stimulus, it is also affected by competing stimuli and impulses coming from the brain. Their contribution to the study of chronic pain was to show that i) the brain is not a passive receiver of pain impulses, but ii) an action system which influences our experience of, and behaviours towards, pain.

This theory however, still inadequately explains multiple interactions which occur in our perception of, and responses to, pain. More recently, Prof Gifford in 1998 put forward the bio-psycho-social model, called the Mature Organism Model. It attempts to incorporate the interaction of biological variables, psychological processes and the social context of pain into how we experience it.

This model regards input from somatic, visceral nociceptive or peripheral neurogenic sources as either inflammatory or mechanical. We then process it together with our thoughts/emotions/possible catastrophic thinking, fear, anxiety and depression, from which our brains interpret the pain and direct the next course of action. This may be to protect the injured part. There may be excessive guarding, inappropriate reluctance to move, taking all kinds of medications, doctor shopping and so forth, and there may also be autonomic responses; hormonal changes and an immune system reaction in the body.

Allodynia is a condition where patients with OA feel pain in response to stimuli which don't normally induce pain. Another aspect of chronic pain e.g. knee OA, is that while the body is in pain, there is a decrease in the volume of grey matter in the brain. The good news is that this is not permanent – the grey matter will regenerate within six to nine months of successful surgery. An explanation for the decrease may be that the body is attempting to reduce the brain area where pain can be felt, and once the pain is gone, it is safe to regenerate the lost brain matter.

## Assessing OA joint damage

### X-RAYS

There is only a modest correlation between pain intensity and the degree of joint degeneration. Pain appears to be more closely related to the narrowing of the joint space than to the presence of bony outgrowths called osteophytes. Indeed, pain intensity is a more significant predictor than joint damage for joint replacement in knee OA.

## MRI SCANS

Synovitis and bone marrow oedema are more closely correlated to pain in OA than osteophytes, bone cysts, meniscal changes or ligament tears. In patients with equal radiographic grades of knee OA, synovitis was shown to be the most important predictor of pain, and in most cases the pain can be controlled with corticosteroid injections.

## INFLUENCE OF GENETIC, GENDER AND OTHER FACTORS ON PAIN

- Women experience 60% higher pain than men regardless of the degree of degeneration.
- While exercise may increase pain in the short term, it is vital to keep the joints moving, therefore the type and amount of exercise is important.
- Mood, weather conditions, even genetic factors influence pain perception.
- Age and obesity are significant factors for developing OA.
- Weight loss programmes are essential to relieve pain.
- The reduction in pain is related to modification in the pain pathways and not only to mechanical joint modifications.

## EXERCISE AND PHYSICAL ACTIVITY

Although the clinical guidelines for the treatment of OA, chronic lower back pain, fibromyalgia etc. are often conflicting, the one treatment modality common to them all is exercise and physical activity. Nonetheless despite slogans such as 'exercise is medicine', and 'sitting is the new smoking', many therapists struggle to implement exercise programmes for chronic pain into their practice due to the barriers that exist such as patient, environment and healthcare delivery factors.

## Barriers to exercise for patients with chronic pain

- Patient factors
  - Fear of pain
  - Dysfunctional endogenous pain modulation
  - Strong belief that exercise will cause harm
  - Deconditioning
  - Lack of understanding of the neurophysiology of pain
  - Depression
  - Lack of self-efficacy
  - Environmental factors
  - Lack of access to a place to exercise
  - Perceived or real lack of time
  - Lack of support from the family
  - Healthcare delivery factors
- Overly strong focus on the biomedical model of pain
  - Lack of attention to psychological and CNS/brain contributions to pain
  - Poor communication between patient and healthcare providers on the value and importance of exercise
  - Poor understanding by the patient about the meaning of pain
  - Lack of supervision to enable the patient to feel safe about what exercises to do.

## Hyperalgesia and hypoalgesia in chronic pain

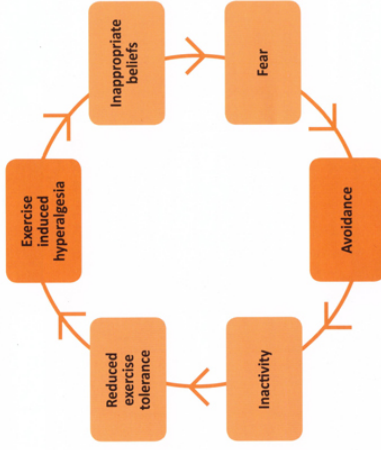
In healthy participants, exercise will always mean reduced pain, while in chronic pain conditions, patients may develop

hyperalgesia or hypoalgesia. For Fibromyalgia patients, aerobic and isometric exercises mean less pain and higher pain thresholds. Conditions characterised by pain hypersensitivity mean that the patient's pain inhibition is not working properly. There are many more chronic pain conditions that are not dominated by central sensitisation like RA, OA or chronic shoulder pain. These are mostly nociceptive or inflammatory pain.

Hyperalgesia to exercise plus a poor understanding of pain may well result in inappropriate pain perceptions and beliefs. Patients may not be aware that pain is often disproportionate to the amount of tissue damage, and they will become less tolerant of exercise through fear of movement, thinking it will cause harm. Unless exercise is tailored to the individual needs and beliefs of each patient, it will fail.

## Assessing the individual patient

Before introducing an exercise programme, try to provide the patient with some neuroscience education, then explain that although exercise might be painful in the short term it will not cause further damage. Make sure the patient sees exercise as a therapy tool to retrain their faulty pain inhibition mechanism.



## Tailoring the exercise programme

Combine exercises to counter the short-term pain increase e.g. specific strength training together with general fitness. Aim to train the brain along with the rest of the body.

## Facilitating adherence to the programme

Come to an agreement with the patient about their self-efficacy i.e. the extent or strength of one's belief in one's own ability to adhere to a programme, complete set tasks and reach agreed goals. The programme should be time-based rather than based on the patient's symptoms.

In summary then, physical activity is absolutely vital for patients with chronic joint pain. Its treatment requires seeing the patient in a broader bio-psycho-social framework and doing a thorough assessment of the patient on that basis. There will be barriers to success but – like that the goat and the million kilowatt dam – if therapist and patient work together with honesty and trust, these barriers will be overcome.