

Piercing the puzzle of persistent pain

Definition

Pain has been defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”

Three phases of pain

- 1) Immediate - This is fleeting and protective e.g. a slap on the face or a pinprick.
- 2) Inflammatory - an injury causes substances (bradykinins, histamine, prostaglandins) to be released locally. These activate nociceptors. For example, an acutely sprained ankle is hot, sore, red and swollen.
- 3) Chronic - Pain persists in the absence of any inflammation or permanent injury.

Dealing with inflammatory pain is relatively easy for the health care community but it is chronic pain that has previously been challenging. Research in a variety of fields (neuroscience, cellular biology, education, physiotherapy etc.) has significantly increased our understanding of persistent pain.

Chronic pain

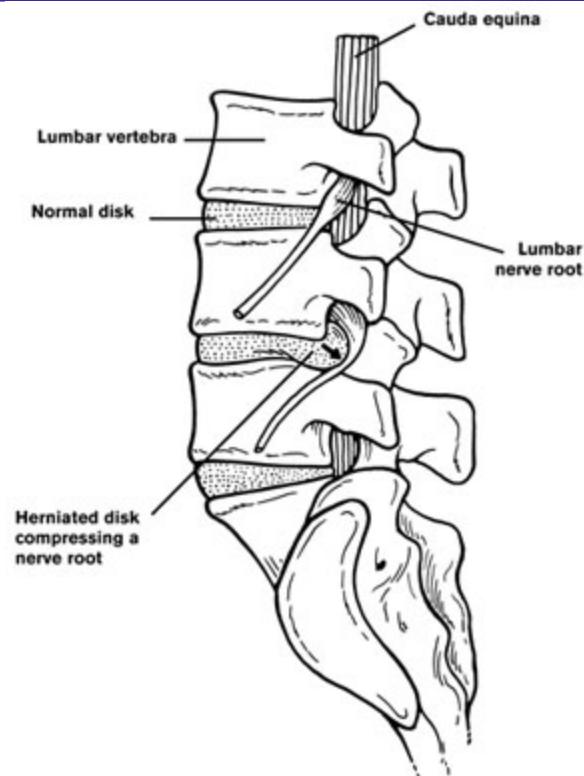
Chronic pain is also known as neuropathic pain. Neuropathic pain occurs in:

- 1) ongoing inflammation e.g. rheumatoid arthritis
- 2) psychiatric disorders e.g. clinical depression
- 3) a dysfunctional nervous system (altered function and/or structure)

Various assessments can aid the diagnosis of the first two causes of neuropathic pain. This leaflet will concentrate on the third cause of neuropathic pain – a dysfunctional nervous system.

Why is my nervous system not working properly?

The central nervous system (brain and spinal cord) is well protected by the skull and spine. The peripheral nervous system is, however, not so well protected. It is thus more susceptible to damage especially at the junction between the two systems – an area where the nerve exits the spinal cord, known as the nerve root (picture 1).



from www.dana.org

Picture 1: side view in the lower back disc and nerve root

Many things can damage the nerve root including trauma, infection, cancer etc., but the most common cause is changes in the shape and structure of the intervertebral disc and its surrounding structures.

Why does the disc change shape and structure?

Degeneration of the intervertebral disc and its surrounding structures (known as spondylosis) is part and parcel of life. We are all slowly but surely degenerating. Age related degeneration normally proceeds imperceptibly, often, with no symptoms. Certain factors can speed up the degeneration process such as an unhealthy lifestyle (smoking and no exercise), major accidents, surgery, poor posture etc.

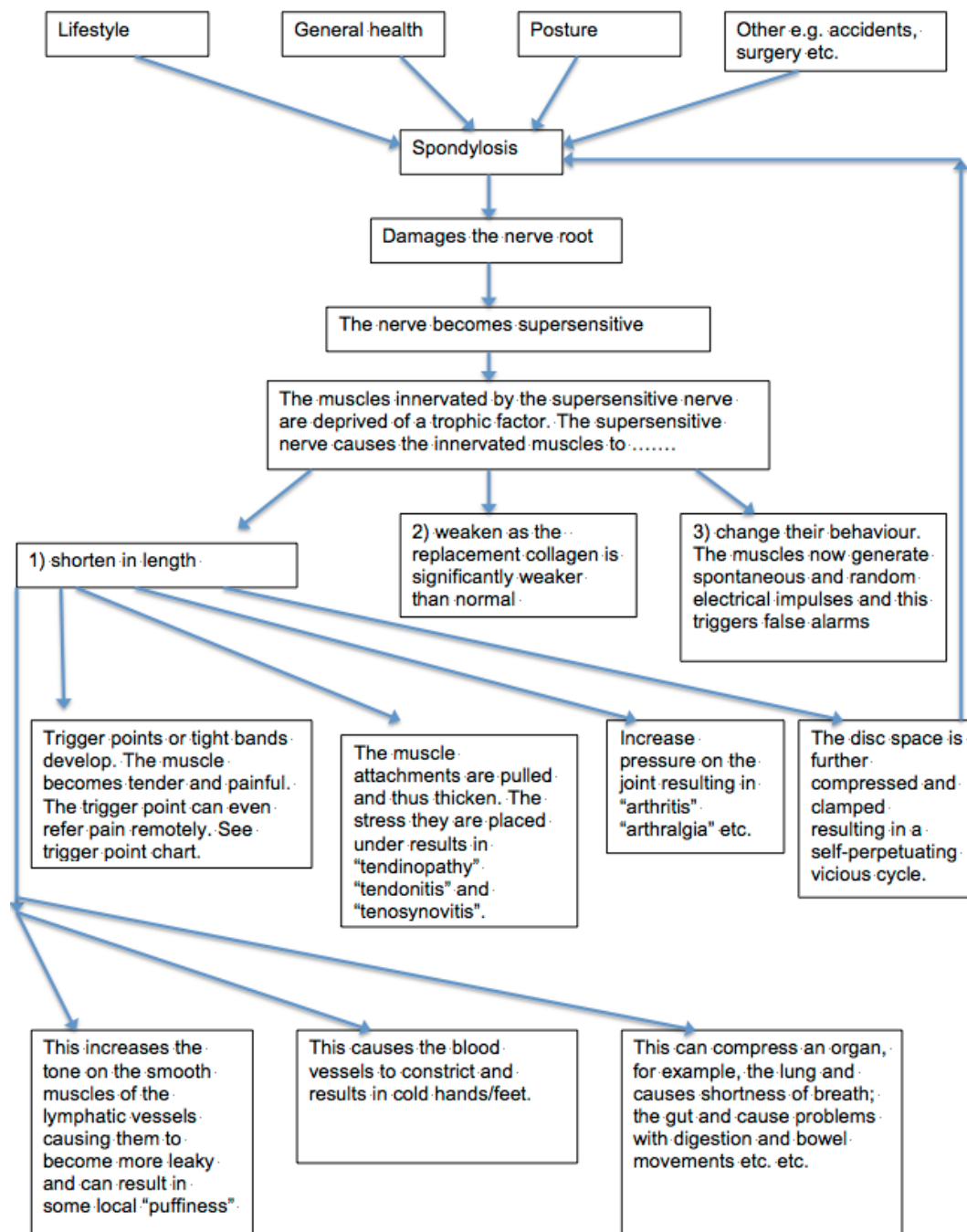
How does spondylosis cause pain?

Degenerative changes to the intervertebral disc and its surrounding tissues (decreased disc height, osteophyte formation, ligament thickening etc.) can put pressure on the nerve root. This pressure on the nerve root may or may not cause you pain. But the pressure on the nerve root does cause the nerve to become supersensitive.

When the nerve becomes supersensitive, it starts to not function properly and to behave erratically. The supersensitive nerve becomes super reactive and hyper excitable to any stimulus and its response lasts for an unusually long

time. When the nerve is supersensitive, the structures (muscles, skin, bone etc.) that it supplies become deprived of a trophic factor. Supersensitive nerves tend to have the worse impact on muscles.

The flowchart below illustrates the effect the supersensitive nerve has on the muscle.



Flowchart 1: How pain develops and can persist

As we go through life, many of our episodic minor aches and pains and twinges can pass by unnoticed but do damage the once healthy nerve root. As we age we accumulate all these episodic aches and pains into our “injury pool”. The unresolved residue of these episodic aches and pains makes the now damaged nerve root even more susceptible to future injury. And this explains why some people develop severe pain after an apparently minor injury or even no event, and why the pain persists beyond a “reasonable” period of time.

So my supersensitive nerve is causing all my pain?

For the most part, yes but we must remember that pain is complex and the brain is also involved. The leaflet called “pain explained” will give you more information in this regard.

But to keep it simple, a helpful analogy may be that of comparing your body’s nervous system to the alarm system of a house. The sensors (receptors) on the windows (skin, muscle, ligament, tendon, joint etc.) normally only respond if the window is smashed (strain/sprain/tear). In persistent pain, the window sensors become so sensitive, that even a light breeze (gentle touch) triggers the alarm off (threat of supposed damage) and the police (pain) arrive.

So the problem is not only in the tissues (muscles, ligaments, joint etc.) but also in the danger messengers that have become sensitive and provide the brain with inaccurate and exaggerated information. The brain then concludes that a threat is still prevalent and then constructs and produces pain in order to get you out of danger and protect/save you.

Ok I now understand that my nervous system is dysfunctional and causes my pain to persist but what can be done about it?

A comprehensive assessment can:

- Detect the presence of on-going inflammation/infection (red flags)
- Detect the presence of a psychiatric problem (orange flags)
- Identify any psychosocial contributors (yellow, blue and black flags)
- Identify factors that are contributing to the spondylosis
- Determine the most appropriate course of action

A multidisciplinary approach is helpful in addressing the various contributing factors and treatment may consist of:

- Education
- Relaxation and visualisation
- Graded motor imagery
- Re-education of a healthy breathing pattern
- Acupuncture
- Graded exercise

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- Cognitive-behavioural therapy
 - Ergonomics
 - Postural correction
 - Re-education of healthy movement patterns
 - Medication

Please chat to your healthcare professional about the options that are appropriate for you.

References

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Gunn, C. (1996) *The Gunn approach to the treatment of chronic pain: intramuscular stimulation for myofascial pain of radiculopathic origin* (2nd ed.). Churchill Livingstone: London.