Pain physiological mechanisms used in physiotherapy

By Inger Skjærbæk

In his introduction to the Textbook of Pain, Patrick Wall writes:

> "So long as one person remains in pain and we cannot help, our knowledge of pain remains inadequate",

which is very true. No doubt there is still a lot we do not know about pain.

Definition of pain

When in 1979 the Association for the study of pain defined pain as:

> "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage",

they emphasized the subjectivity and the emotional feeling behind pain, and at the same time they recognized the existence of pain even in the absence of a detectable physical cause.

It is therefore important for us who treat patients in pain to realize and accept that pain, the intensity of pain, and the effect of our treatment can be perceived only by the patients themselves.

Pain physiology

The perception of pain is either effected by the nociceptors situated in our tissues, through the Aδ and C fibres, respectively, as a noxious signal extrinsic to the nervous tissue; or it can arise as a result of functional with/without structural disturbances inside the nervous tissue itself. It can also be a combination of the two.

Approximately 70% of all axons within a nerve are nociceptive. The C and Aδ fibres differ fundamentally from each other in various ways:

The *C fibres* are morphologically old; they terminate in the limbic system (see below), where they affect the mood. They are morphine modulated, many in number, medially situated, multisynaptic and slow, 2-3 m/sec. They transmit the dull, aching, burning, and poorly localized pain, and are mainly responsible for chronic pain.

The *Aδ fibres* are morphologically young, few in number, and externally situated. With only 3 synapses, the consequence will be that they transmit the sharp, well localized and mainly acute pain fast, 5-10 m/sec.

Pain modulation

The perception of pain can be influenced because the tolerance of pain can be increased or decreased (fig. 1). The sum of aggravating/modifying factors will determine the severity of the pain that is being felt at a given moment.

The pain modulatory mechanisms are of special interest to us. The limbic system plays an important role in this context.

The limbic system includes the morphologically oldest parts of the brain (fig. 2). They are in close connection with each other and are essential for some typical behaviour patterns in all species of living beings, such as:

1. emotional conditions, which bridge Psyche with Soma, e.g. joy, fear, grief, anger, and depression
2. learning behaviour/memory, learn from experience
3. integration of homeostatic mechanisms related to:
   a. hunger/thirst
   b. fight/flight
   c. sexual behaviour
   d. regulation of muscle tone
   e. regulation of temperature
   f. sleep
4. motivation
5. pain tolerance, how to cope with pain
6. fragrance.

This is important knowledge regarding our approach to and treatment of the patients. We can actually influence the pain tolerance, the perception of pain, and motivate the patients deliberately by knowing this. Besides, it gives us a better understanding regarding some of our clients' behaviour – aggressive, frightened, depressive – and why some fragrances can provoke flash backs.

Increasing pain tolerance	*Decreasing pain tolerance*
information	uncertainty
security/confidence	worry
comprehension/sympathy	boredom
hope	anxiety/anger
rest/sleep	fatigue/insomnia
being close to the family	loneliness
touch	depression
heat	being cold
light, sounds, fragrances	light, sounds, fragrances
Endogenous opioids	
Stimulation-produced analgesia	
massage, mobilisation	
myofascial release techniques	
muscle energy techniques	
tens	
acupuncture/intra-muscular stimulation	

Fig. 1. *Factors influencing the pain tolerance.*

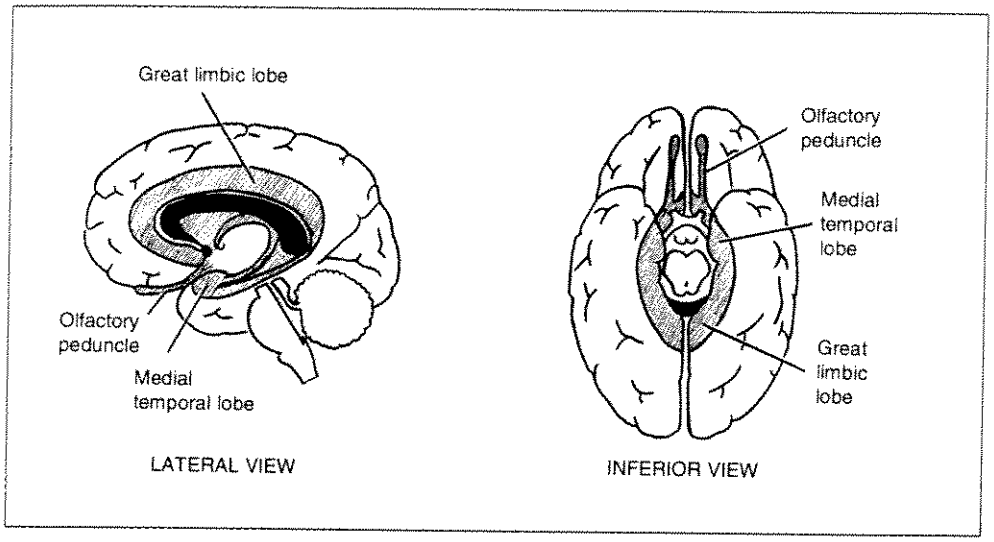

Fig. 2. *Schematic illustration of the location of the limbic system.*

The presence of *endogenous opioids* (fig. 1) is believed to function in two ways, one of which is in the limbic system itself, preventing the information of pain from gaining access into the limbic structures. The other one is a descending pain suppression system, inhibiting pain at the spinal level.

The theory of the *stimulation produced analgesia* (fig. 1) lies within the gate control theory, and for some treatment modalities in the above-mentioned opiate analgesia as well. Modalities which cannot be used in the treatment of torture survivors, not in the beginning anyway, and especially not if the torture given was electrical, because these modalities have to provoke some pain/discomfort to stimulate the production of the endogenous opioids.

When, at a later stage, security and confidence has been created, and the torture survivor has been informed and motivated, it might be possible to use these modalities in some cases.

Patrick Wall was the first to describe 3 sequential types of pain, each with its own characteristics and treatment. Each phase may exist independently, or in any combination and proportion with the others.

1. nociception: the acute pain, with/without tissue damage.
2. inflammation: the vital response of tissues to an injury or infection, normally as self-limiting process.
3. chronic pain: persisting, ongoing pain.

The reactions to acute and chronic pain are totally different in the individual (fig. 3).

Chronic pain

In the true sense of the word, chronic pain is a misnomer. I do not think any one will claim that a patient with an ongoing inflammation, e.g. rheumatoid arthritis, is not suffering chronic pain.

The terms radiculopathy/neuropathy, as Chan Gunn suggests, are much more appropriate, if only the original definitions are used, namely: Altered function with/without altered structure in a nerve root/peripheral nerve, respectively.

In other words, the reasons for the pain are to be found inside the nervous tissue itself.

The brain thinks, gets the impression of a damaged tissue, and reacts accordingly with pain. One could say that it is a hidden problem because for a long period there is no detectable physical cause of tissue damage – no neurological deficits. Sometimes the patients complain of pins and needles, but then we know that they have ischaemia in the nerves supplying that area from time to time.

The tissue has become hypersensitive to a quite normal stimulus, and words like psychosomatic and malingering may arise, which is rather unfortunate.

Two factors are important in the comprehension of pain arising as a result of intrinsic reasons:

1. nourishment of nerve cells and their target tissues
2. denervation-/disuse supersensitivity.

Nourishment

The nervous tissue is probably the most oxygen-demanding tissue we have. Normal functions, for instance conduction of impulses, metabolism, and production of endogenous opiates and trophic factor, depend on this supply.

Ischaemia in a nerve will reduce these vital functions, but not equally in all axons; the thickest myelinated fibres, which in fact supply our muscles with impulses and trophic factor, are those which suffer first, having the highest oxygen demand of them all.

"Acute"	*"Chronic"/persistent*
increased pulse rate	sleep disturbances
increased cardiac stroke volume	irritability
increased blood pressure	decreased/increased appetite
pupillary dilatation	weakened psychomotorics
increased sweating	decreased pain tolerance
hyperventilation	social withdrawal
hypermobility/incr. tension	abnormal illness behaviour
escape behaviour	(masked) depression
anxiety	
vocalisation	

Fig. 3. *Reactions to pain.*

How do we get ischaemia in nerves?

Chan Gunn draws our attention to prespondylosis, and direct compression of the spinal nerves in the intervertebral foraminae, whilst Sunderland claims that indirect pressure on the nerves in the tunnels, foramina being but one type, is a much greater threat to the axons.

In order that the blood can flow uninterruptedly through the vascular system in nerves, a downward pressure gradient must be maintained within the tunnels. The greatest pressure must be in the epineurial arterioles and then progressively less in the endoneurial capillary, the fascicles, epineurial venules and least in the tunnel itself.

If, for instance, the tunnel pressure rises above the pressure in the venules, these cannot drain the blood from the endoneurial space, and oedema, ischaemia, and eventually inflammation result. At a later stage irreversible fibrosis and thus structural changes will appear within the roots/nerves.

Long before this stage, the target tissues have become denervation-/disuse supersensitive due to the altered function.

Denervation-/disuse supersensitivity

If biological tissues, e.g. muscles, are deprived of/get reduced amounts of trophic factor, they atrophy and become hypersensitive, according to Cannon & Rosenbluth's law of denervation. Furthermore, these muscles will over-react to many chemical and physical inputs, including stretch and pressure.

Clinical manifestations

Clinical manifestations include all our neuromusculo-skeletal structures. The musculo-skeletal components are probably the most noticeable and traditionally the ones treated by physiotherapists, but more recently our attention has been drawn towards adverse neural tensions within the connective tissue of the nervous system.

Motor component. The first step might be muscular imbalance, as defined by Vladimir Janda. Muscle spasm and shortening due to the supersensitivity will put stress on the soft tissue attachments, and myofascial pain syndromes such as tendonitis, epicondylitis, tendovaginitis and so forth will develop. Active and latent trigger points, as defined by Travell and Simons, will be part of the problems. Bursitis and arthrosis are also well-known diagnostic entities.

Neural component. As mentioned above, some of the consequences of direct/indirect pressure on nerve roots and nerves are oedema and inflammation. The clinical reaction to this is that the connective tissues with relation to the nervous system will begin to show signs of decreased mobility and extensibility – adverse neural tensions. These changes can be found on test movement and thereafter treated.

The neural component will gradually show autonomic manifestations as well; the involved areas will be colder, showing goose bumps and decreased sweating (vaso-, pilo- and sudomotoric changes) together with signs of trophoedema due to dysfunction in the lymphatic vessels.

Treatment

One of the main messages in the treatment of these syndromes is not to fall into the ditch of treating the peripheral structures only. Rather, address the treatment to the spinal column and adverse neural tensions as well.

We should always remember these syndromes as being results of altered function without/with altered structures of either roots and/or nerves.

As Chan Gunn puts it: "It is a pity we call an elbow for an elbow and a foot a foot, when in fact they are extensions from the neck and low back, respectively."

References

Afifi, AD, 1986, Basic neuroscience, page 312-313: The limbic system, Urban & Schwarzenberg.

Butler, David, 1991, Mobilisation of the nervous tissue, Churchill Livingstone.

deGroot/Chusid, 1988, Correlative Neuroanatomy, Chap. 14: The limbic system, Appleton & Lange.

Gunn, C, January 1978, "Pre-spondylosis" and some pain syndromes following denervation supersensitivity, Annual Meeting 27 January 1978, Royal College of physic & Surg.

Gunn, C, 1984, Pain and misperception in neuropathways or type three pain, Proceedings 5th IFOMT conference, Vancouver, pp. 192-201.

Gunn, C, 1989, Treating Myofascial Pain, ISBN: 1-55910-003-6.

Gunn, C, 1989, Neuropathic Pain: A new theory for chronic Pain of intrinsic origin, Annals of the Royal College of physicians and surgeons of Canada, vol 22, No. 5.327-30.

Gunn, C, 1990, Mechanical Manifestations of neuropathic Pain, Annals of sports Medicine 5: 138-141.

Janda, Vladimir, 1986, Muscle weakness and inhibition (pseudoparesis) in back pain syndromes chapt. 19 in Modern manual therapy, ed. Greg. Grieve, Churchill Livingstone.

Lundborg, G, 1988, Nerve injury and repair, Churchill Livingstone

Melzack/Wall, 1991, The challenge of pain, ISBN: 0 14 01.5660 7, Penguin book.

Sternbach, R, 1984, Acute versus Chronic pain, Chapt. 1.14. Textbook of Pain, ed. Melzack & Wall, Churchill Livingstone.

Stratton, SA, 1982, Role of endorphins in pain modulation, JOSPT 0196-60011/82, pp. 200-205.

Sunderland, S, 1980, The anatomy of the intervertebral foramen and the mechanisms of compression and stretch of nerve roots, Modern Developments in the Princ. & Pract. of Chiropract., ed. S. Haldeman. ACC.

Travell & Simons, 1983, Myofascial Pain and dysfunction, The Trigger Point Manual, Williams & Wilkins.

Watson, J., 1986, Pain and nociception – Mechanisms and modulation, chapt. 21 in Modern Manual therapy, ed. Greg. Grieve, Churchill Livingstone.

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