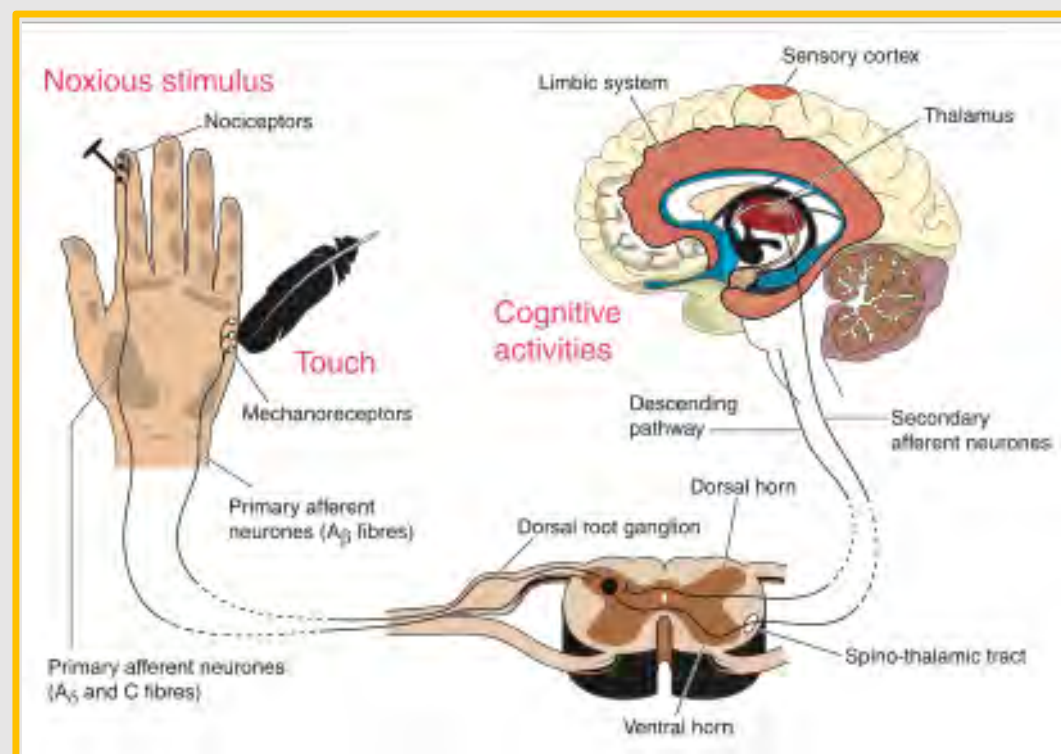


Understanding Pain And The Scientific Osteopathic Approach Of Pain



The same author also published books on:

- Cranial Nerve Disorders and the Scientific Osteopathic Approach
- Fascial Chains
- Nutrition and Physical Complaints
- Posturology and its Scientific Osteopathic Approach
- Scientific Osteopathic Approach to Patients with Abdominal Complaints
- Scientific Osteopathic Approach to Patients with Cervical Pain
- Scientific Osteopathic Approach to Patients with Headache
- Scientific Osteopathic Approach to Patients with Knee or Foot Pain
- Scientific Osteopathic Approach to Patients with Low Back Pain
- Scientific Osteopathic Approach to Patients with Shoulder, Elbow, Wrist or Hand Pain
- Scientific Osteopathic Approach to the Immune System
- Scientific Osteopathic Approach to Vascularization and Oxygen Supply in Patients
- Understanding Pain and the Scientific Osteopathic Approach of Pain
- Understanding Stress and the Scientific Osteopathic Approach of Stress
- Understanding the Autonomic Nervous System and its Scientific Osteopathic Approach
- Perimenopausal Women and their Complaints
- Cerebrospinal Fluid and its Influence on Health
- Attention Deficit Disorder / Hyperactivity and the Scientific Osteopathic Approach
- Principles of Modern Osteopathy – Integration of Osteopathy into General Healthcare
- Evidence Based Practice
- Patient Information – What can Osteopathy do for You?

All rights reserved: Luc Peeters © 2022

No part of this book may be reproduced or made public by printing, photocopying, microfilming, or by any means without the prior written permission of the author.

Contact: Luc Peeters

Mail: info@osteopathybooks.com

Pain

Since most patients that consult osteopaths come with pain; we have to know and stay UpToDate of the most recent knowledge of pain mechanisms.

Osteopaths first look for the structure(s) that cause/produce the pain in the patient. Then they try to determine the pain generator because this will give us a good idea on what we want to achieve (Goals) in our osteopathic treatment.

1. Definition of Pain

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (*Ref.: International Association for the Study of Pain - IASP*).

It is a positive warning signal and an imperative protective reflex.

The word 'Pain' is derived from the Latin word 'Poena' which means penalty/punishment from God.

2. Types of Pain

- Acute and chronic pain.
- Nociceptive and neuropathic pain.
- Somatic and visceral pain.
- Referred pain and non-referred pain.
- Somatogenic and psychogenic pain.

When we ask a patient about his/her pain, we ask for:

- **P** attern: onset and duration.
- **A** rea: location of the pain.
- **I** ntensity of the pain.
- **N** ature: description of the pain.

2.4. Referred Pain

Pain experienced at a place distant from the origin.

The area of the pain is supplied by the same spinal segment as the actual pain location.

Dermatomal rule: example: the heart and the arm have the same segmental origin.

Convergence theory: the number of peripheral pain impulses exceed the number of lateral spinothalamic fibers. This means that both somatic and visceral afferent convergence upon the same spinothalamic tract neurons, the individual gets the feeling that the pain originates from the somatic area.

Facilitation theory: visceral and somatic pain afferents connect with adjoining spinothalamic neurons and there may be some overlap of the neurons, visceral afferents have collaterals connecting to the spinothalamic neurons receiving somatic pain afferents. This can cause impulses to travel up the somatic spinothalamic path and causes the sensation of pain in the skin.

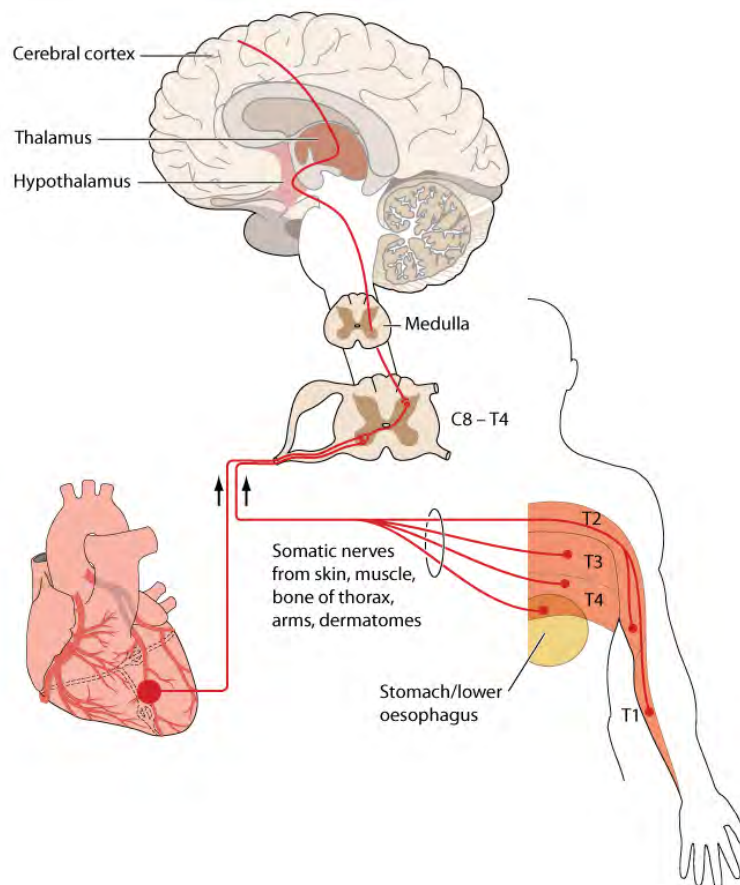


Figure 1 - Example of referred pain

3. Neurology of Pain

There is:

- **Reception.**
- **Transduction** (Stimulus is translated into electrical activity at the sensory nerve endings. This stimulus then sends an impulse across a peripheral nerve fiber).
- **Transmission** (Propagation along Delta and C fibers. The pain stimulus then travels up the spinothalamic tracts).
- **Modulation** (Modification of the transmission by for example inhibitory neurotransmitters).
- **Perception** (Awareness of pain. The somatosensory cortex identifies the location of the pain as well as the intensity).

3.1. Reception

Nociceptors are endings (Free nerve endings) of small mostly unmyelinated afferent neurons.

Stimulators for these nociceptors are:

- Mechanical (Damage, tearing, stretching, crushing) stimuli.
- Chemical (Inflammation, infection, ischemia, lactic acid) stimuli.
- Thermal (Heat or freeze) stimuli.

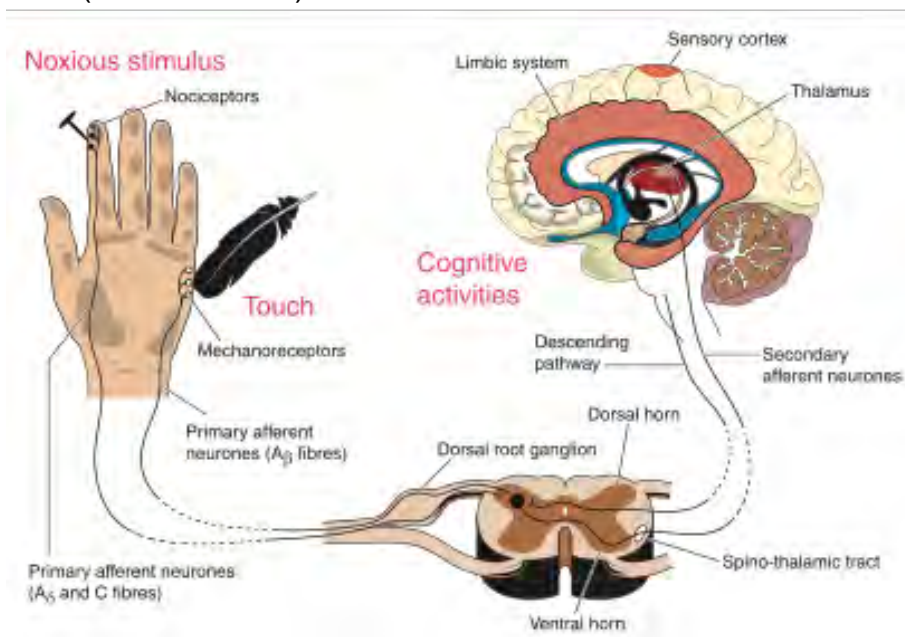


Figure 2 - Nociceptors

- Histamine (Increases the permeability of the capillaries to white blood cells and some proteins to allow them to engage pathogens in infected tissues).
- Acetylcholine (Neurotransmitter).
- Substance P (Peptide, neurotransmitter, mostly of type C nerve endings, vasodillator, inflammator).
- Potassium(K) ions (Mineral, cell communication).
- Glutamate gives fast pain sensation.

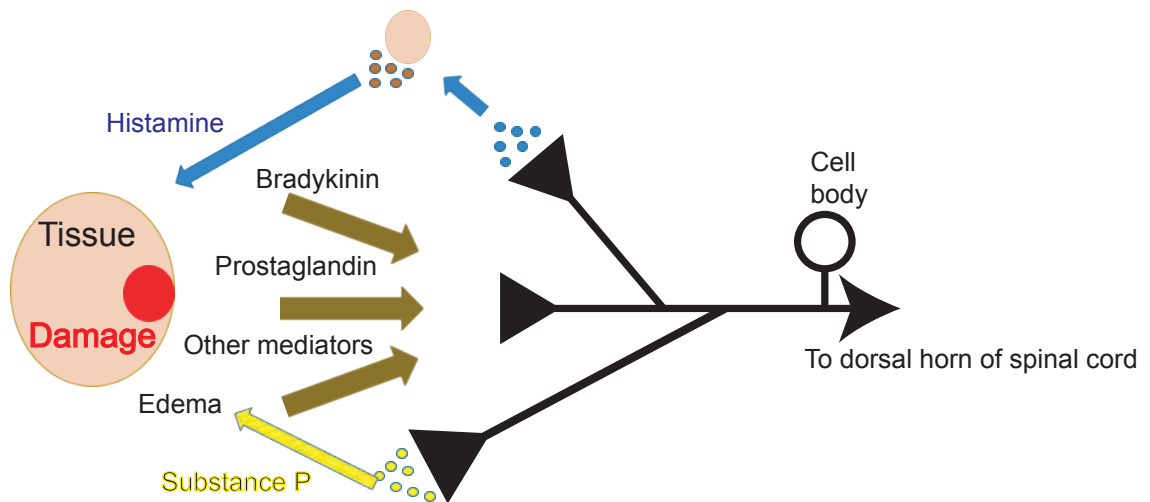


Figure 3 - Molecules that produce pain

3.3. Transmission

3.3.1. Transmembrane Receptors

(Also called ion transporters or ion pump proteins)

- Are present in the cell membranes.
- They transmit the stimulus from extracellular to intracellular.
- They send the information to the brain through 3 types of fibers:
 - A-beta-fibers
 - A-delta-fibers
 - C-fibers
- Paracetamol for example blocks these receptors.

Adelta fibers	C-fibers
Myelinated (Fat and protein surroundings)	Non-myelinated
Fast conduction (5-35m/sec)	Slow conduction (0.5-2 m/sec), delayed onset
Large diameter	Small diameter

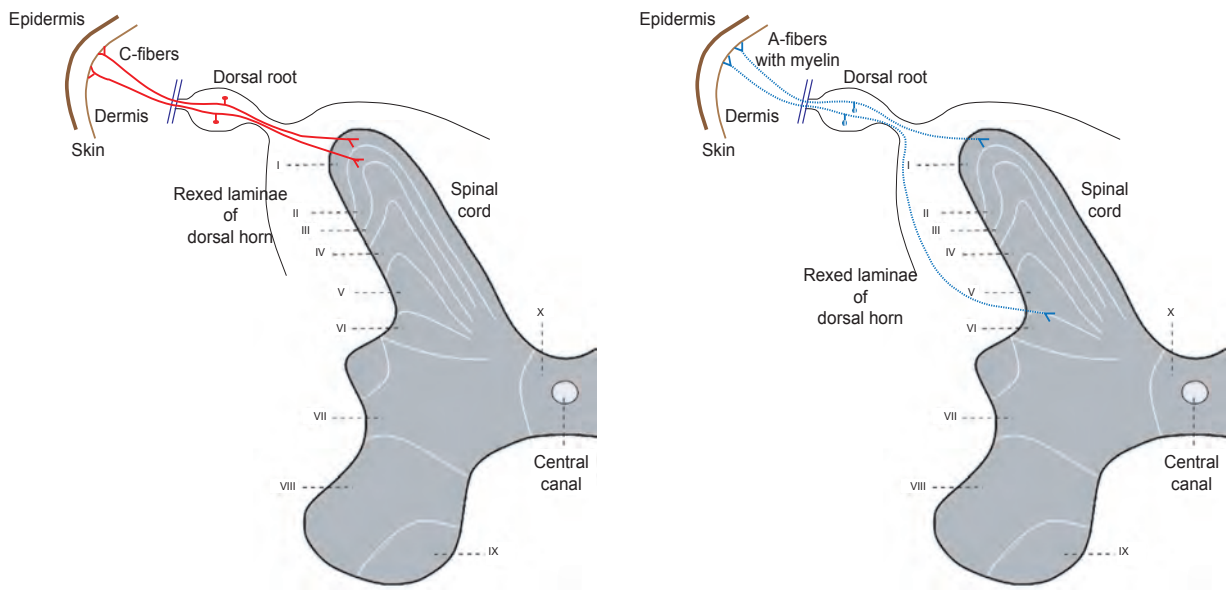


Figure 6 - Connection to the rexed laminae

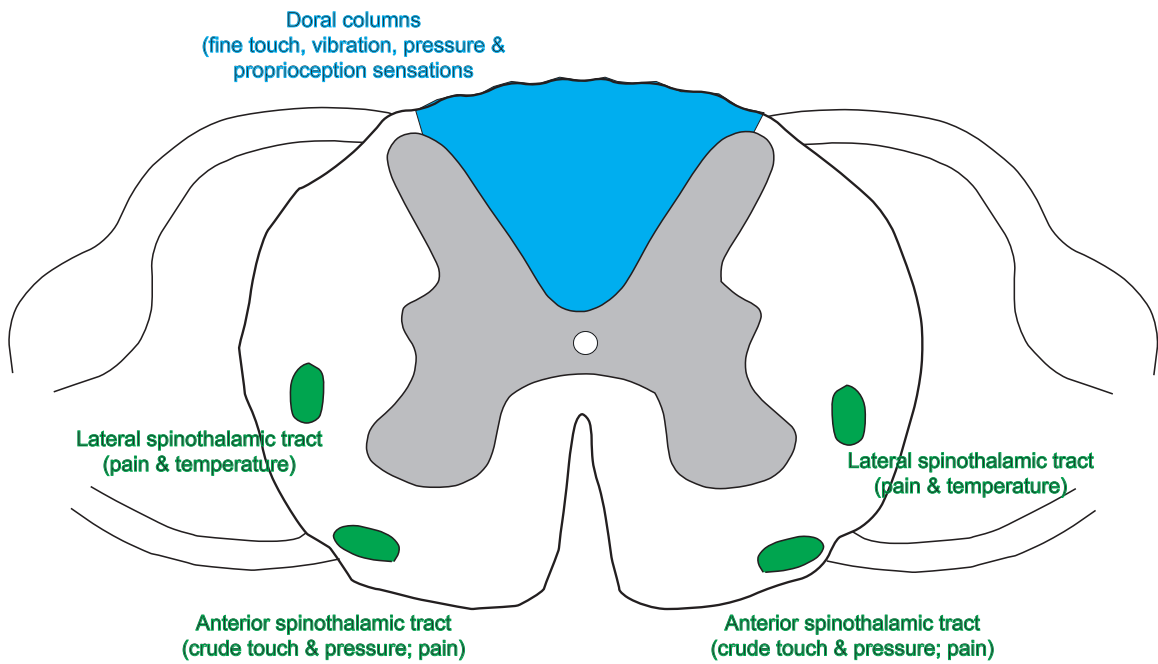


Figure 7 - Pain related ascending sensory tracts

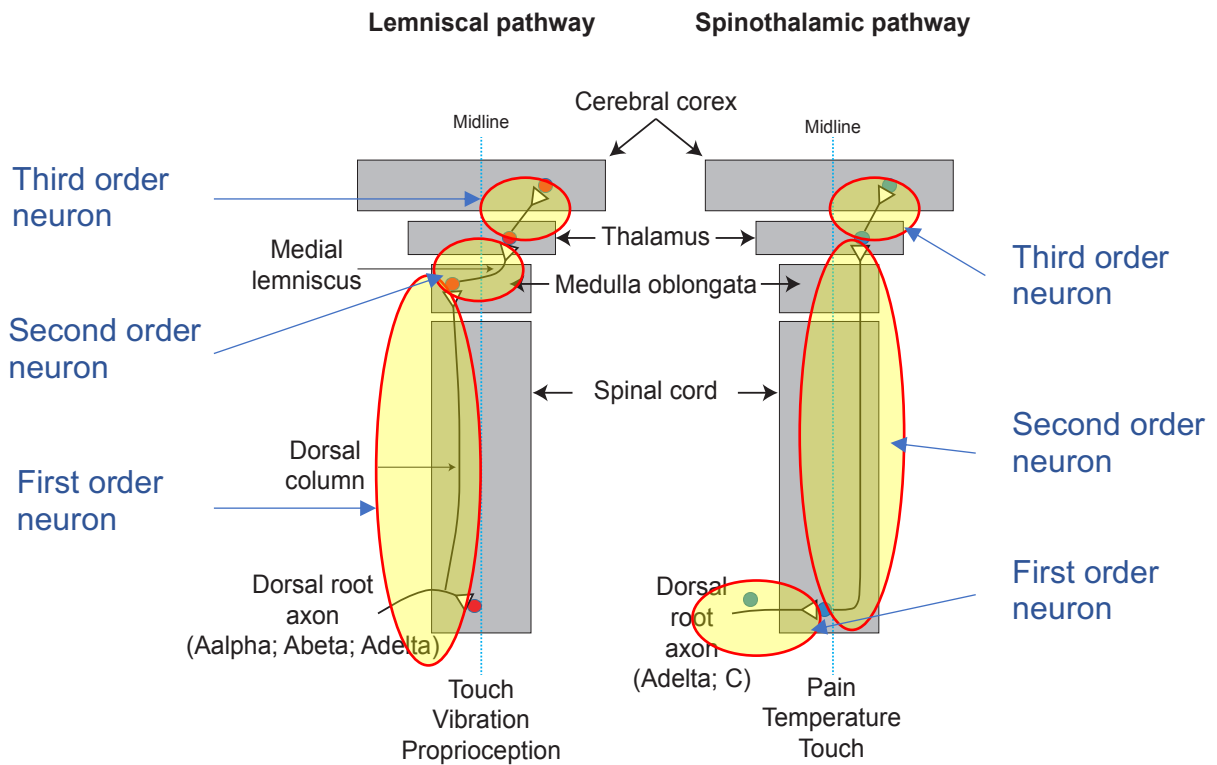


Figure 10 - 3 order neurons

The different neurons can be divided depending on their order in.

First order neurons:

- Aalpha (Non-pain).
- Abeta (Non-pain).
- Adelta (Pain).
- C-fibers (Pain).

Second order neurons:

- Receive impulses from Abeta, Adelta and C fibers.
- End in thalamus.

Third order neurons:

- Start in thalamus.
- Ends in specific brain centers (Cerebral cortex).
- Allows to feel pain with integration of past experience, emotions.
- Determination of reaction stimulus.

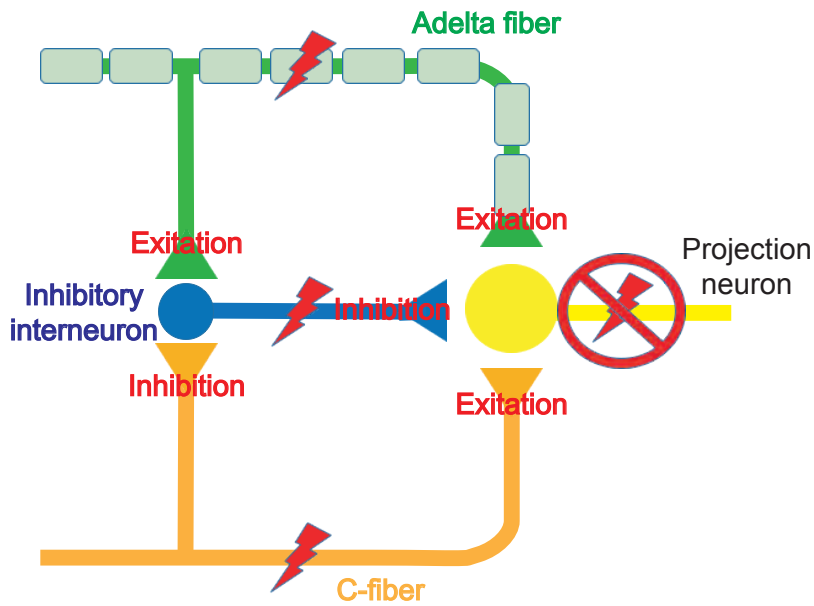


Figure 13 - No pain

3.3.4. Transport of Pain Stimuli

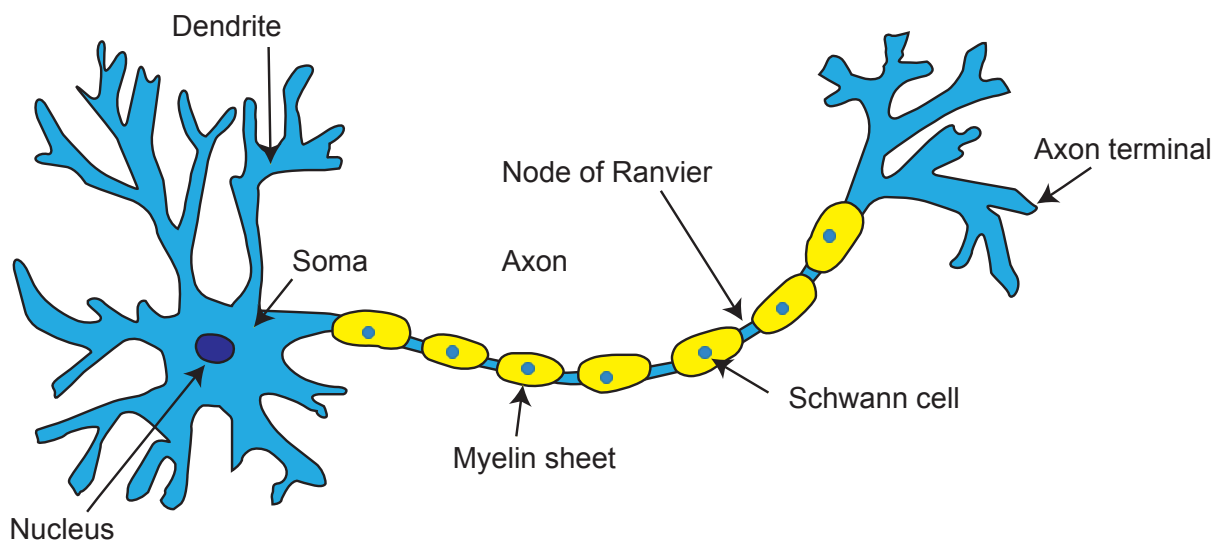


Figure 14 - Neuron

There are 3 types of neurons:

- Sensory neurons (Signals to the central nervous system).
- Motor neurons (Signals away from the central nervous system).
- Interneurons (Signals within the central nervous system).

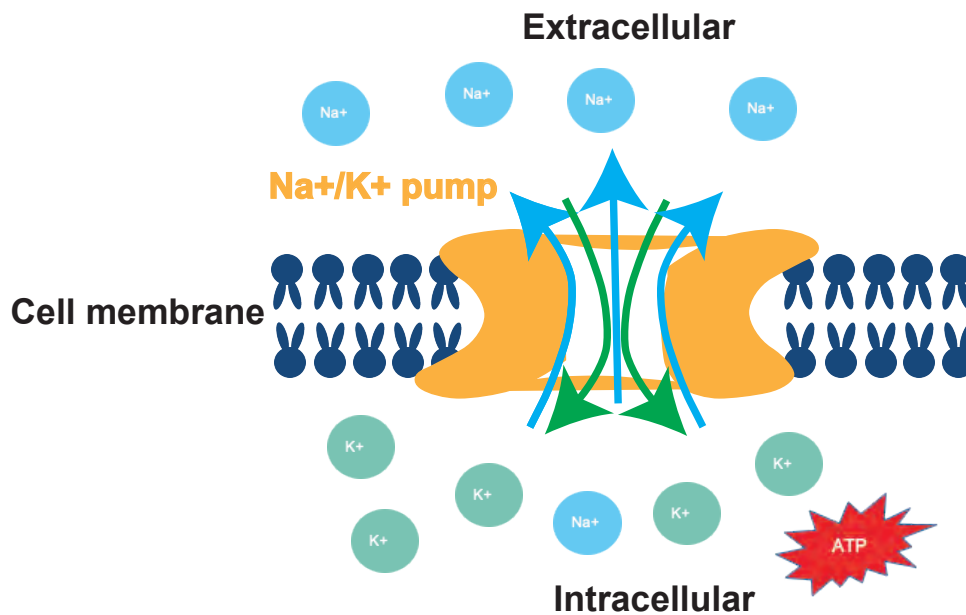


Figure 18 - Sodium/potassium pump

When an action potential (= Nerve impulse) travels down an axon there is a change in polarity across the axon membrane.

In response to a signal from another neuron, sodium and potassium gated ion channels open and close as the membrane reaches its threshold potential.

Sodium channels open at the beginning of the action potential and sodium moves into the axon, causing depolarization.

Repolarization occurs when the potassium channels open and potassium moves out of the axon, creating a change in polarity between the outside of the cell and the inside.

The impulse travels along the axon in 1 direction only to the axon terminal where other neurons are signalled.

Graded potentials

These are changes in membrane potential that vary in size as being opposite to all or none. They make the membrane potential less negative or more positive. They make therefore the cell more likely to have action potentials.

Many can happen at once and can come in different sizes. (Action potentials are always excitatory and only one at the time: all-or-none.)

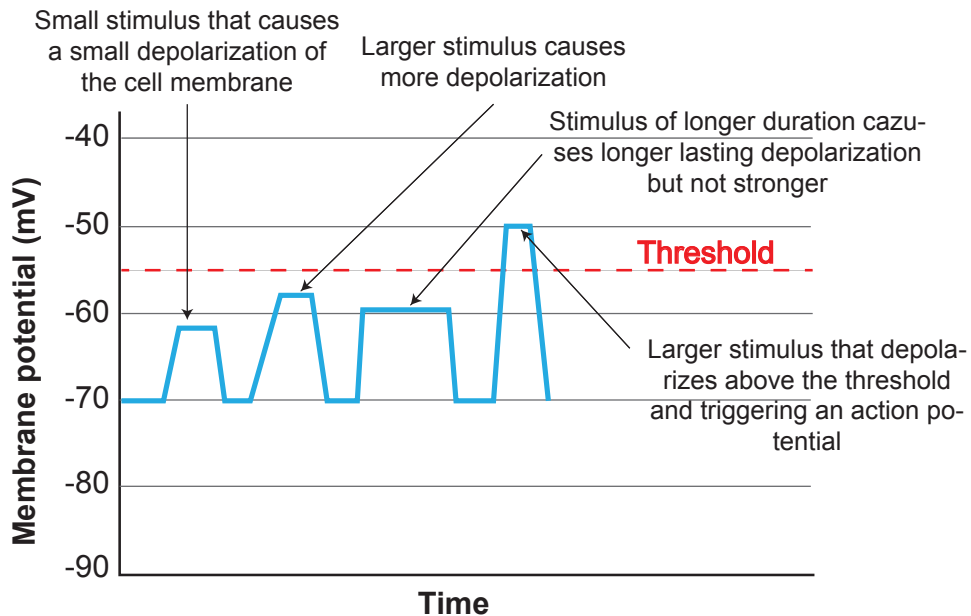


Figure 22 - Graded potentials

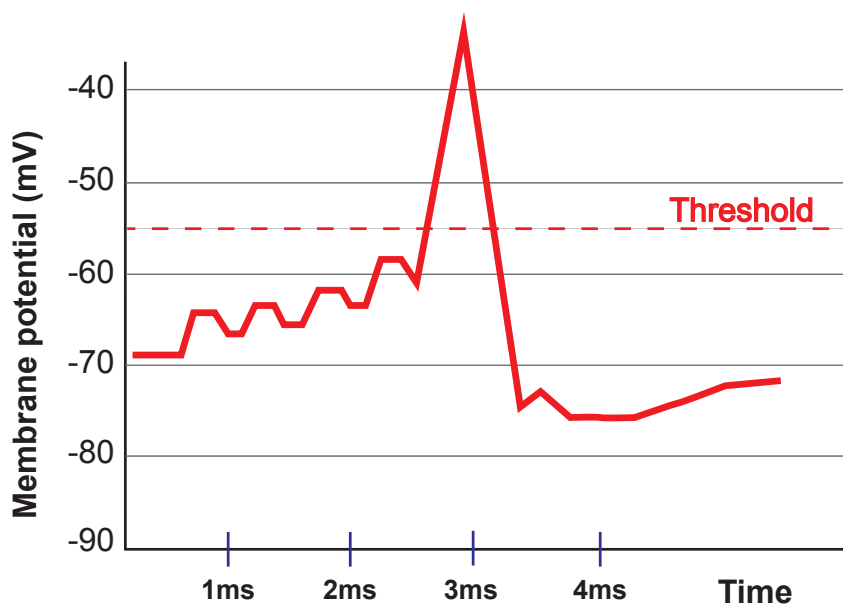


Figure 23 - Graded potentials

Glia cells are at the cross-road between the immune system and the central nervous system. This interaction is crucial in patients with persistent pain. They all show alteration of the immune system.

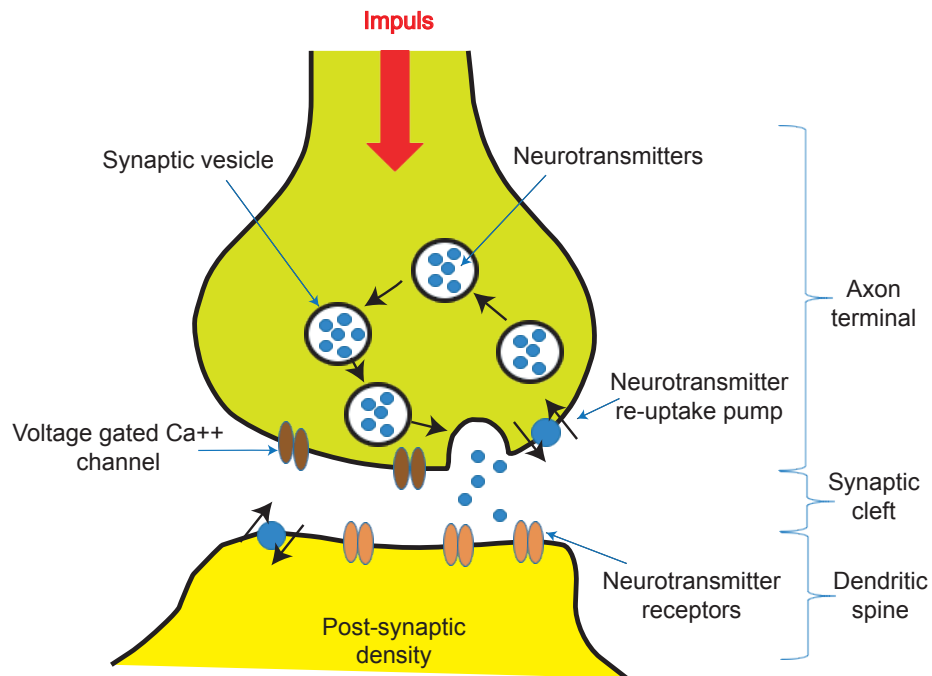


Figure 24 - Synaptic bouton: chemical synapse and action potential

Neurotransmitters

Neurotransmitters are products that:

- Are synthesized in the nerve cell.
- Are present in the presynaptic neuron parts and can induce an action on the postsynaptic neuron or organ.

There are different neurotransmitters. We explain the most common.

Acetylcholine: it is a small excitatory molecule neurotransmitter involved in synaptic transmission at neuromuscular junctions controlling:

- The vagus nerve.
- Cardiac muscle fibers.
- The skeletal and visceral motor systems and various sites in the central nervous system.
- Inadequate amounts lead to depression and possibly Alzheimer's disease.

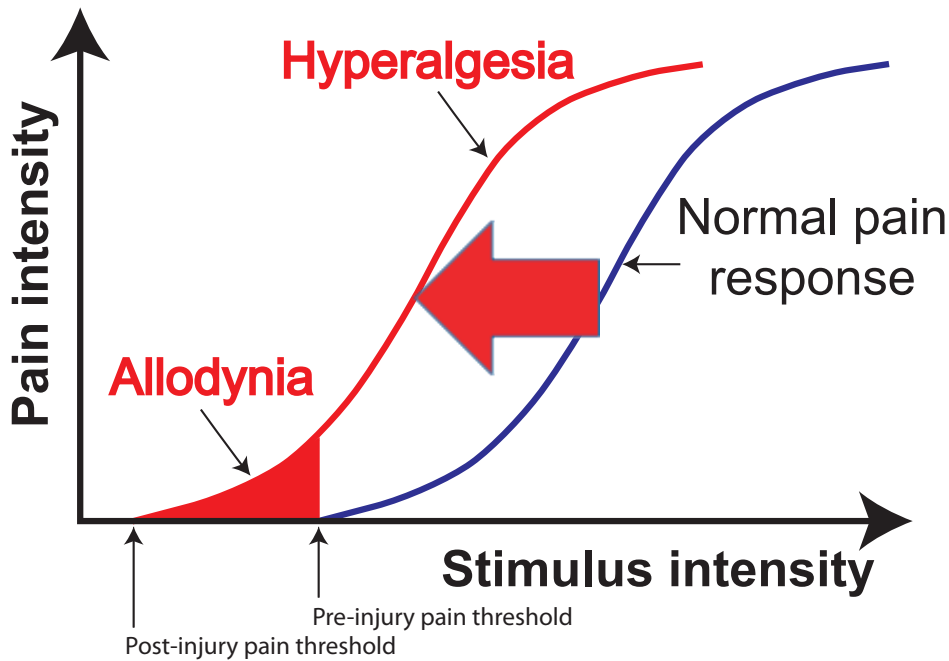


Figure 30 - Hyperalgesia and allodynia

Hypoalgesia is decreased sensibility to pain. Hypoalgesia can be caused by exogenous chemicals such as opioids, as well as by chemicals produced by the body in phenomena such as fear- and exercise- induced hypoalgesia. Hypoalgesia can also be associated with diseases, such as some rare neurological disease or in less severe cases with diabetes or other diseases associated with hypertension.

Analgesia is the absence of pain to a noxious stimulus. It is the interruption in the nervous system pathway between the sense organ and the brain (Through injury or disease – examples are tabes dorsalis, syringomyelia, tumors in the spinal cord). It can also be induced by medication.

Paresthesia (Sometimes called numbness) is an abnormal sensation (Spontaneous or provoked) that is not unpleasant. Can be tingling, tickling, itching or crawling.

The most common, everyday cause is temporary restriction of nerve impulses to an area, commonly caused by leaning or resting on parts of the body such as the legs (Often followed by a pins and needles tingling sensation).

Other causes include conditions such as hyperventilation syndrome and panic attacks.

Also, rheumatoid arthritis, psoriasis, carpal tunnel syndrome are common sources of paresthesia.

Paresthesia can also be caused by poor circulation in the limbs such as in peripheral vascular disease and arteriosclerosis.

Without a proper supply of blood and nutrients, nerve cells can no longer adequately send signals to the brain. Because of this, paresthesia can also be a symptom of vitamin deficiency and malnutrition.

Acroparesthesia is severe pain in the extremities caused by some diseases and by hypocalcemia (For example in the case of hypoparathyroidism or vit. D deficiency).

The neuromuscular symptoms of hypocalcemia are caused by an increased responsiveness due to the decreased interaction of calcium with sodium channels. Since calcium blocks sodium channels and inhibits depolarization of nerve and muscle fibers, reduced calcium lowers the threshold for depolarization.

Ischemic pain (For example chest pain in the case of a heart attack, angina pectoris or intermittent claudication when walking) is not only caused by lactic acid because the pH change is too small.

Another compound released from the ischemic muscle, ATP (Adenosine triphosphate), works together with acid by increasing the pH sensitivity of ASIC3 (Acid sensing ion channel #3), the molecule used by sensory neurons to detect lactic acidosis.

Visceral pain (Over-distention of hollow organs, ischemia, obstruction or spasm of hollow organs, overstretching of capsules of solid organs, abrupt anoxemia of visceral muscles, necrosis, inflammation) runs over C-fibers (Autonomic). Often associated with unpleasant emotions and autonomic changes such as nausea, vomiting, lowering blood pressure.

Be aware that some visceral tissues are insensitive to pain such as the parenchyma of the liver, brain tissue, alveoli in the lungs, inside stomach and intestines.

On the other side, for example meninges, parietal pleura, liver capsule and bronchi are very sensitive to pain.

There are many visceral sensations that are unpleasant but below the level of pain, for example feeling of disagreeable fullness or acidity of the stomach or undefined and unpleasant thoracic or abdominal sensation. These visceral sensations may precede the onset of visceral pain.

Painful visceral afferent impulses activate anterior horn motor cells to produce rigidity of muscles (= Viscero-motor reflexes).

These nerve sprouts and neuromas can generate spontaneous activity. This then means that the sensitivity raises. The area becomes more sensitive to physical stimuli, manifested as tenderness.

Between the nerve sprouts, atypical connections can develop, thus permitting crosstalk between somatic or sympathetic efferents and nociceptors.

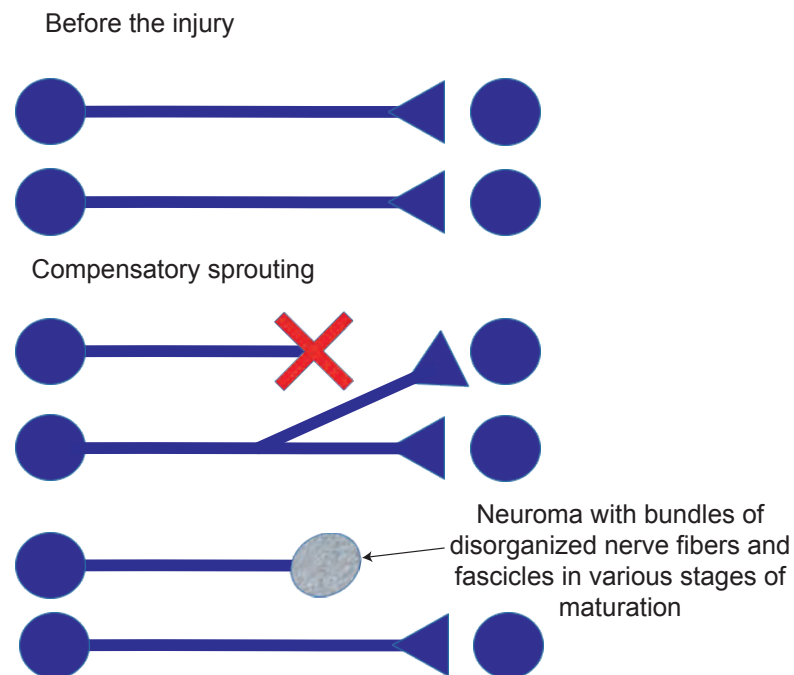


Figure 32 - Sprouting and neuroma

Neuromas can be asymptomatic, but they can also be painful if they are chronically irritated or if the axons within are constantly stimulated.

The firing is in burst mode.

This type of pain is related to hyperalgesia.

The repeated spontaneous burst firing in C-fibers can lead to wind-up phenomenon associated with worsening of the pain perception. This is sensitization.

Neuropathic pain can also be caused by diseases such as diabetes mellitus or spinal cord injury.

Wind-up phenomenon is a repeated nociceptor stimulus that evokes a period of facilitated transmission in spinal dorsal horn neurons characterized by a progressive increase in action potential output, associated with worsening pain perception. It is related to bursting in C-fiber plasticity. So, the pain is getting a worse intensity.

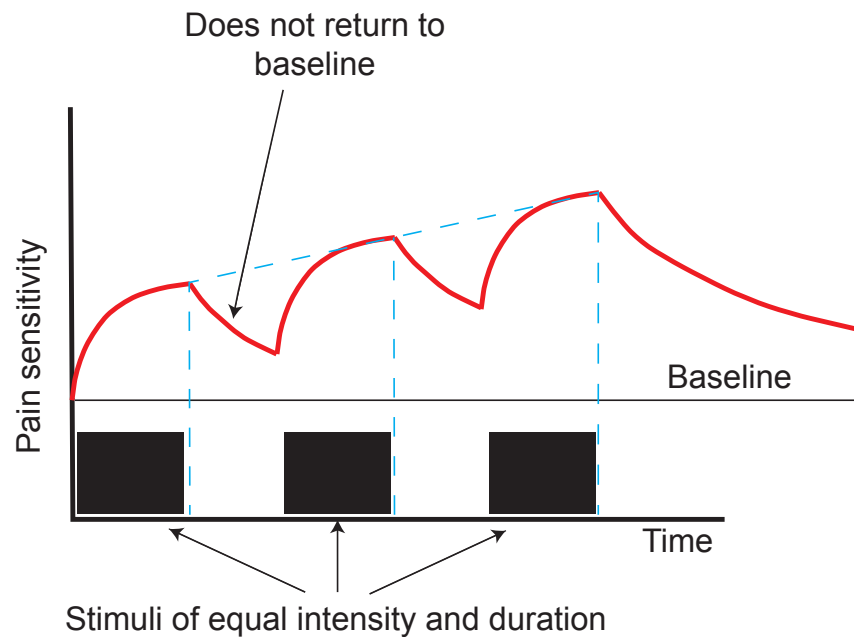


Figure 33 - Wind-up phenomenon

Menstruation pain. Menstrual cramps usually refer to a dull, throbbing, cramping pain in the lower abdomen, just above the pelvic bone.

Other symptoms may include:

- Pain in the lower back and thighs.
- Nausea and vomiting.
- Sweating.
- Faintness and dizziness.
- Diarrhea or loose stools.
- Constipation.
- Bloating.
- Headaches.

Approximately once every 28 days, if there is no sperm to fertilize the egg, the uterus contracts to expel its lining.

Hormone-like substances called prostaglandins trigger this process.

Prostaglandins are chemicals that form in the lining of the uterus during menstruation. They cause muscle contractions and cramps that are similar to labor

Meerveld B.G. & Johnson A.C. (2018) Mechanisms of Stress-induced Visceral Pain. *J. Neurogastroenterol. Motil. Jan.*; 24(1): 7–18. PubMed #29291604. *PainSci.* #52282.

Meyer R., Ringkamp M., Campbell J.N. & Raja S.N. (2006) Peripheral mechanisms of cutaneous nociception. In: McMahon SB, Koltzenburg M. (eds) *Textbook of Pain*, 5th edn. London: Elsevier, pp. 33–35.

Melzack R., Wall P.D. (1965) Pain mechanisms: a new theory. *Science* 1965;150: pp. 971–979.

Mitchell J., Cohen, William C. & Neff J.D. (2018) *Challenging Neuropathic Pain Syndromes; Chapter 1 - Pathophysiology of Pain*, Elsevier, Pages 1-5, ISBN 9780323485661,

Moayedi M. & Davis K.D. (2013) Theories of pain: from specificity to gate control. *J. Neurophysiol.* 2013; 109:5-12

Ossipov M.H., Morimura K. & Porreca F. (2014) Descending pain modulation and chronification of pain. *Current opinion in supportive and palliative care*, 8(2), 143–151. doi:10.1097/SPC.0000000000000055

Pinheiro E.S., de Queirós F.C., Montoya P., Santos C.L., do Nascimento M.A., Ito C. H. & Baptista A.F. (2016) Electroencephalographic Patterns in Chronic Pain: A Systematic Review of the Literature. *PloS one*, 11(2), e0149085. doi:10.1371/journal.pone.0149085

Portenoy R.K. & Hagen N.A. (1990) Breakthrough pain: definition, prevalence and characteristics. *Pain* 1990, 41:273–281.

Queme L.F. & Jankowski M.P. (2019) *Current Opinion in Physiology. Sex differences and mechanisms of muscle pain.* Volume 11, Pages 1-6, ISSN 2468-8673,

Scerbo T. Colasurdo J., Dunn S., Unger J., Nijs J. & Cook C. (2017) Measurement Properties of the Central Sensitization Inventory: A Systematic Review. *Pain Pract.*

Sherman S.M. (2001) Tonic and burst firing: dual modes of thalamocortical relay. *Trends in Neurosciences.* Vol. 24, Issue 2, pp. 122-126.

Staud R. & Spaeth M. (2008) Psychophysical and neurochemical abnormalities of pain processing in fibromyalgia. *CNS Spectr.* Mar. 13 (3 Suppl. 5): 12-17.

Tracey W.D. (2017) Nociception. *Current Biology.* Vol. 27, Issue 4, pp. R129-R133.

Annex 1 - The Central Sensitization Inventory - CSI

Give an indication on whether you have this complaint or not.

Name Patient.....

Date.....

PART A

1	I feel tired and unrefreshed when I wake from sleeping.	Never	Rarely	Sometimes	Often	Always
2	My muscles feel stiff and achy.	Never	Rarely	Sometimes	Often	Always
3	I have anxiety attacks.	Never	Rarely	Sometimes	Often	Always
4	I grind or clench my teeth.	Never	Rarely	Sometimes	Often	Always
5	I have problems with diarrhea and/or constipation.	Never	Rarely	Sometimes	Often	Always
6	I need help in performing my daily activities.	Never	Rarely	Sometimes	Often	Always
7	I am sensitive to bright lights.	Never	Rarely	Sometimes	Often	Always
8	I get tired very easily when I am physically active.	Never	Rarely	Sometimes	Often	Always
9	I feel pain all over my body.	Never	Rarely	Sometimes	Often	Always
10	I have headaches.	Never	Rarely	Sometimes	Often	Always
11	I feel discomfort in my bladder and/or burning when I urinate.	Never	Rarely	Sometimes	Often	Always
12	I do not sleep well.	Never	Rarely	Sometimes	Often	Always
13	I have difficulty concentrating.	Never	Rarely	Sometimes	Often	Always
14	I have skin problems such as dryness, itchiness, or rashes.	Never	Rarely	Sometimes	Often	Always
15	Stress makes my physical symptoms get worse.	Never	Rarely	Sometimes	Often	Always
16	I feel sad or depressed.	Never	Rarely	Sometimes	Often	Always
17	I have low energy.	Never	Rarely	Sometimes	Often	Always
18	I have muscle tension in my neck and shoulders.	Never	Rarely	Sometimes	Often	Always
19	I have pain in my jaw.	Never	Rarely	Sometimes	Often	Always
20	Certain smells, such as perfumes, make me feel dizzy and nauseated.	Never	Rarely	Sometimes	Often	Always
21	I have to urinate frequently.	Never	Rarely	Sometimes	Often	Always
22	My legs feel uncomfortable and restless when I am trying to go to sleep at night.	Never	Rarely	Sometimes	Often	Always
23	I have difficulty remembering things.	Never	Rarely	Sometimes	Often	Always
24	I suffered trauma as a child.	Never	Rarely	Sometimes	Often	Always
25	I have pain in my pelvic area.	Never	Rarely	Sometimes	Often	Always
				Total score:		

Acknowledgment

I am grateful to those colleagues who knowingly or unknowingly assisted in the development of this book.

I also acknowledge that the constant questioning by students has significantly contributed to the development of the material in this book.

My greatest debt of gratitude goes to those family members and good friends whose tireless support made the writing of this book possible.



Luc Peeters
Master of Science in Osteopathy (MSc.Ost) - UAS

Luc Peeters is an osteopath since 1985. He was the Joint-Principal of the largest Academy of Osteopathy in Europe from 1987 till 2020.

This book gives a practical overview of the neurophysiological mechanisms of pain and how osteopaths deal with patients with pain.

The theory and procedures in this book are checked on their scientific background and esotericism is avoided.

Author & Publisher: Luc Peeters

Mail: info@osteopathybooks.com