Pain

Pain is a subjective unpleasant sensation mediated by the afferent nervous system and the cerebral cortex, related to possible or actual tissue damage. **Pain** is the most common reason for a patient to seek medical treatment.

Anatomy and physiology

- The pain pathway is a **three-neuron afferent pathway** with numerous connections to other areas of the brain - therefore painful stimuli are associated with unpleasant sensations, activation of sympathetic, parasympathetic, and motor responses.
- Pain is a danger signal that can be followed by tissue damage. It can help to localize the disease process.
- **Nociception** (the generation and transmission of the pain signal) is a neurohumoral process involving the generation of pain by irritation of nociceptors, its conduction through nerve fibres to the brain and its subsequent processing by the central nervous system.
- **Pain** - is the result of the processing of this painful stimulus in the central nervous system, i.e. the perception of pain as a subjective sensation (therefore the final perception of pain always depends on the processing in the CNS and not on the nature of the original stimulus).

The trajectory of pain

- Three-neuron.
- **The first neuron** - a pseudounipolar cell of the spinal ganglion conducts the stimulus from the nociceptor (pain receptor).
  - Nociceptive fibres of the first neuron enter the superficial part of the posterior horns of the spinal cord, where they ascend and descend a few segments higher and lower and form synapses with neurons of the posterior horns of the spinal cord – hence the transfer to α-motoneurons in the anterior horns of the spinal cord and the reflex motor response to the pain stimulus.
- **The second neuron** - in the posterior corners of the spinal cord (Rexed zones 1, 2, 3, 5, 6, 7), hence:

  Tractus spinothalamicus - rapid transmission of excitement to the ventral posterolateral part of the thalamus. Sharp, stabbing, stabbing pain is transmitted.
  Tractus spinoreticularis - slow transmission of excitation to the reticular formation and from there through the third neuron of the connection to the thalamus (hence sometimes in the literature the name of the pathway tr. spinoreticulothalamicus). Developmentally older pathway. Transmission of slow, dull, difficult to localize pain.
  Tractus spinoparabrachioamygdalaris and **tractus spinoparabrachiohypothalamicus** - connections to the limbic system, affect the emotional component of pain.

- **Third neuron** or fourth neuron - rewiring from thalamus to somatosensory cortex and association cortical areas.
  - Analysis of information from the periphery and motor or other response is made by efferent fibres. Central control allows modification of pain.

Nociceptors

- **Free nerve endings** - normally silent receptors sensitive to pH changes, increased extracellular concentration of potassium ionts, prostaglandins E_2 a E_1, leukotrienes, histamine, substance P, CGRP (calcitonin gene-related peptid).
- **Polymodal nociceptors** - besides sensitivity to pain, they are also sensitive to sensations such as cold, heat and mechanical stimuli. These are Ruffini's corpuscles, Krause's corpuscles, proprioceptors.
- **High threshold nociceptors** - normally sense touch, pressure, thrust and vibration. These are the Vater-Pacini bodies, Merckel's discs and Meissner's bodies.

Nerve fibres for pain conduction

- ***A δ*** - weakly myelinated fibres conduct excitation at a speed of 5-30 m/s. They conduct sharp, well-defined pain.
- ***Unmyelinated C fibres*** - their free endings belong to polynodal receptors. They conduct excitations slowly at a speed of 0.5-2 m/s and mediate conduction of deep poorly discriminated diffuse pain.
- ***Aα/Aβ fibres*** - strongly myelinated, mediate conduction of tactile stimuli at 30-70 m/s.

Pain from high-threshold and polynodal receptors can also be conducted by other fiber types.

Pain at the level of the spinal cord

- In the bowl, the pain is arranged into **Rexed zones**. Zone 1, 2, 3 - superficial skin and acute pain. Rexed zone 5, 6, 7, 8, 10 for deep visceral pain.
- The conduction of pain is regulated (dampened) by the **so-called gating mechanism**, which allows only a limited number of impulses to pass through.

Pain control at the central level

- Direct stimulation of certain areas of the brain.
- Descending control to spinal neurons.
- Opioid-induced analgesia.
- Endogenous opioid peptides.

Types of pain

- **Acute** - duration of seconds to weeks, but up to a maximum of three months.
  - Formation by trauma mechanism, surgery, disease.
  - It acts as a powerful stressor and induces the release of catecholamins, stress hormones; catabolism and a decline in immunity.
  - It is accompanied by vegetative symptoms such as: tachycardia, tachypnoea, mydriasis, sweating, urinary retention, slowing of peristalsis, hyperglycaemia.
- **Chronic** - lasts longer than 3 months and persists even after removal of the provoking stimulus or healing of tissue damage.
  - It deteriorates the quality of life, leading to physical and psychological hardship.

- **Superficial pain** - sharp, well localized. Localization depends on the amount of afferent fibers in the area (see sensitive homunculus).
- **Deep somatic and visceral pain** - dull character, longer duration, the extent is diffuse, poorly delineated. May project to different parts of the body (referred pain) within Head's zones. Vegetative reaction and hyperesthesia are evident.

- **Root pain** - arises from irritation of the posterior spinal roots and the nerves arising from them. The pain involves the entire innervation area of the affected nerve (areae radiculares).
- **Phantom pain** - is felt in the amputated part of the body. The reacting neurons have an altered threshold of sensitivity and a number of stimuli are generated in them, which are interpreted in the CNS as pain.
- **Causalgia** - damage to tissue or nerves that painfully stimulates a neuron in the posterior horns of the spinal cord and transmits painful stimuli further to higher brain centers. Normally painless stimuli can then trigger a painful reaction. Pain may be accompanied by hyperalgesia and hyperesthesia, vasomotor and trophic damage to the area (see post-traumatic neuralgia, Sudeck's analgodystrophy).
- **Neuralgia** - painful sensations spreading along the cranial and spinal nerves. Sharp pain can be provoked by trauma, infectious process.

Examination of a patient with pain

- Detailed medical history:
  - Personal, family, social, medication, allergic, current illness - own history of pain.
- General clinical examination:
  - Interdisciplinary cooperation.
- Auxiliary examinations:
  - RTG, CT, MRI, EEG, EMG, USG, scintigraphy of bones, laboratory tests.

Clinical assessment of pain intensity

There is no objective measurement of pain as a subjective sensation. Any measurement of pain is therefore dependent on the patient's perception of pain - i.e. it is individual for each patient.

Verbal evaluation

- **Verbal evaluation**
  - 0 =no pain,
  - 1 = a little pain,
  - 2 = moderate pain,
  - 3 = severe pain,
  - 4 = excruciating pain.
  - 5
  - 6
  - 7
  - 8
  - 9
  - 10

Visual analogue and numeric scale

- **Visual analogue scale (http://www.stopache.us/vas/)** – rates pain on a scale from no pain to excruciating pain.
- **Numerical scale** - correlates with the analogue scale 0 - no pain to 10 - unbearable pain.

FLACC scale

The FLACC (***F**ace, **L**egs, **A**ctivity, **C**ry, **C**onsolability*) scale is used for patients with whom there is no valid verbal contact (patients with quantitative impairment, aphasia, advanced cognitive deficits, young children, etc.).

Brief overview of therapy

Pain treatment depends on the severity of the pain reported by the patient - therefore **individual** for each patient. Pain is most often treated **medically**, but there are other therapies such as surgery or alternative treatments such as acupuncture.

Drugs used in pain therapy

- Non-opioid analgesics.
- Opiates.
- Local anaesthetics (catheter techniques).

WHO pain therapy scheme:

- Grade 1 – Non-opiate analgesics (Paracetamol, Metamizol).
- Grade 2 – Moderate opiate (Tramadol, Codein) + non-opiate analgesics.
- Grade 3 – Strong opiate (Morphine, Oxycodone) + non-opiate analgesics.

Links

Related articles

- General anaesthesia
- Regional anaesthesia

External links

- Bolest (česká wikipedie)
- Pain (anglická wikipedie)
- JANČÁLEK, Radim – DUBOVÝ, Petr. *Základy neurověd v zubním lékařství* [online]. MEFANET, ©2011. [cit. 26.11.2011]. <http://portal.med.muni.cz/clanek-560-zaklady-neuroved-v-zubnim-lekarstvi.html>.

- Neuroanatomie bolesti – Prof. Richard Rokyta (http://www.tigis.cz/images/stories/psychiatrie/2006/Suppl_2/09_rokyta_psych_s2-06.pdf)
- Bolest a její léčba – MUDr. Jarmila Hložková (http://public.fnol.cz/www/urgent/seminare/20080313/BOLKAR.pdf)

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- KRETZ, Franz-Josef – TEUFEL, Frank, et al. *Anästhesie und Intensivmedizin.* 1. edition. Heidelberg : Springer Medizin Verlag, 2006. 695 pp. ISBN 3-540-62739-1.

References