



Klassifikation och Analys av Smärta

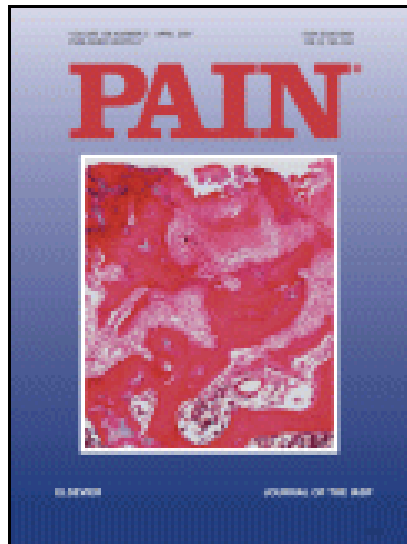
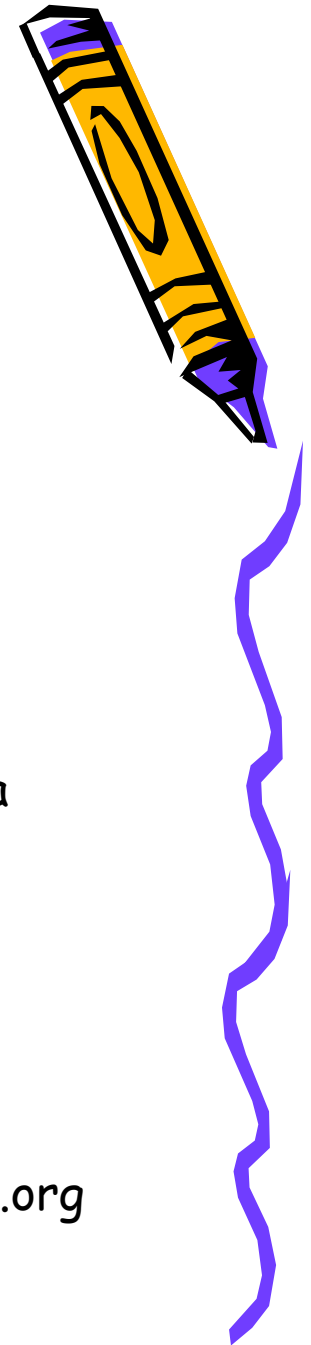


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IASP®

International Association for the Study of Pain®



Grundades 1973.

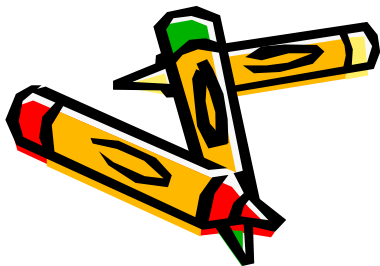
Forskare, kliniskt verksamma läkare och annan sjukvårds/hälsovårdspersonal

Stimulerar och stödjer studier inom smärta och förmedlar denna kunskap över världen

6900 medlemmar i 106 länder

Utgivare av PAIN, 18 nummer per år.

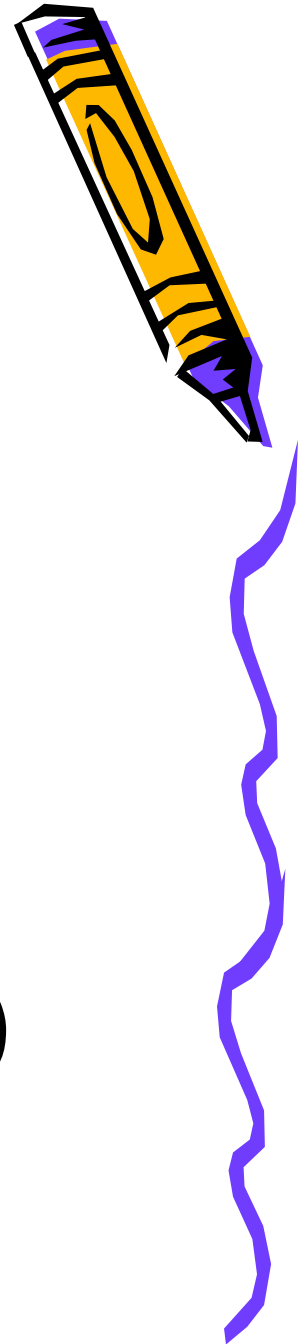
<http://www.iasp-pain.org>



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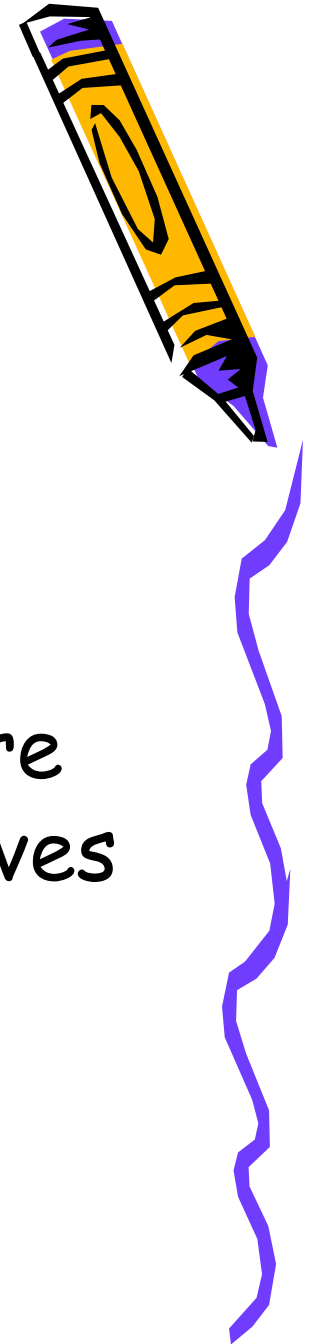
Vad är Smärta ?

- "Smärta är en obehaglig *sensorisk och känslomässig* upplevelse förenad med vävnadsskada eller hotande vävnadsskada eller beskriven i termer av sådan skada" (IASP)



The need for a Taxonomy

- "The development and widespread adoption of universally accepted definitions of terms and a classification of pain syndromes are among the most important objectives and responsibilities of the IASP...
(Bonica, 1979)



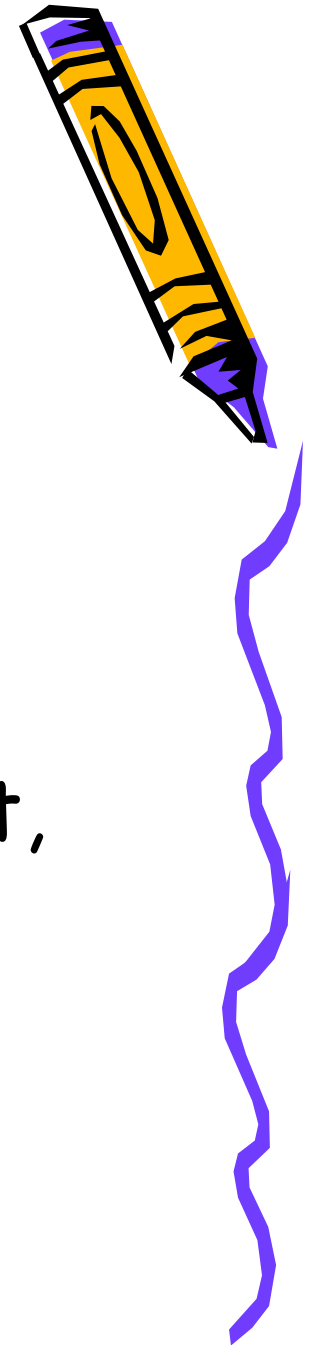
Varför klassifikation ?

- Underlättar kommunikation (muntlig/skriftlig)
- Läroböcker
- Vetenskapliga artiklar/böcker
- Forskning (metodbeskrivning)
- Patientjournaler



Indelning av smärta

- Akut eller kronisk (långvarig)
- Benign eller malign
- Anatomisk: ansikte, bäcken,...
- Organsystem: Hud, muskel, skelett, led, viscera....
- Utlösande orsak



CLASSIFICATION OF CHRONIC PAIN

DESCRIPTIONS OF CHRONIC PAIN SYNDROMES
AND DEFINITIONS OF PAIN TERMS

Second Edition

Task Force on Taxonomy

Editors: Harold Merskey
Nikolai Bogduk

INTERNATIONAL
ASSOCIATION FOR THE
STUDY OF PAIN



I A S P P R E S S

B. RELATIVELY LOCALIZED SYNDROMES OF THE HEAD AND NECK

GROUP II: NEURALGIAS OF THE HEAD AND FACE

Trigeminal Neuralgia (Tic Douloureux) (II-1)

Definition

Sudden, usually unilateral, severe brief stabbing recurrent pains in the distribution of one or more branches of the Vth cranial nerve.

Site

Strictly limited to the distribution of the Vth nerve; unilateral in about 95% of the cases. Usually involves one branch; may involve two or, rarely, even all three branches. The second, third, and first branches of the Vth cranial nerve are involved in the foregoing order of frequency. The pain is more frequent on the right side.

System

Nervous system.

Main Features

Prevalence: relatively rare. *Incidence:* men 2.7, women 5.0 per 100,000 per annum in USA. Most patients have a lesion compressing the nerve where it leaves the brain stem. In patients with multiple sclerosis, there is also an increased incidence of tic douloureux. *Sex Ratio:* women affected perhaps more commonly than men. *Age of Onset:* after fourth decade, with peak onset in fifth to seventh decades; earlier onset does occur, but onset before age 30 is uncommon. *Pain Quality:* sharp, agonizing electric shock-like stabs or pain felt superficially in the skin or buccal mucosa, triggered by light mechanical contact from a more or less restricted site (trigger point or trigger zone), usually of brief duration—a few seconds (but reportedly occasionally up to 1–2 minutes)—followed by a refractory period of up to a few minutes. *Time Pattern:* paroxysms may occur at intervals or many times daily or, in rare instances, succeed one another almost continuously. Periodicity is characteristic, with episodes occurring for a few weeks to a month or two, followed by a pain-free interval of months or years and then recurrence of another bout. *Intensity:* extremely severe, probably one of the most intense of all acute pains.

Precipitation

Pain paroxysms can be triggered by trivial sensations from various trigger zones, that is, areas with increased sensitivity, which are located within the area of trigeminal innervation. The trigger phenomenon can be elicited by light touch, shaving, washing, chewing, etc.

Associated Symptoms and Signs

Occasionally, a mild flush may be noted during paroxysms. In true trigeminal neuralgia, apart from the trigger point, gross neurological examination is usually negative; in many patients, however, careful sensory testing to light touch will show a subtle sensory loss. No particular aggravating factors.

Relief

From carbamazepine, diphenyl hydantoin, and baclofen. If medical measures fail, radio-frequency treatment of the ganglion or microsurgical decompression of the trigeminal root are appropriate.

Usual Course

Recurrent bouts over months to years, interspersed with more or less prolonged asymptomatic phases.

Complications

Usually none. During exacerbations, nourishment may be a (transitory) problem.

Social and Physical Disability

Only as related to the recurrent pain episodes.

Pathology

When present, always involves the peripheral trigeminal (primary afferent) neuron. Impingement on the root by vascular loops, etc., appears to be the most common cause. Demyelination and hypermyelination on electron microscopy.

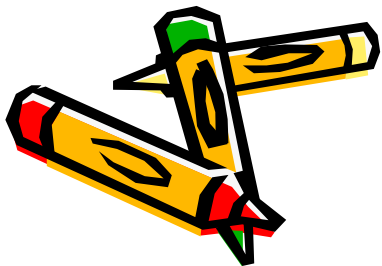
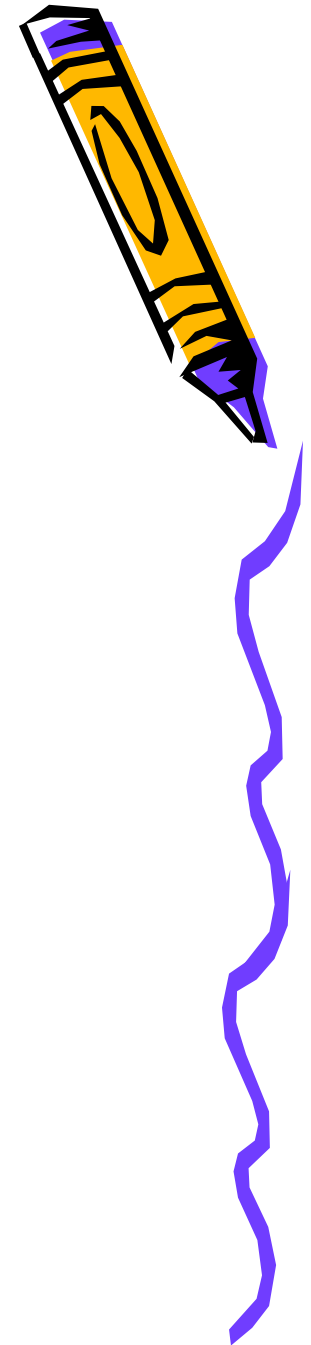
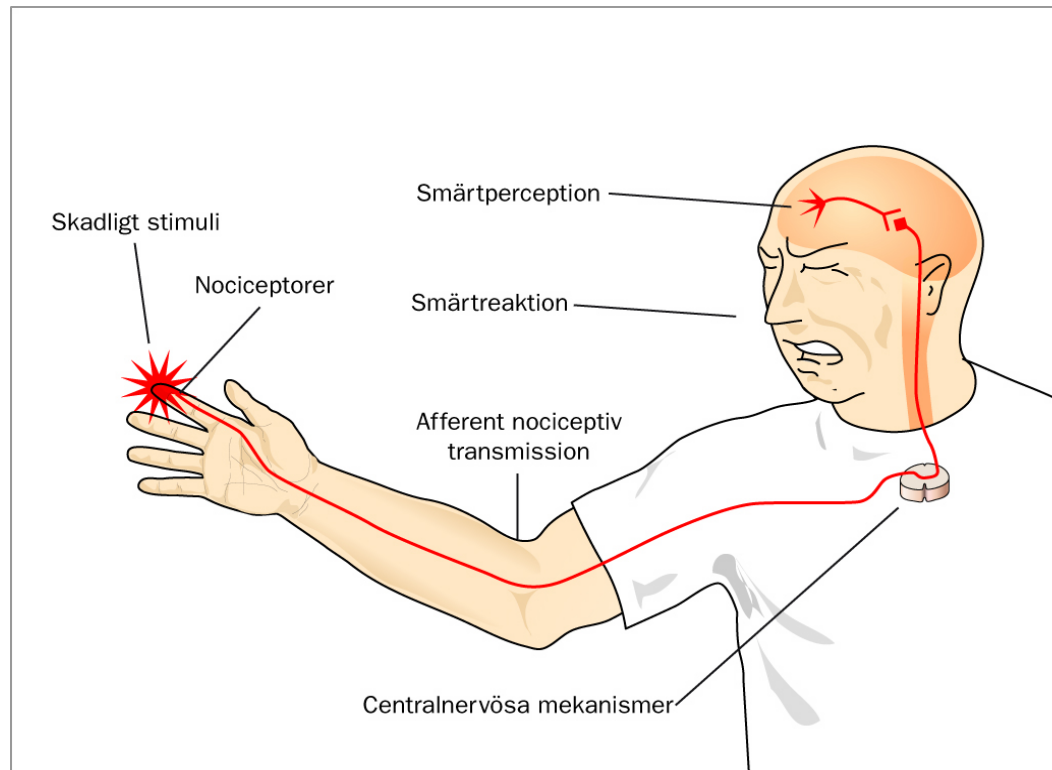
Essential Features

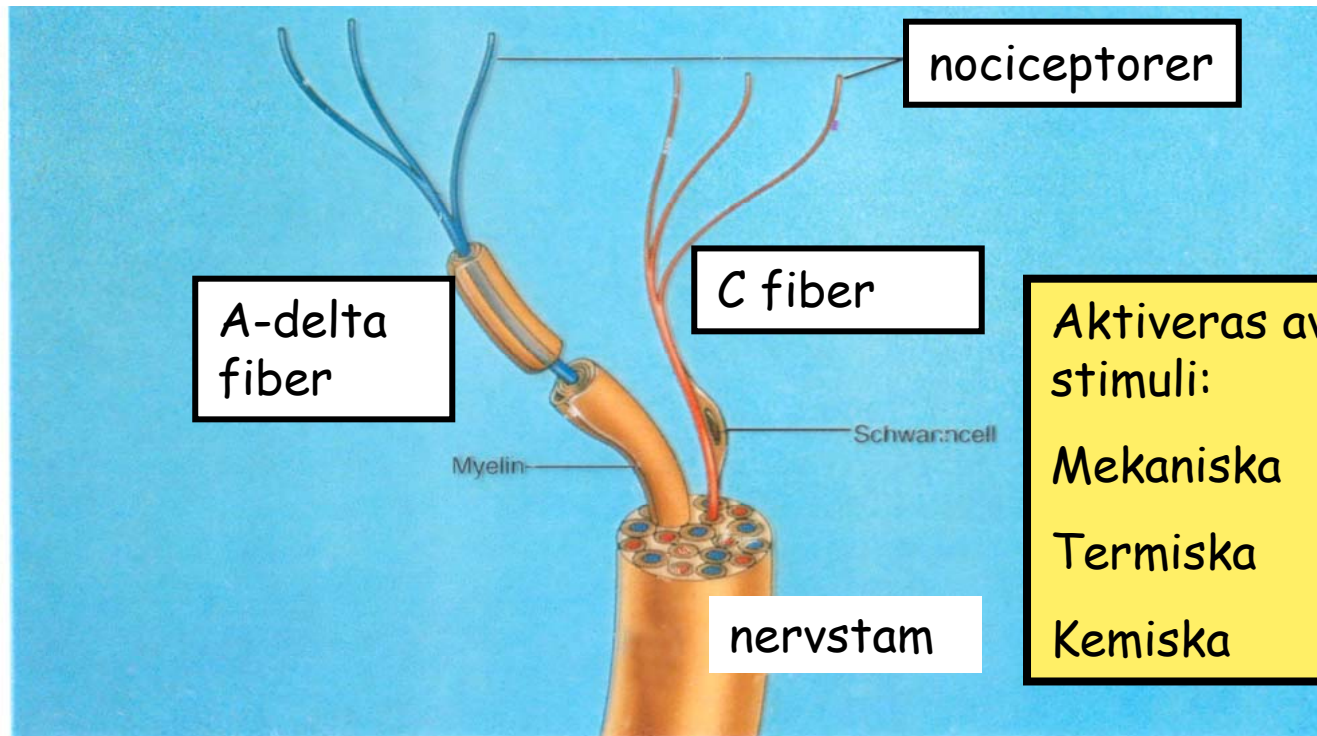
Unilateral, sudden, transient, intense paroxysms of superficially located pain, strictly confined to the distribution of one or more branches of the trigeminal nerve, usually precipitated by light mechanical activation of a trigger point. No sensory or reflex deficit detectable by routine neurologic testing.

Differential Diagnosis

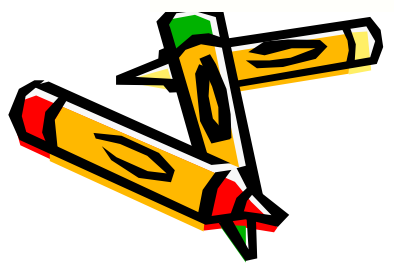
Must be differentiated from symptomatic trigeminal neuralgia due to a small tumor such as an epidermoid or small meningioma involving either the root or the ganglion. Sensory and reflex deficits in the face may be detected in a significant proportion of such cases. Differential diagnosis between trigeminal neuralgia of mandibular division and glossopharyngeal neuralgia may, in rare instances, be difficult. Jabs and Jolts syndrome ("multiple jabs," "ice-pick pain"). SUNCT syndrome.

Smärtimpulsens väg

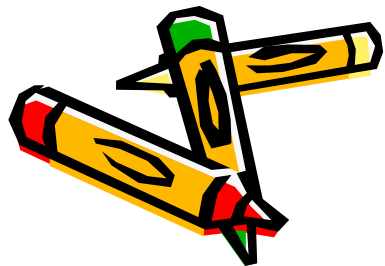
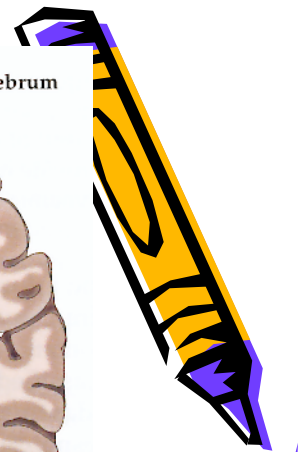
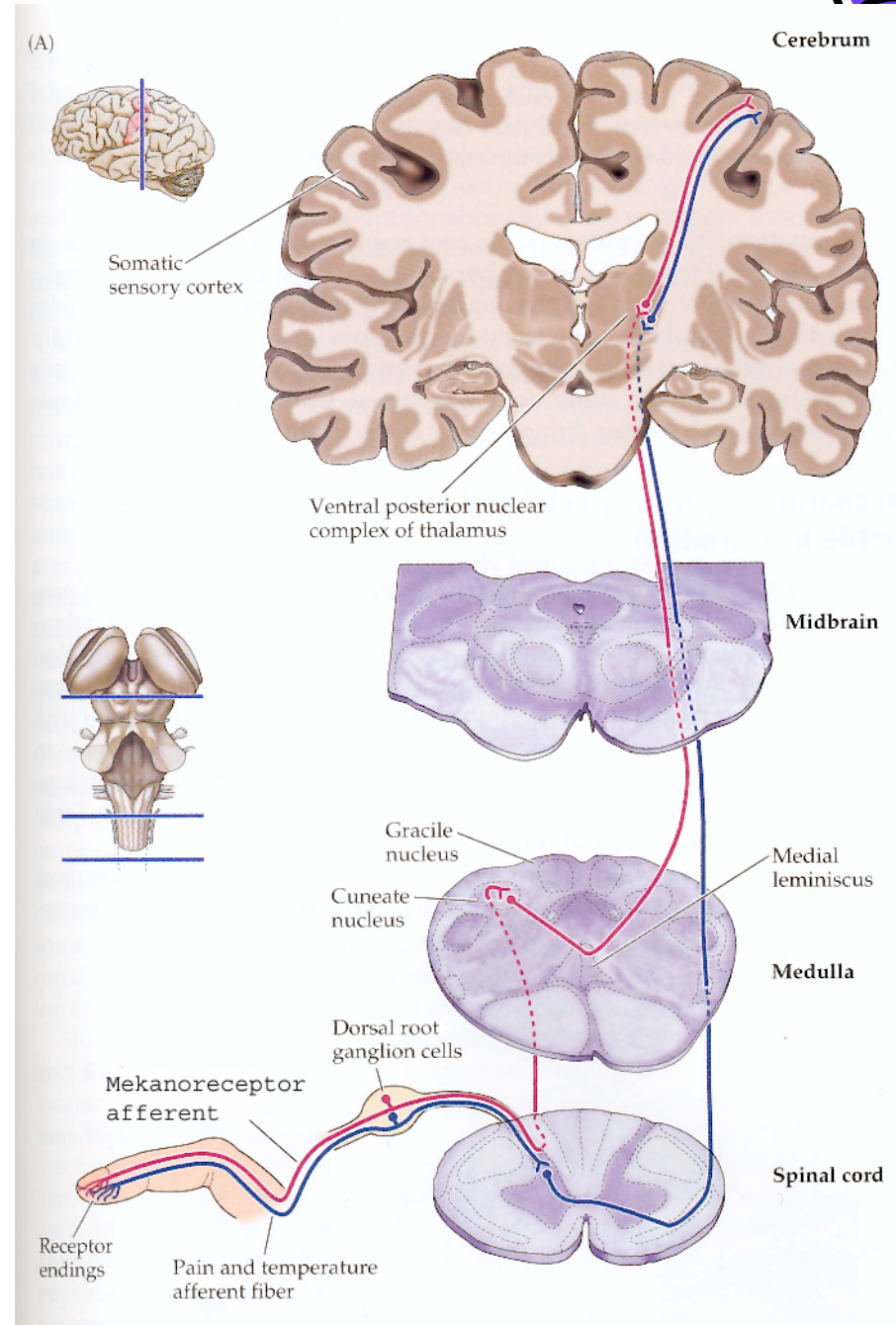




Aktiveras av följande stimuli:
Mekaniska
Termiska
Kemiska



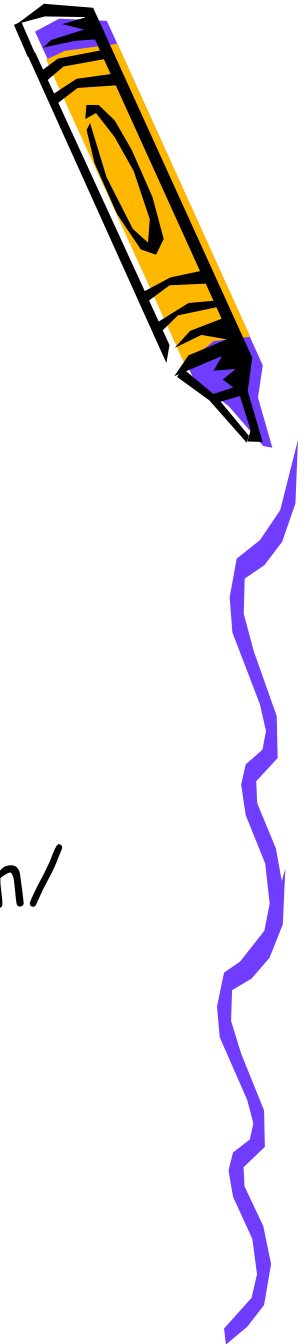
Det somatosensoriska systemet.



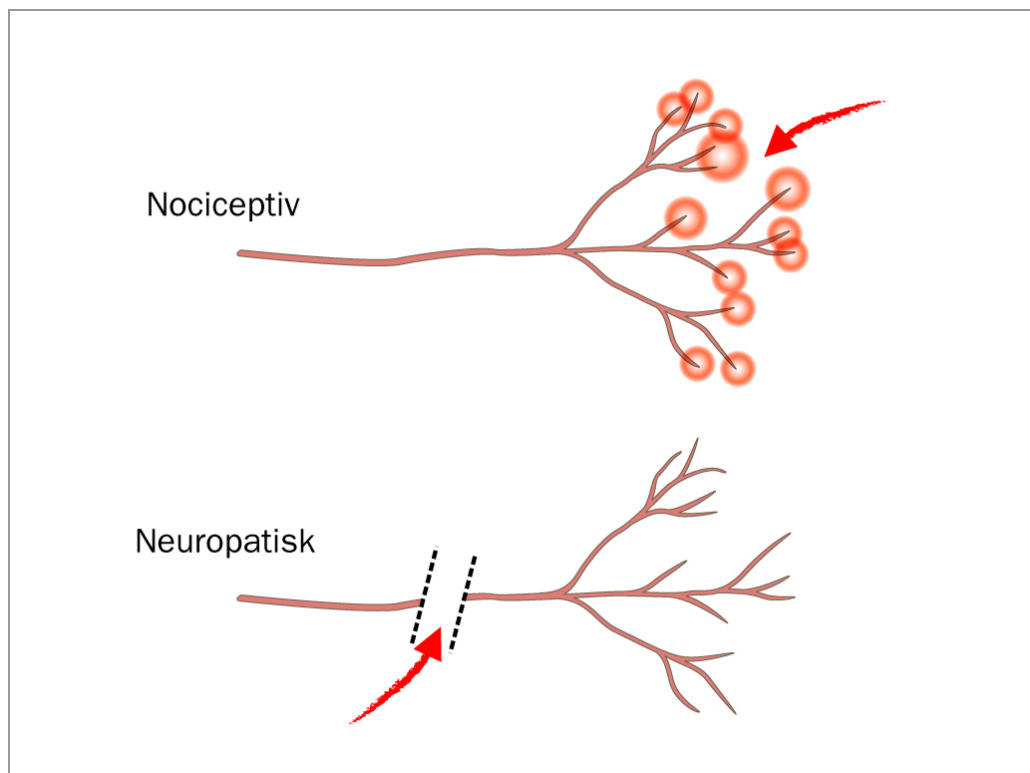
SMÄRTANALYS

- MÅL
 - Att fastställa en smärtdiagnos

Olika terapier
vid nociceptiv/neuropatisk/somatoform/
psykogen smärta



NOCICEPTIV & NEUROPATISK SMÄRTA



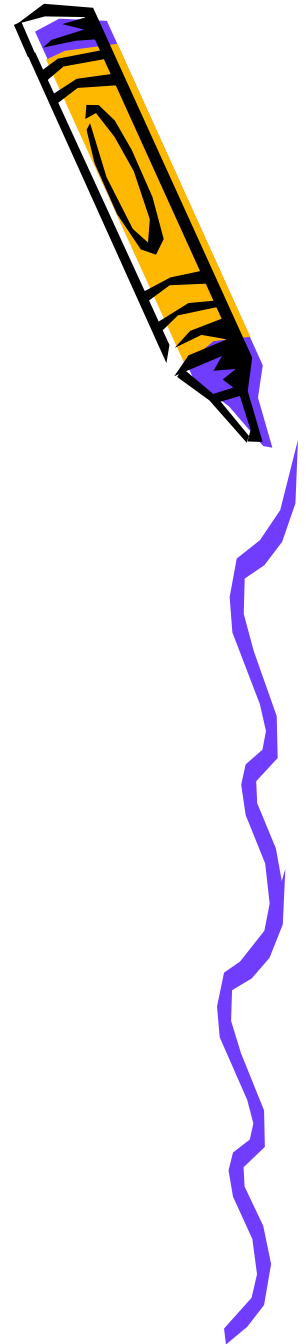
Källa:
Pharmacia Utbildningsmaterial. Jensen NH. De osynliga smärtorna.
Amtsykehuset i Herlev, Danmark. 2003

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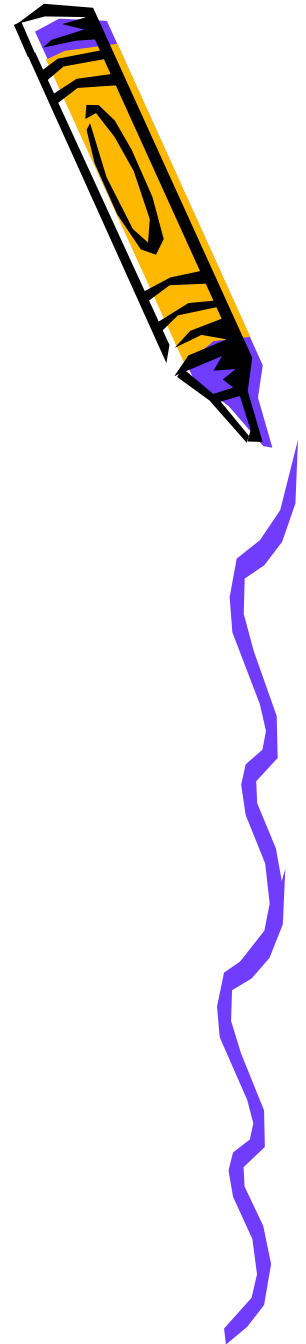
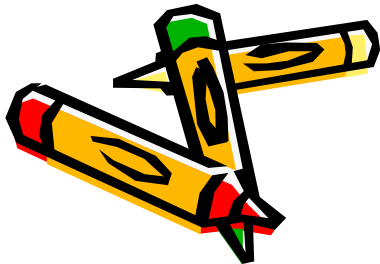
Huvudtyper av smärta

- *Nociceptiv smärta*
- *Neuropatisk smärta*
- *Psykogen smärta*
- *Smärta av okänd orsak*



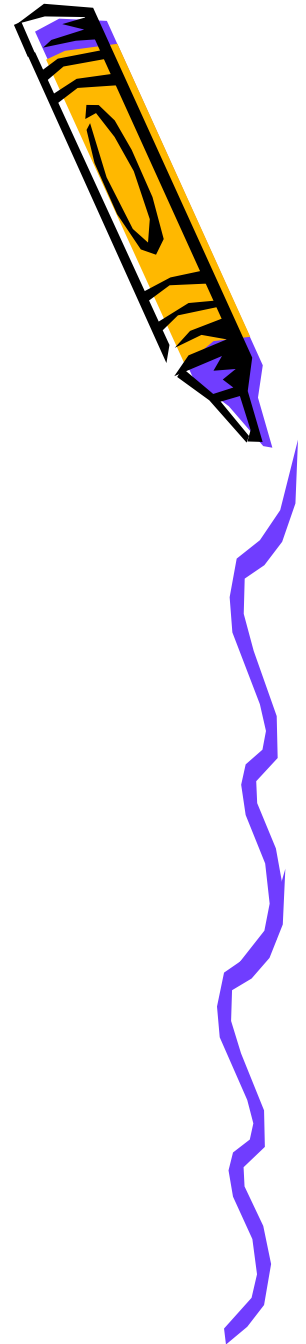
Smärtanalys

- Anamnes (smärtformulär)
- Somatiskt status
- Smärtteckning
- Sensibilitetsundersökning



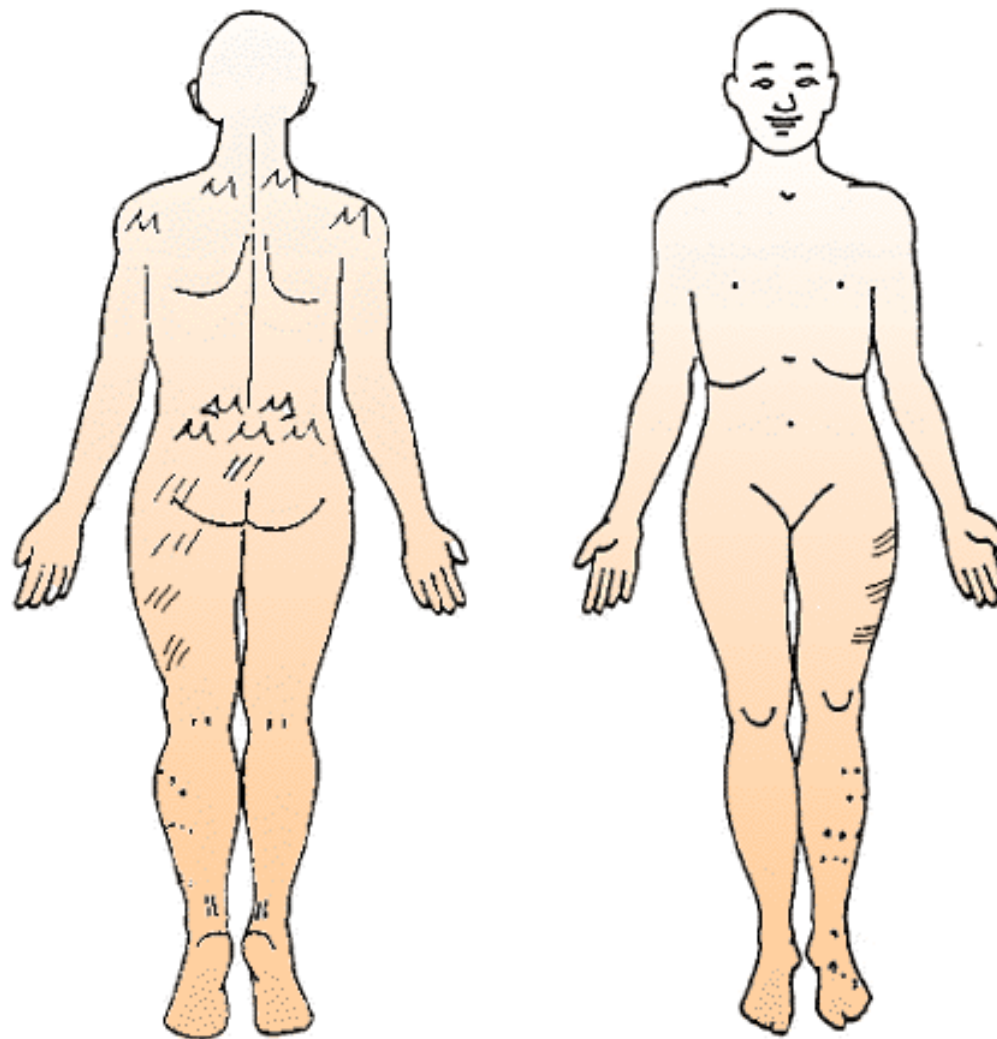
SMÄRTANAMNES

- Smärtdebut
- Smärtduration
- Smärtans karaktär
- Smärtans svårighetsgrad
- Lokalisation
- Utstrålning
- Annan anamnes (tidigare sjukdom)



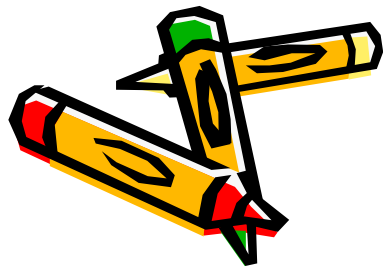
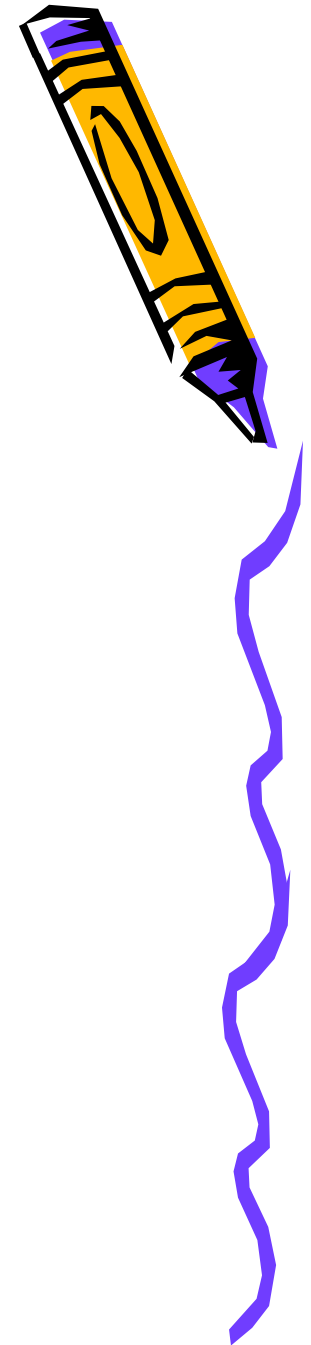
SMÄRTTECKNING

| | |
|------|-----------|
| MMM | Molande |
| XXXX | Brännande |
| == | Ilande |
| /// | Huggande |
| | Stickande |
| BBB | Bultande |
| TTT | Tryckande |
| SSSS | Övrigt |



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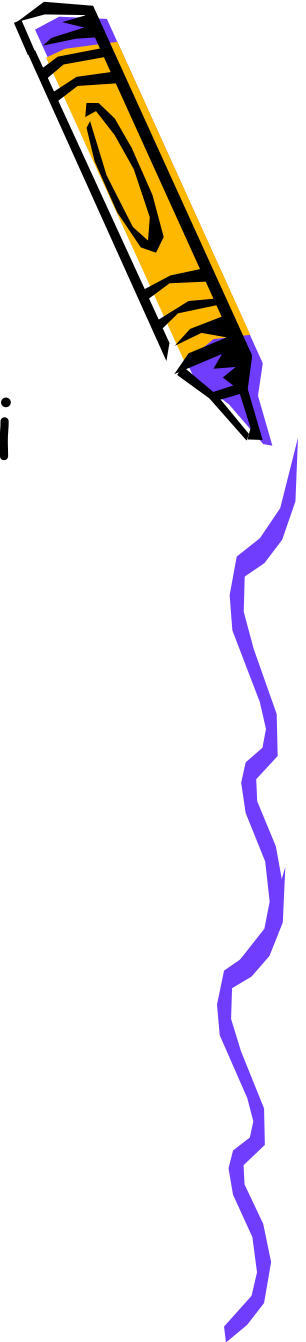
SMÄRTANALYS



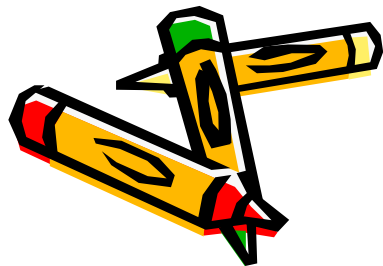
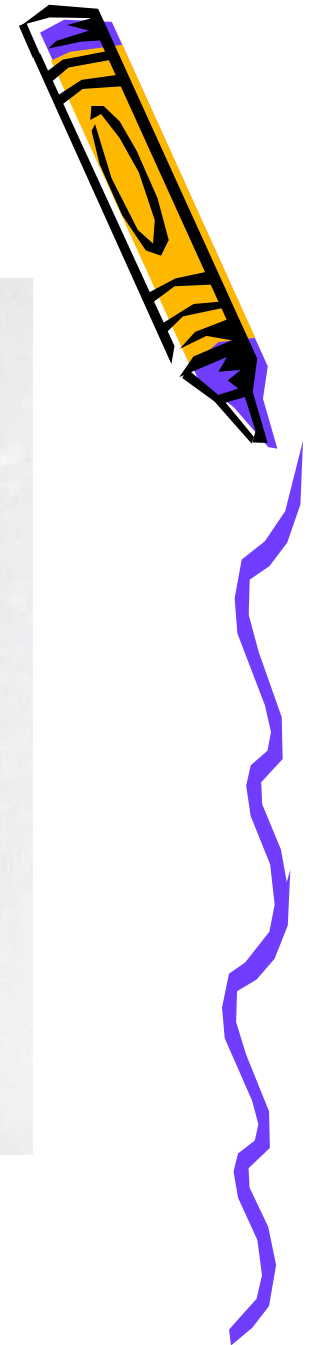
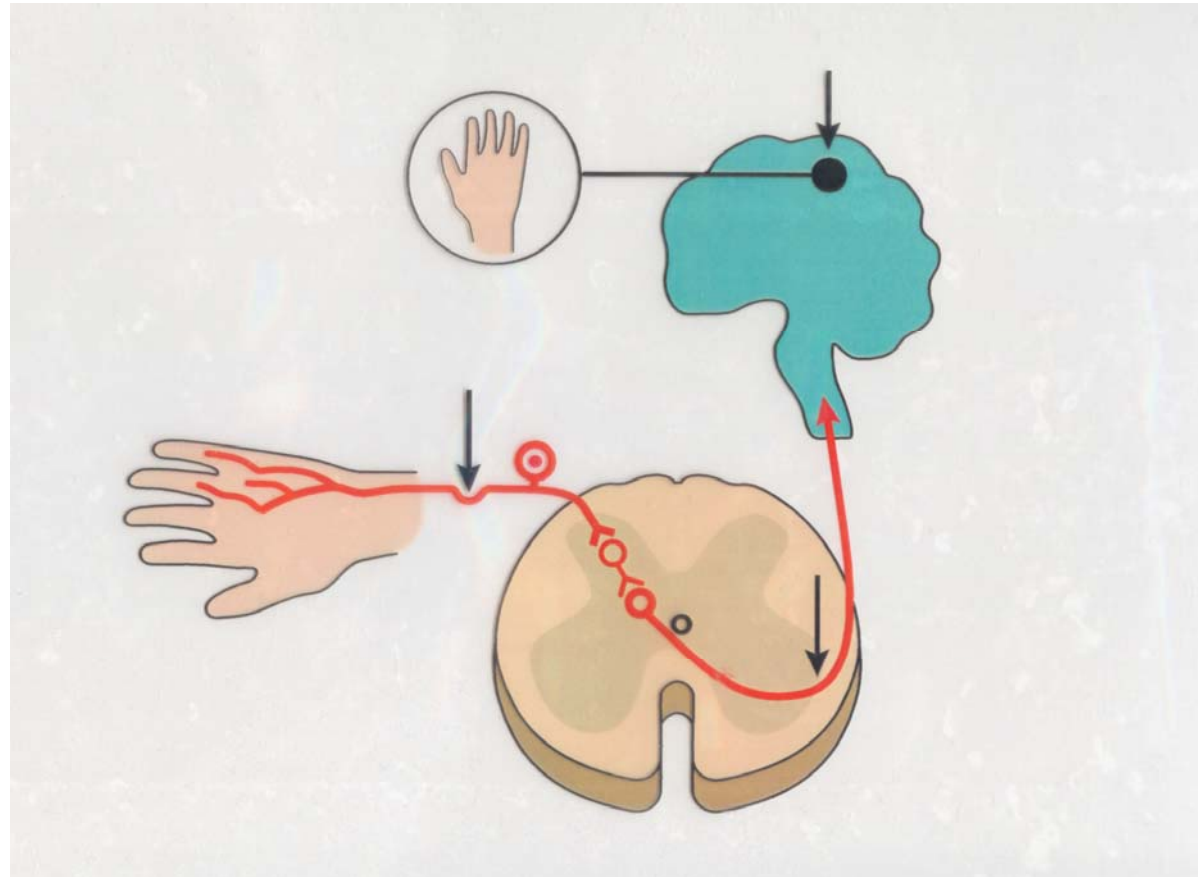
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Diagnostiska kriterier (Neuropatisk smärta)

- Sjukhistoria talande för rubbning i nervsystemet
- Neuroanatomisk korrelation av smärtutbredningen
- Nervdysfunktion

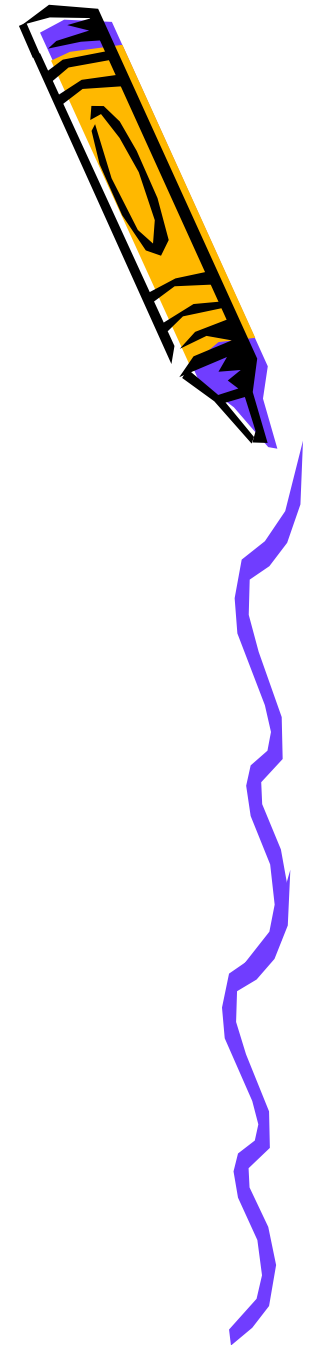


Projicerad smärta



Klinisk undersökning

- **Sensorisk bedside diagnostik:**
 - **Beröring** (målarpensel) $A\beta$ -fibrer
 - **Temperatur**
 - **Värme** (varm metallrulle, 40 grader) C -fibrer
 - **Kyla** (rumstemp. metallrulle) $A\delta$ -fibrer
 - **Smärta** (nålstick) $A\delta$ -fibrer, C -fibrer



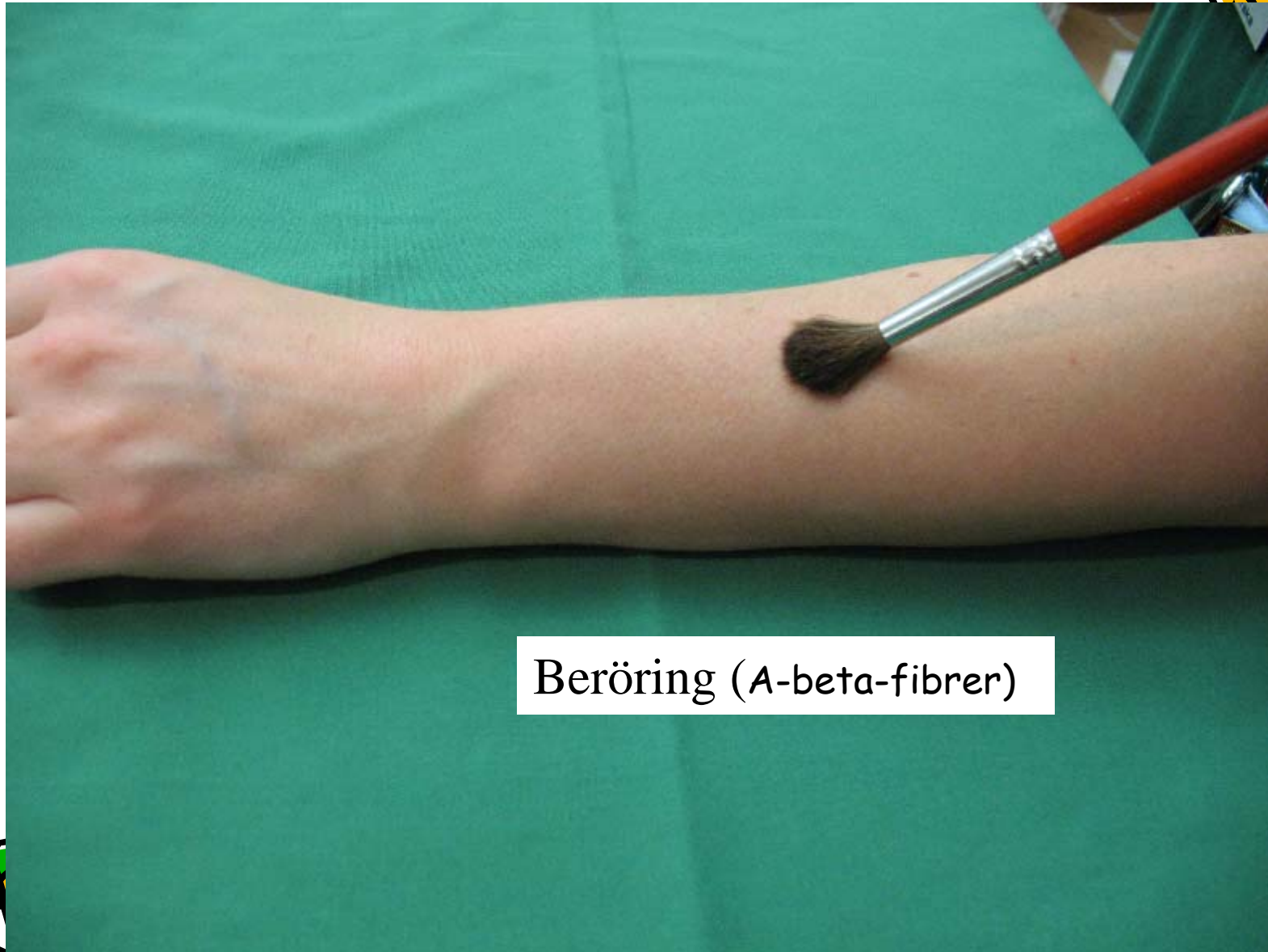


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Sensibilitetsundersökning

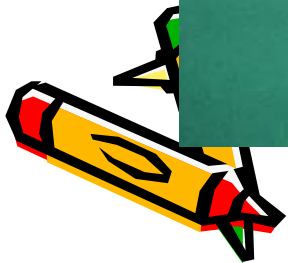
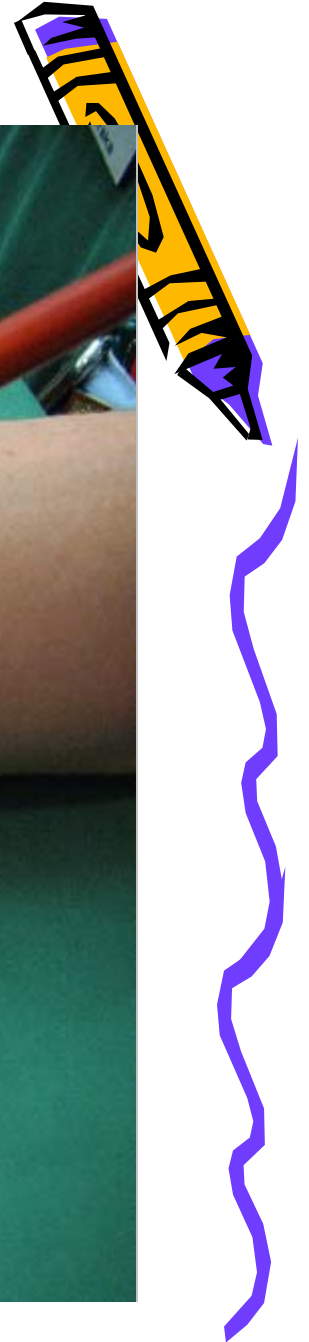
- *Rikta undersökningen* mot område med förväntande positiva fynd
- Undersök **alla modaliteter** beröring, värme, kyla och smärta
- **Jämför** med "frisk" sida
- Använd **protokoll** (sensibilitetsschablon el liknande)

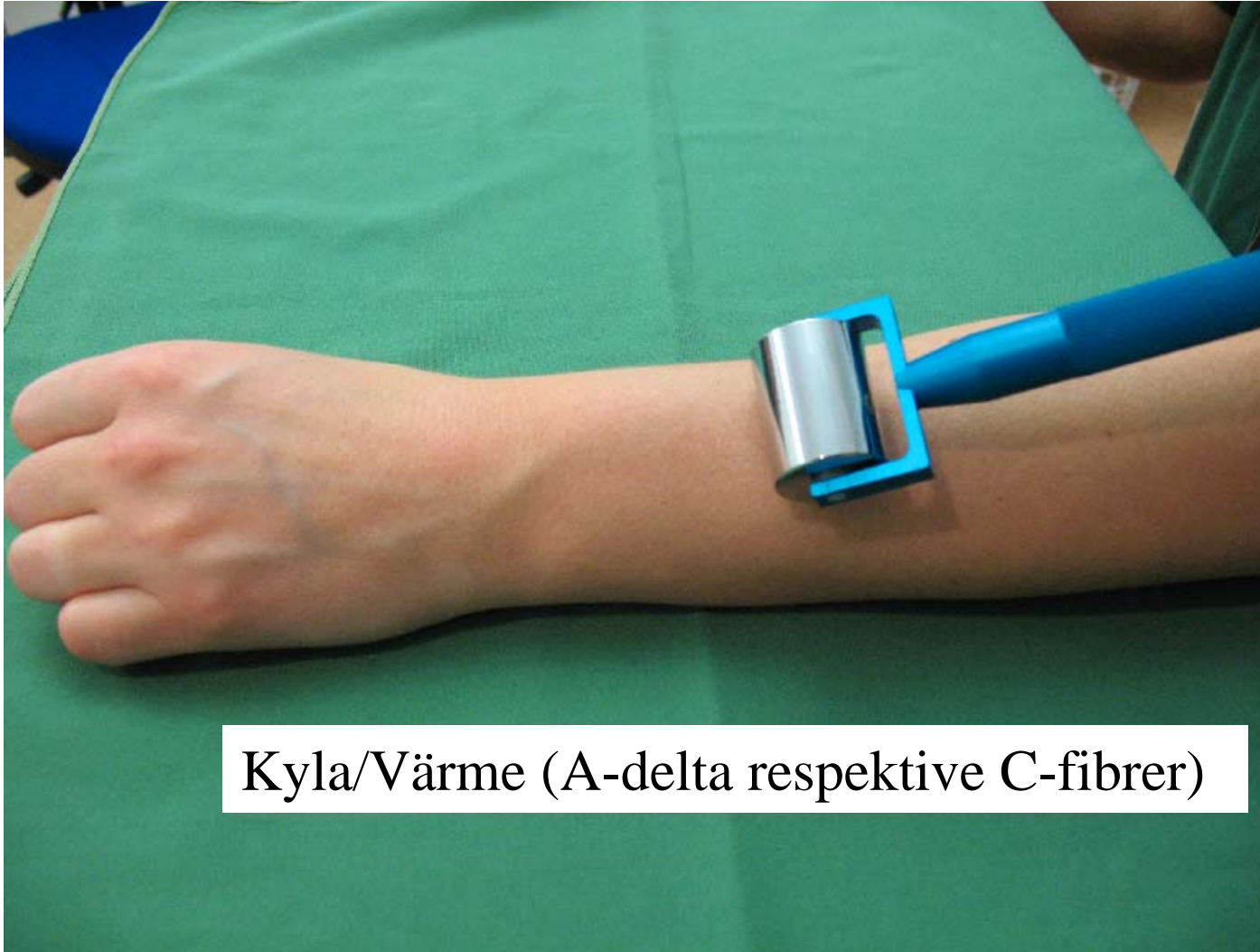




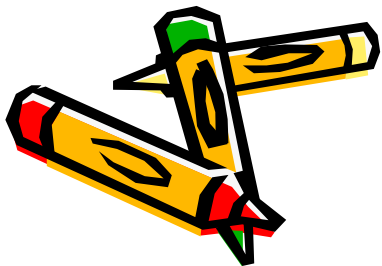
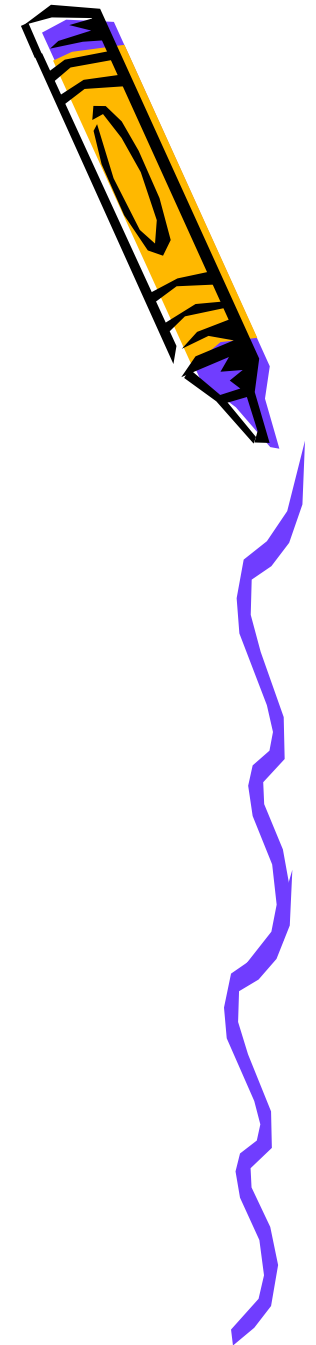
Beröring (A-beta-fibrer)

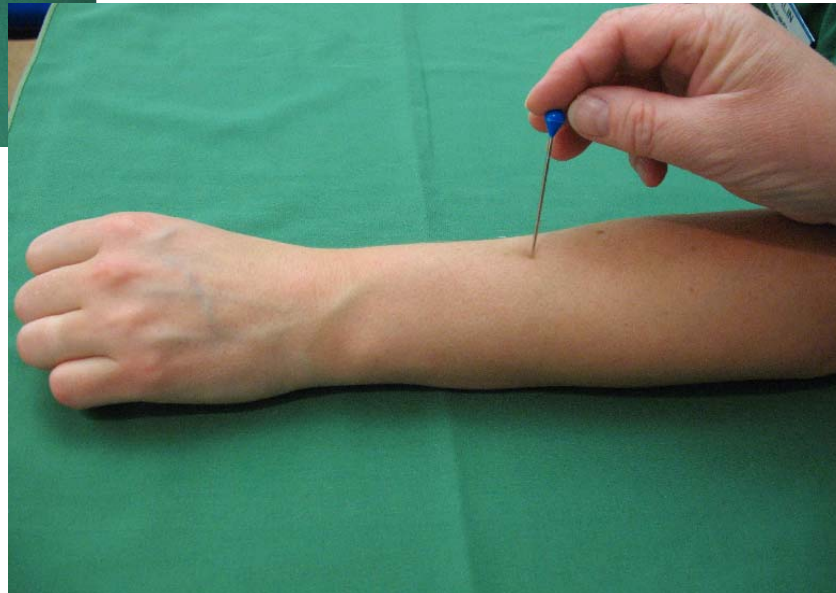
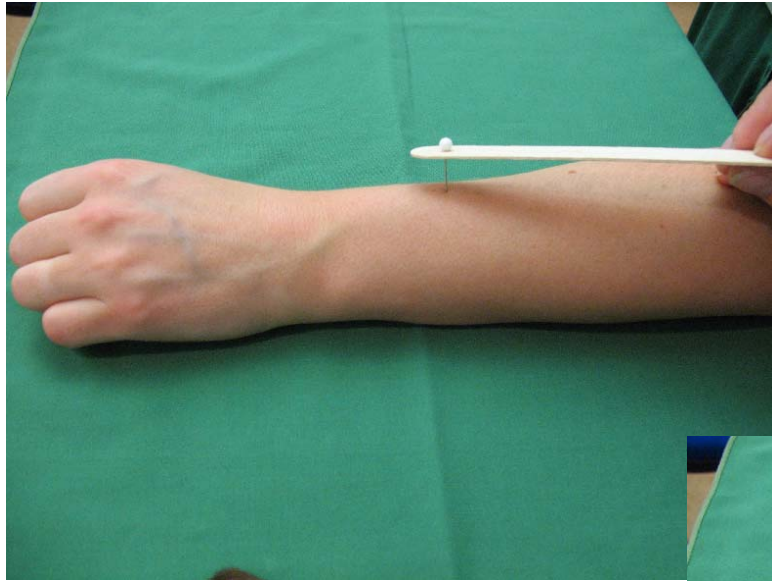
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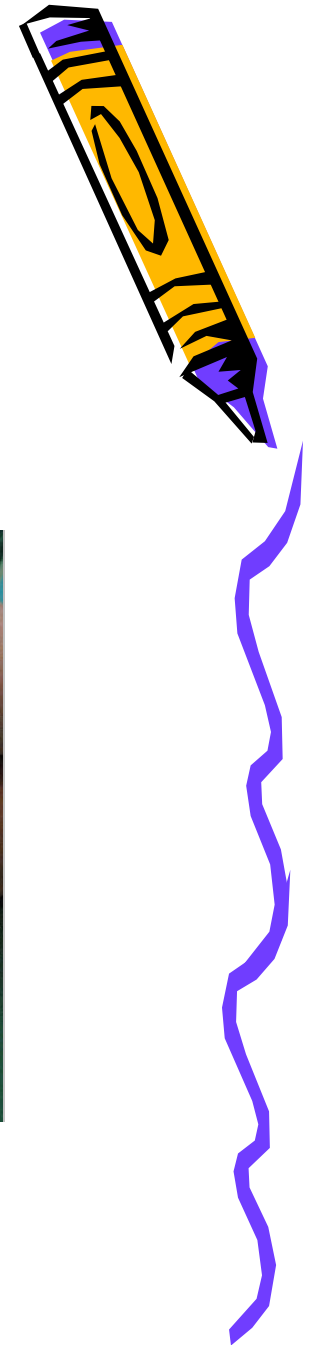


Kyla/Värme (A-delta respektive C-fibrer)





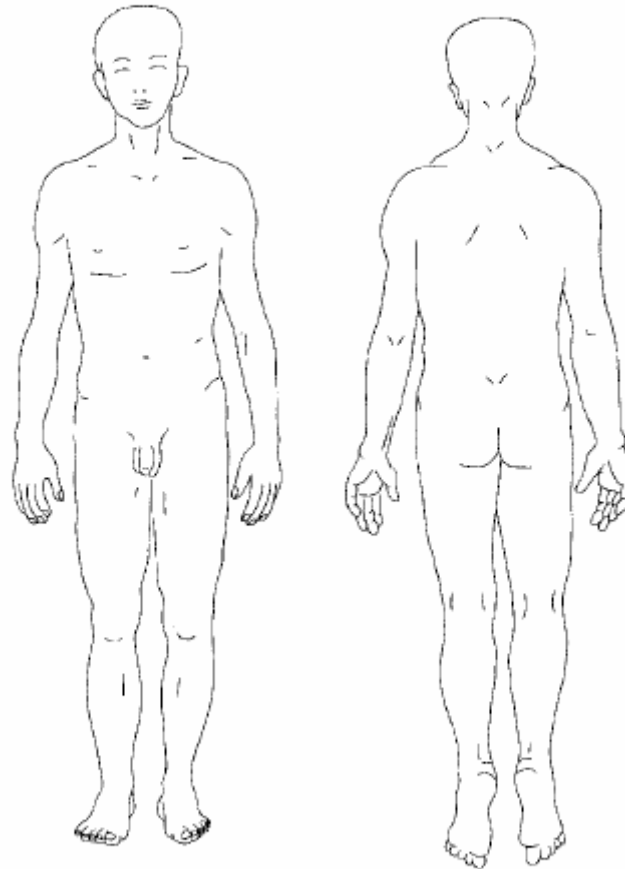
Smärta (nålstick)
(A-delta och C-fibrer)


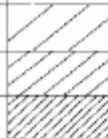









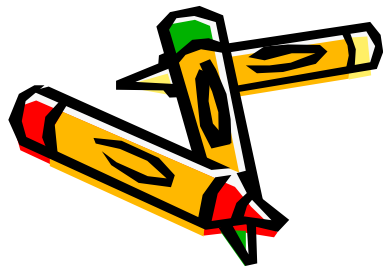
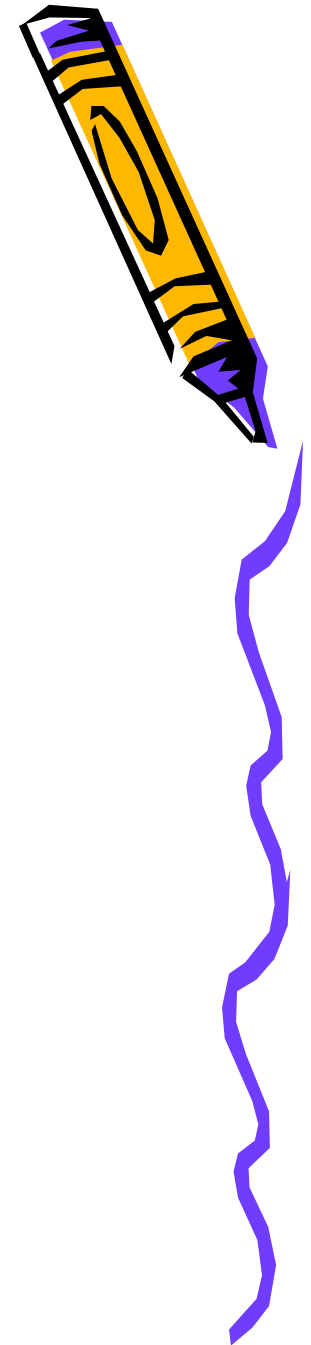
Sensibilitetsschablon

Platz: _____ Datum: _____

Name: _____

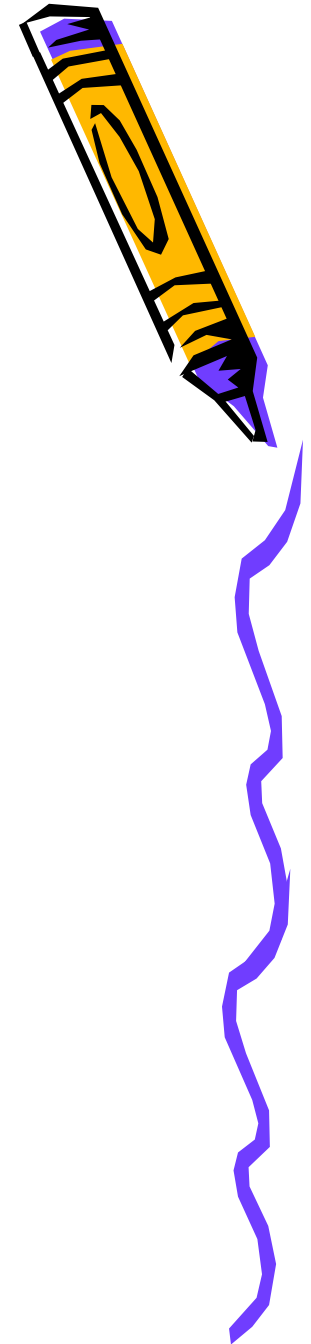


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| Lätt nedsatt |  |  |  |
| Måttigt nedsatt |  |  |  |
| Kraftigt nedsatt |  |  |  |



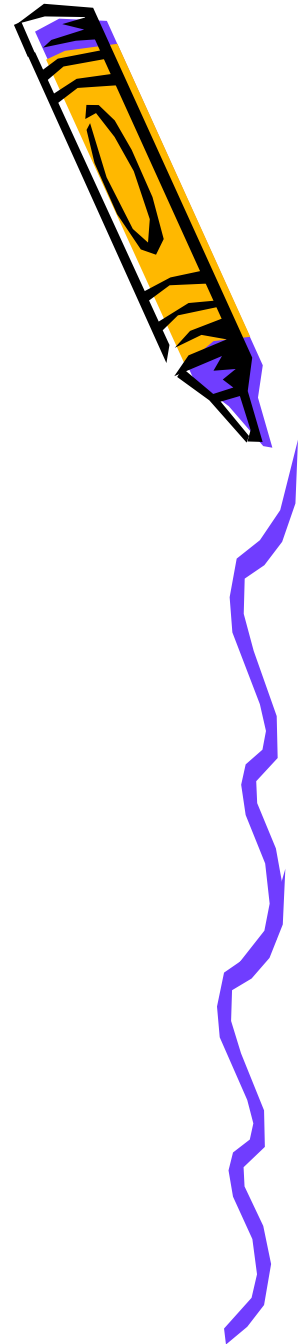
Terminologi

- Analgesi
 - Ingen smärta vid smärtretning
- Hypoalgesi
 - Minskad smärtekänslighet
- Hyperalgesi
 - Ökad smärtekänslighet



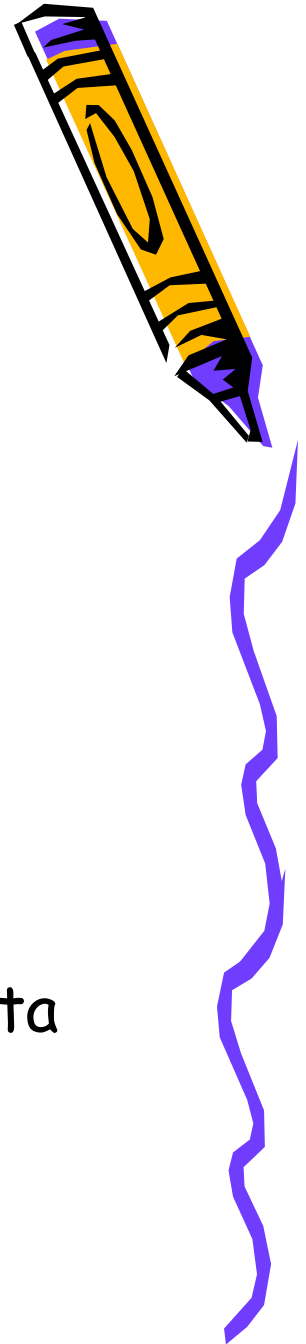
Terminologi forts.

- Hypoestesi
 - Minskad känsel
- Hyperestesi
 - Ökad känsel
- Dysestesi
 - Onormal spontan/stimulusutlöst obehaglig upplevelse



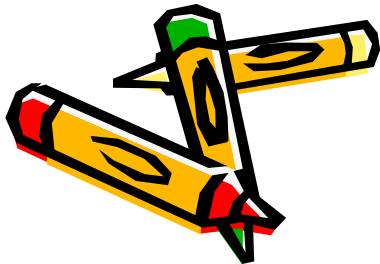
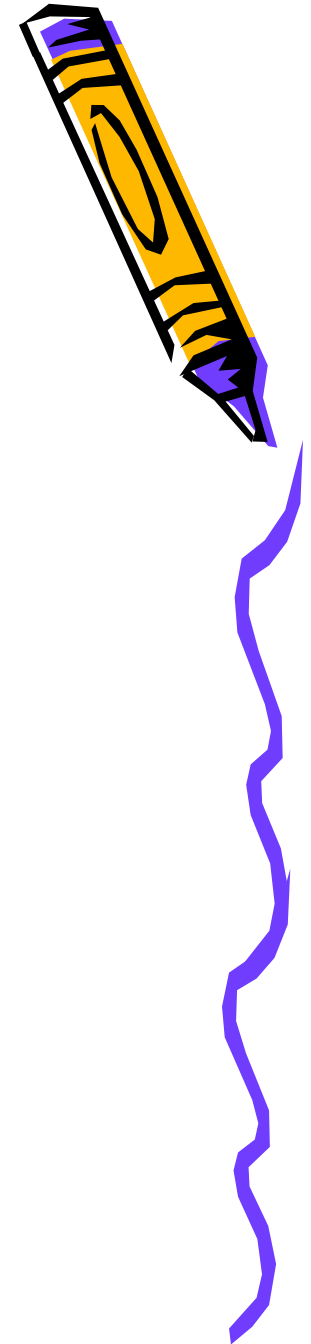
Terminologi forts.

- Parestesi
 - Onormal spontan/stimulusutlöst upplevelse
- Neuralgi
 - Smärta i en eller flera nervers utbredningsområde
- Allodyni
 - En normalt icke smärtsam retning ger smärta (Beröring/temperatur)



Känslrubbingar

- Kvantitativa (hypoestesi, hyperestesi,..)
- Kvalitativa (allodyni, dysestesi, parestesi,..)
- Spatials (abnorm lokalisation)
- Temporala (abnorm latens, eftersensation)



Hyperalgesi

- Primär
 - Ökad känslighet för icke smärtsam termisk/mechanisk stimulering (allodyni)
 - Sensitisering av nociceptorer
 - Prostaglandiner viktig faktor (inflammation)
- Sekundär
 - Ökad mekanisk känslighet (ömhet)
 - Central sensitisering
 - Ökad Calcium nivå intracellulärt (?)





Pain 77 (1998) 227-229

PAIN

Editorial

Towards a mechanism-based classification of pain?

Clifford J. Woolf*, Gary J. Bennett, Michael Doherty, Ronald Dubner, Bruce Kidd, Martin Koltzenburg, Richard Lipton, John D. Loeser, Richard Payne, Eric Torebjork

Received 18 May 1998; accepted 1 June 1998

It is self evident that the recent explosive growth in our understanding of the molecular, cellular and system's mechanisms responsible for nociception and pain has important implications for the clinical diagnosis and treatment of pain. A small group of independent basic scientists and clinicians met in New York in January 1998, for a wide ranging discussion on the possible need for and implications of a mechanism-based classification of pain. The group believed that acceptance of a mechanism-based classification could have profound implications: drugs may be developed which target distinct mechanisms, basic scientists may have new guidelines for experimental design, and clinicians may be eventually armed with more reliable and valid diagnostic tools for treatment and clinical investigation. Furthermore, a mechanism-based classification for clinical syndromes might generate testable hypotheses for selecting treatments which interact with specific mechanisms. We wish to initiate a wide debate on this important topic by highlighting what we consider to be some of the key issues.

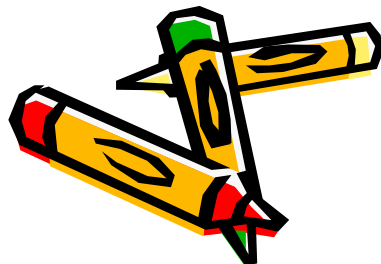
In general, taxonomies can be either natural or artificial. Examples of each, respectively, are the division of objects into animate or inanimate groups (which reflects order in nature) and a telephone book (which is merely a conventional way of listing peoples' numbers and addresses). Natural taxonomies are based on theoretical ideas of how the world is organized. Artificial taxonomies provide convenient or practical methods for organizing the world. Consequently they do not easily facilitate the development of new ideas.

A mechanism-based classification of pain requires a conceptual understanding of organization in nature, and would,

therefore, set a framework for scientific development. Current methods of classifying pain have, we believe, a number of major limitations. Pain syndromes are identified by parts of the body, duration, and causative agent. We believe that an anatomical-based classification of pain is limiting because the innervation of distinct anatomical regions is often analogous, bearing in mind differences of target organ innervated (e.g. skin vs. viscera), length of axon, myelination, etc. To the extent that universal mechanisms can be identified, anatomical differences should be disregarded in favor of mechanisms that apply to all parts of the body. The acute/chronic dichotomy is also not helpful. Acute and chronic do not readily differentiate mechanisms. The benign/malignant dichotomy too has no mechanistic basis for pain, although it will influence treatment strategies. Greater care needs also to be taken with the definition of terms such as allodynia and hyperalgesia. Both terms are a description of clinical symptoms and do not imply a mechanism. Allodynia (pain evoked by normally non-painful stimuli) is often used in the clinical context to refer to A β -fiber mediated brush-evoked mechanical pain or an altered processing of sensory information in the CNS. However, there are several other distinct types of mechanical hypersensitivity that do not involve A β fibers and probably no significant central reorganization, but which present as pain evoked by commonly non-painful stimuli. Reduction in threshold is not, therefore, useful by itself, for a mechanistic classification.

From a practical perspective, clinicians use classification systems to predict treatment responses as well as prognosis and to search for risk factors and morbidities. Ultimately any classification system must be judged on its utility for clinical practice and research. The most powerful systems could be organized by mechanism, by disease or etiology. In the neuropathic disease category, at least, the disease classification system was considered by the group to fail to

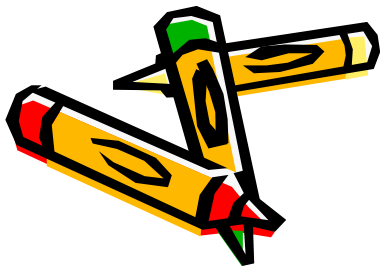
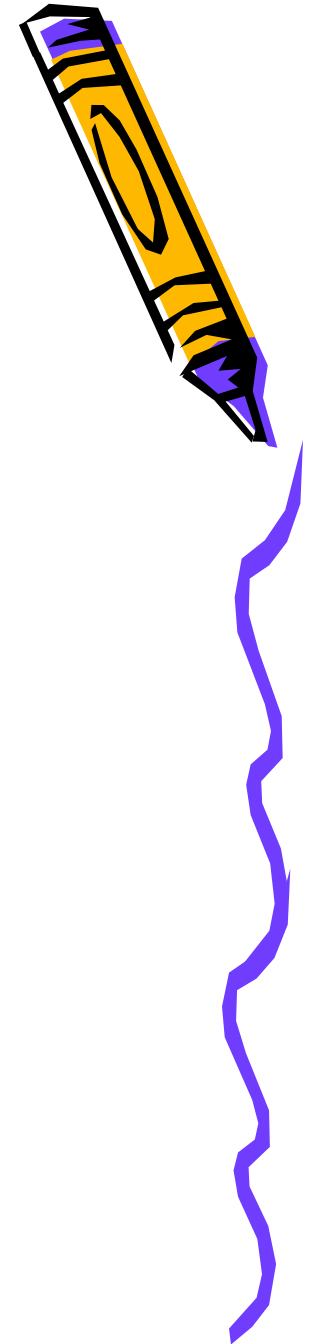
* Corresponding author. Neural Plasticity Research Group, Department of Anesthesia and Critical Care, Massachusetts General Hospital, 149 13th Street, Room 4903, Charlestown, MA 02129, USA. Tel.: +1 617 7243622; fax: +1 617 7243632.



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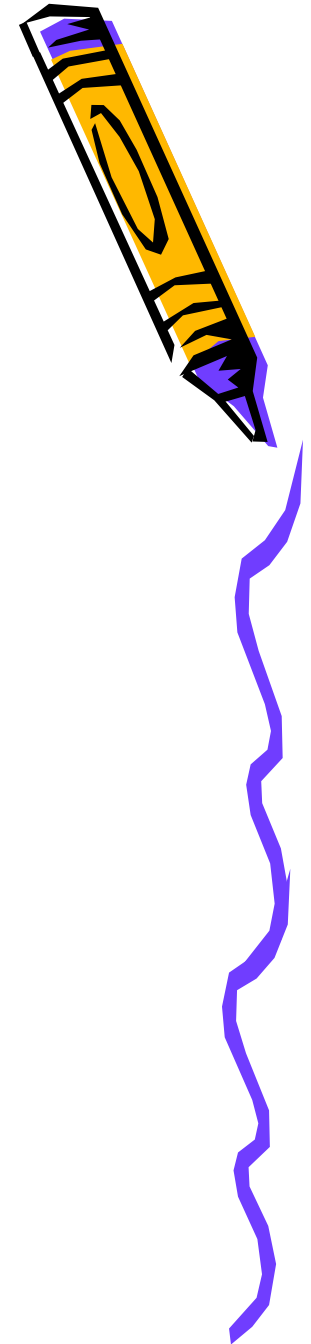
Smärtkategorier och tänkbara mekanismer

- Övergående smärta
 - Nociceptor-aktivering
 - Utan långtidseffekt
(jämför "nålstick-smärta")



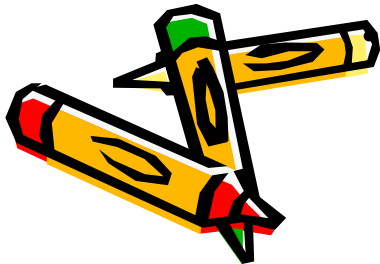
Smärtkategorier och tänkbara mekanismer

- Smärta vid vävnadsskada
 - Primär afferenten
 - Sensitisering
 - "Silent nociceptor" rekrytering
 - Fenotyps förändring
 - CNS medierad
 - Central sensitisering, "wind-up"



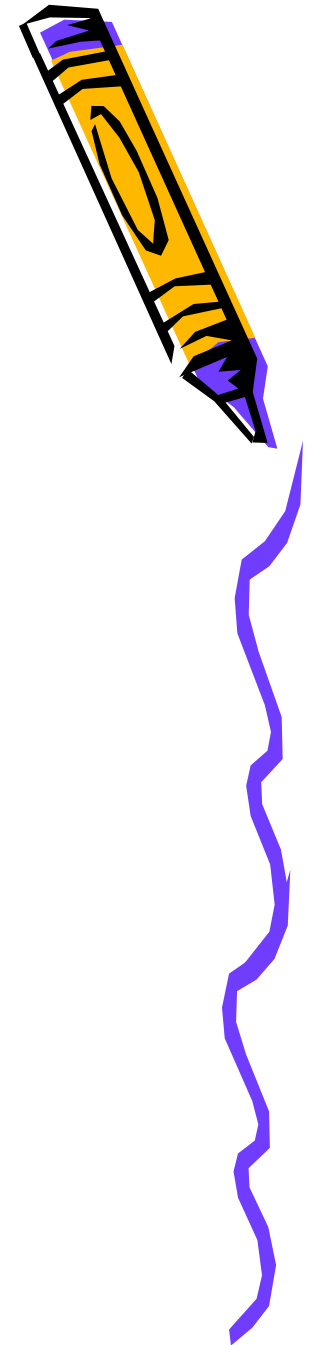
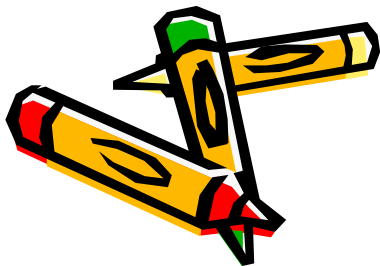
Smärtkategorier och tänkbara mekanismer

- Smärta vid nervskada
 - Primär afferenten
 - Spontan och stimulusutlöst nervaktivitet på annat ställe än vid nociceptorn
 - Fenotyps förändring
 - CNS medierad
 - Central sensitisering,
 - deafferentering,
 - disinhibition,
 - strukturell reorganisation

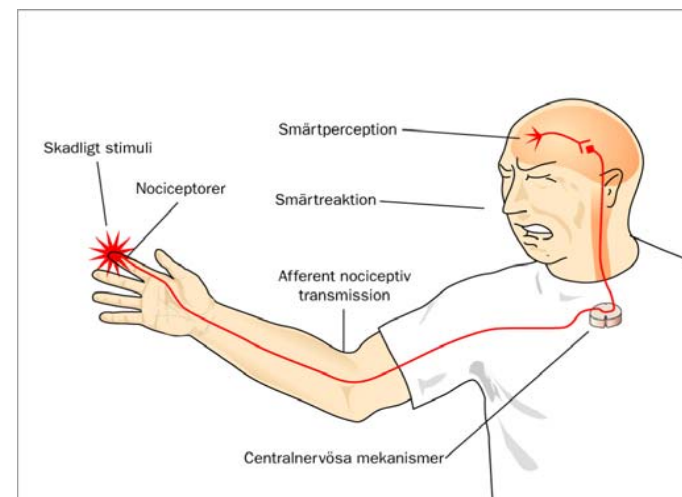
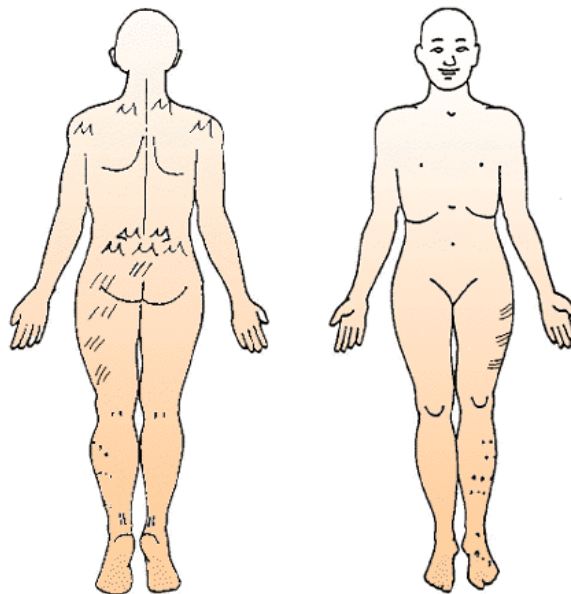
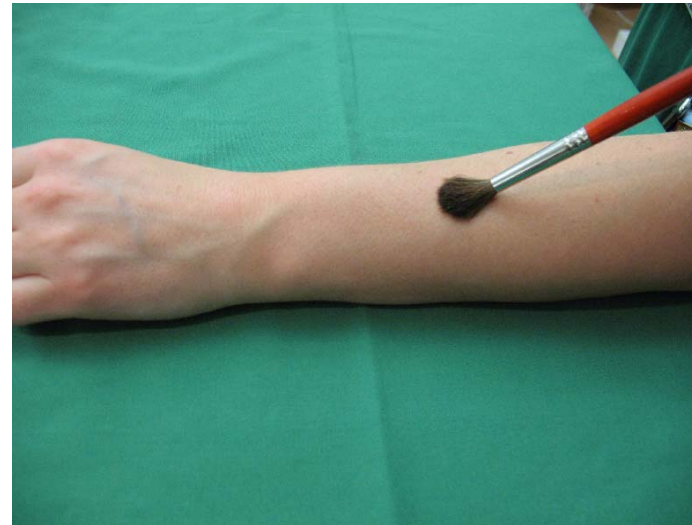


Neuropatisk smärta många orsaker - riktad terapi ??

- Mekanisk
- Infektiös
- Vaskulär
- Cancer
- Metabolisk
- Nutritiv
- Toxisk
- Övrigt



Smärtanalys



Strandbaden 070419

