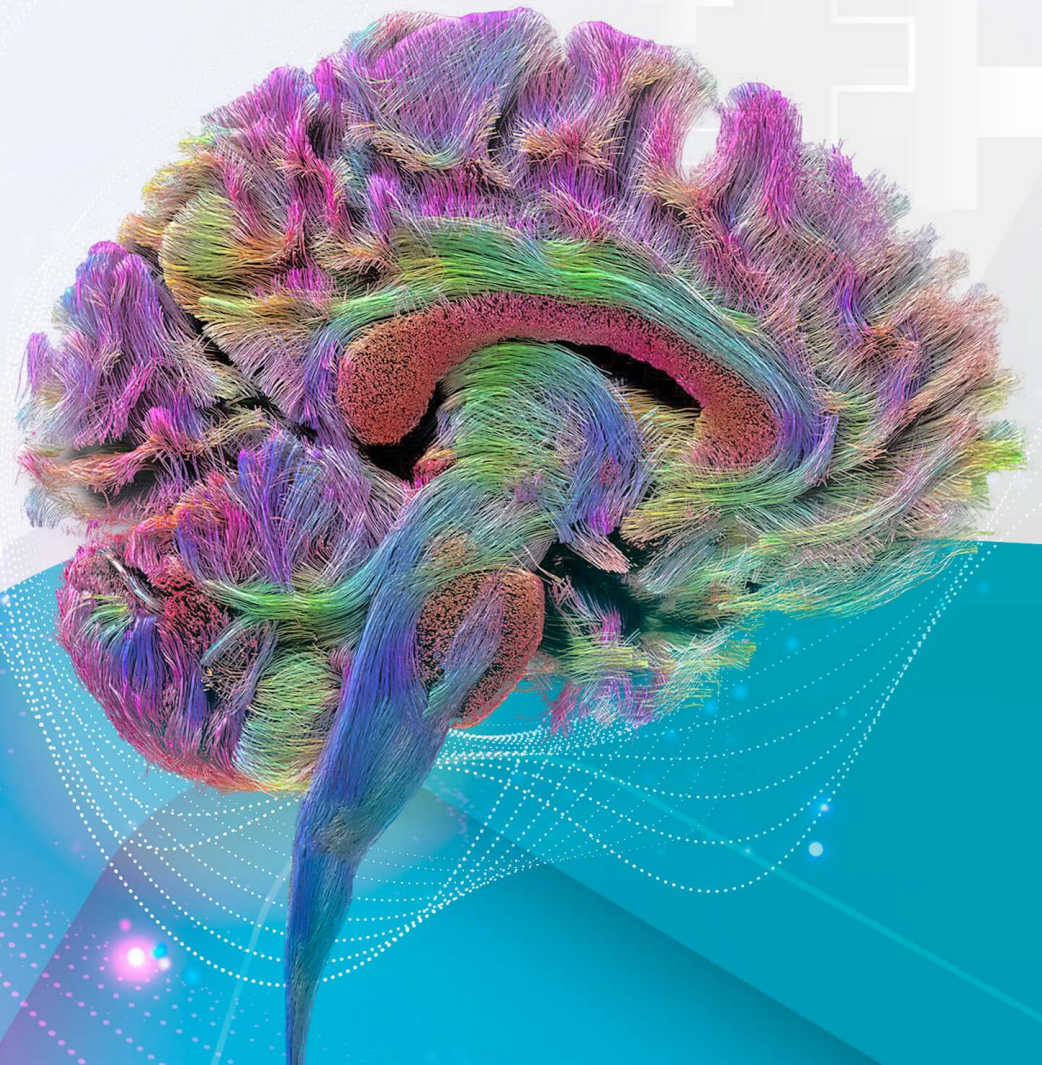


QUESTIONADOR

BASED ON MODERN PAIN NEUROSCIENCE

MASTER'S EDUCATION IN HEALTH SCIENCES AND THE ENVIRONMENT - MECSMA (UniFOA)

→START

CREDITS | PAIN EDUCATION | CURIOSITIES

Authors:
Gustavo Ferraz Cardozo
Carlos Alberto Sanches Pereira

QUESTIONADOR AS AN EDUCATIONAL PRODUCT

Hello, The QuestionaDor Quiz proposes a teaching product based on the modern neurophysiology of pain. The proposal is based on the theory of meaningful learning (SAD) through prior knowledge of the student, cognistivism proposed by theorist David Ausubel, using subsumers as an educational tool in the teaching of new evidence in the neuroscience of pain. The Quiz has 35 questions of multiple choices, where the student has 4 alternatives, being only 1 correct. By marking the correct answer, it has a broader explanation of the question, within the biopsychosocial context integrated with pain education (PNE – Pain Neuroscience Education). By pointing out the wrong alterative, you will have a digital teaching resource (podcast and / or video) that helps you to choose the correct alternative, based on scientific articles on new concepts in pain. The Quiz has questions about the neurophysiology of acute and chronic pain, pain education, pain epidemiology, neurophysiological mechanisms involved with pain, classification and sub-classification of chronic pain, modern pain assessment and clinical cases involving pain. The Quiz was developed as an educational product of the Master's Degree in Teaching in Health and Environmental Sciences (MECSMA) of the Oswaldo Aranha Foundation - University Center of Volta Redonda - UniFOA.

continue

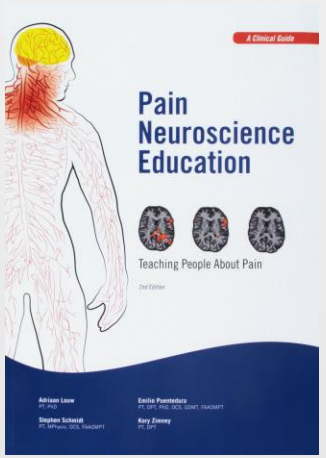
Menu

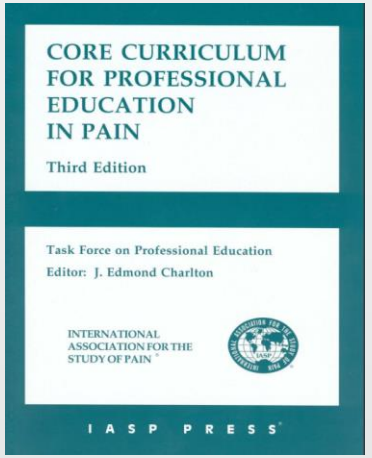
QUESTIONADOR AS AN EDUCATIONAL PRODUCT

continue

Menu

QUESTIONADOR AS EDUCATION IN PAIN





continue

Menu

QUESTIONADOR AS EDUCATION IN PAIN

IASP

FOR IMMEDIATE RELEASE

World Health Assembly of the WHO Approves 11th Version of the International Classification of Diseases (ICD-11), Including New Diagnostic Codes for Chronic Pain

IASP Task Force worked closely with World Health Organization to develop new classification system of chronic pain for improved patient care and research

WASHINGTON, DC – June 3, 2019 – The World Health Organization (WHO) has adopted ICD-11, the latest revision of its International Classification of Diseases, including a new classification system for chronic pain. The decision was made at the World Health Assembly on 25 May 2019.

Topical Review

PAIN

Do we need a third mechanistic descriptor for chronic pain states?

Eva Kosek[a,*], Milton Cohen[b], Ralf Baron[c], Gerald F. Gebhart[d], Juan-Antonio Mico[e], Andrew S.C. Rice[f], Winfried Rief[g], A. Kathleen Sluka[h]

continue

Menu

DEVELOPERS

GUSTAVO FERRAZ CARDOZO – AUTHOR

Graduated in Physical Therapy, Specialist in Sports Physical Therapy by the Federal Council of Physical Therapy and National Society of Sports Physical Therapy (COFFITO/SONAFE), Osteopath CO – Escuela de Osteopatia de Madrid, Post-Graduate in Functional Cardiorespiratory Physiotherapy, Post-Graduate in Acupuncture, currently attending the Professional Master's Degree in Teaching in Health Sciences and Environment (MECSMA) of UniFOA, public servant in the municipality of Itatiaia/RJ, active in the Municipal Center of Rehabilitation and Physical Therapy, Professor of the Postgraduate Course in Functional Traumato-Orthopedic Physical Therapy of the University Center of Barra Mansa (UBM), Post-Graduation in Musculoskeletal Rehabilitation by the Movement of the Educational Phase (RPX) and Post-Graduation in Exercise Physiology Applied to Training of the Geraldo Di Biasi University Center (UGB).

CARLOS ALBERTO SANCHES PEREIRA - ADVISOR

Graduated in Biological Sciences, Specialist in Biochemistry, Specialist in Hematology from UFRJ in 2000, Master in Food Science and Technology from UFRRJ (2001) in the area of concentration in Applied Microbiology; PhD in Industrial Biotechnology (2007) EEL-USP in the area of concentration in Applied Microbiology. He has experience in biotechnology of microorganisms: studies with Lactobacillus and its role in the stimulation of immunity; Clinical and Medical Microbiology; Clinical and Laboratory Hematology. Professor/Advisor of the Professional Master's Degree in Teaching of Health and Environmental Sciences of UniFOA, a program in which he develops studies related to the use of recreational activities as a tool for teaching in Biological Sciences and Health. It also studies the epidemiological aspects of bacteria isolated from Otitis in Dogs, and their relationship with the therapeutic conduct and with medical education.

continue

Menu

1 - According to IASP updates in recent years, on the concept of pain and nociception, we can say that:

A — are synonyms;

B — are pathways of the nociceptive pathway;

C — are distinct phenomena;

D — pain is determined by the activity of sensory neurons.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

Text box 1. IASP definition of pain (1979).

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note

Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience which we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain, eg, pricking, but are not unpleasant, should not be called pain. Unpleasant abnormal experiences (dysaesthesiae) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.

Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.

Text box 2. Revised IASP definition of pain (2020).

Pain

An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.

Notes

- Pain is always a personal experience that is influenced to varying degrees by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience as pain should be respected.*
- Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
- Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.

Etymology

Middle English, from Anglo-French peine (pain, suffering), from Latin poena (penalty, punishment), in turn from Greek poinē (payment, penalty, recompense).

*The Declaration of Montréal, a document developed during the First International Pain Summit on September 3, 2010, states that "Access to pain management is a fundamental human right."

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

Text box 1. IASP definition of pain (1979).

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note

Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience which we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain, eg, pricking, but are not unpleasant, should not be called pain. Unpleasant abnormal experiences (dysaesthesiae) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.

Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.

Text box 2. Revised IASP definition of pain (2020).

Pain

An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.

Notes

- Pain is always a personal experience that is influenced to varying degrees by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience as pain should be respected.*
- Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
- Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.

Etymology

Middle English, from Anglo-French peine (pain, suffering), from Latin poena (penalty, punishment), in turn from Greek poinē (payment, penalty, recompense).

*The Declaration of Montréal, a document developed during the First International Pain Summit on September 3, 2010, states that "Access to pain management is a fundamental human right."

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

Text box 1. IASP definition of pain (1979).

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note

Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience which we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain, eg, pricking, but are not unpleasant, should not be called pain. Unpleasant abnormal experiences (dysaesthesiae) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.

Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.

Text box 2. Revised IASP definition of pain (2020).

Pain

An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.

Notes

- Pain is always a personal experience that is influenced to varying degrees by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience as pain should be respected.*
- Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
- Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.

Etymology

Middle English, from Anglo-French peine (pain, suffering), from Latin poena (penalty, punishment), in turn from Greek poinē (payment, penalty, recompense).

*The Declaration of Montréal, a document developed during the First International Pain Summit on September 3, 2010, states that "Access to pain management is a fundamental human right."

return

Menu

CERTAIN ANSWER!

Pain and nociception are different phenomena: the experience of pain cannot be reduced to activity in the sensory pathways. According to the latest IASP update, pain is: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" is very clear that it requires subjectivity, which in turn requires awareness and the ability to evaluate a stimulus/situation."

Narrative Review

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

Text box 1. IASP definition of pain (1979).

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note

Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience which we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain, eg, pricking, but are not unpleasant, should not be called pain. Unpleasant abnormal experiences (dysaesthesiae) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.

Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.

Text box 2. Revised IASP definition of pain (2020).

Pain

An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.

Notes

- Pain is always a personal experience that is influenced to varying degrees by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience as pain should be respected.*
- Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
- Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.

Etymology

Middle English, from Anglo-French peine (pain, suffering), from Latin poena (penalty, punishment), in turn from Greek poinē (payment, penalty, recompense).

*The Declaration of Montréal, a document developed during the First International Pain Summit on September 3, 2010, states that "Access to pain management is a fundamental human right."

continue

Menu

2 - The nociceptor is a high-threshold sensory receptor of the peripheral somatosensory nervous system capable of transducing and encoding harmful stimuli. They are basically classified by their diameter and degree of myelination, which determines their degree of conduction speed. There are 2 (two) main classes of nociceptors, they are:

A

The A-Delta are myelinated and of medium diameter, and are responsible for the transmission of rapid pain (5 to 30 m/s – unimodal, mechanical and thermal) and the Type C are amyelinic and of small diameter, which transmit nociception slowly (less than 1 m/s – polymodal, mechanical, thermal and chemical);

B

The A-Delta are amyelinated and small in diameter, are responsible for the slow transmission of pain (less than 1m/s) and the Type C are myelin and large diameter and transmit nociception quickly (5 to 30m/s);

C

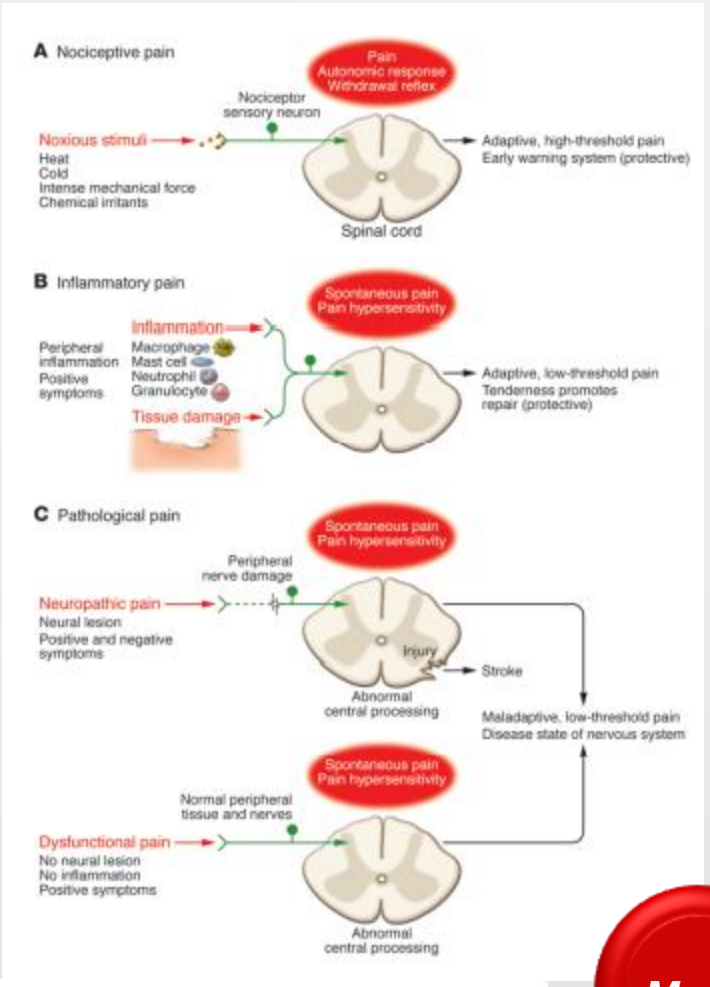
The A-Gamma are amyelinated and small in diameter, are responsible for the slow transmission of pain (less than 1m/s) and Type A are myelin and large diameter and transmit nociception quickly (5 to 30m/s);

D

The slow neospinothalamic and the fast paleospinothalamic.

Menu

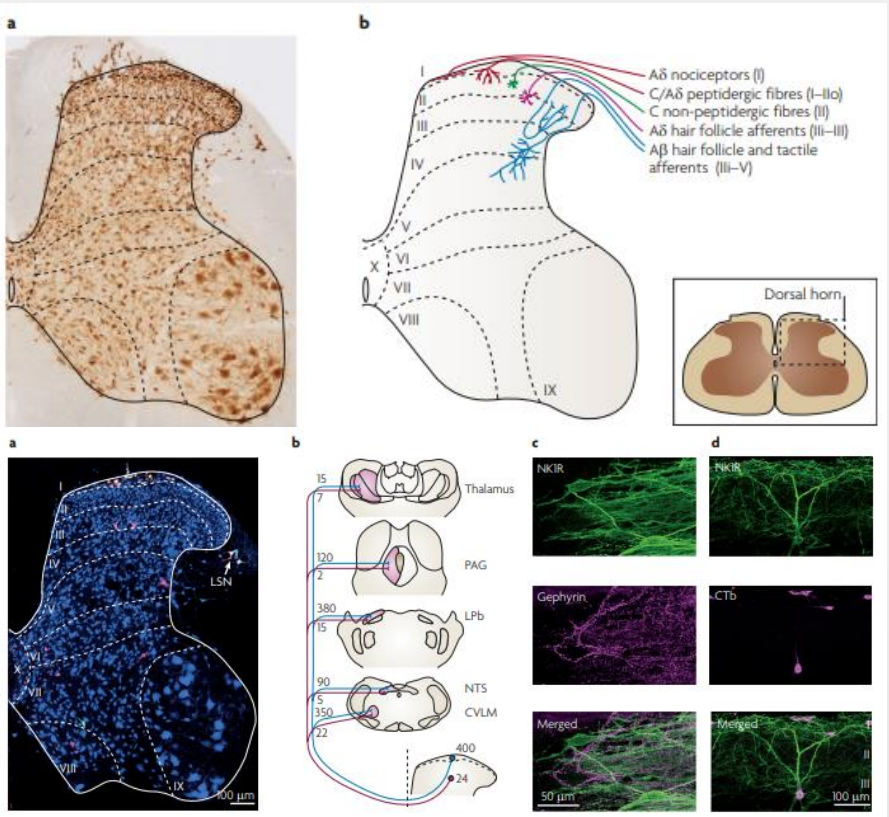
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

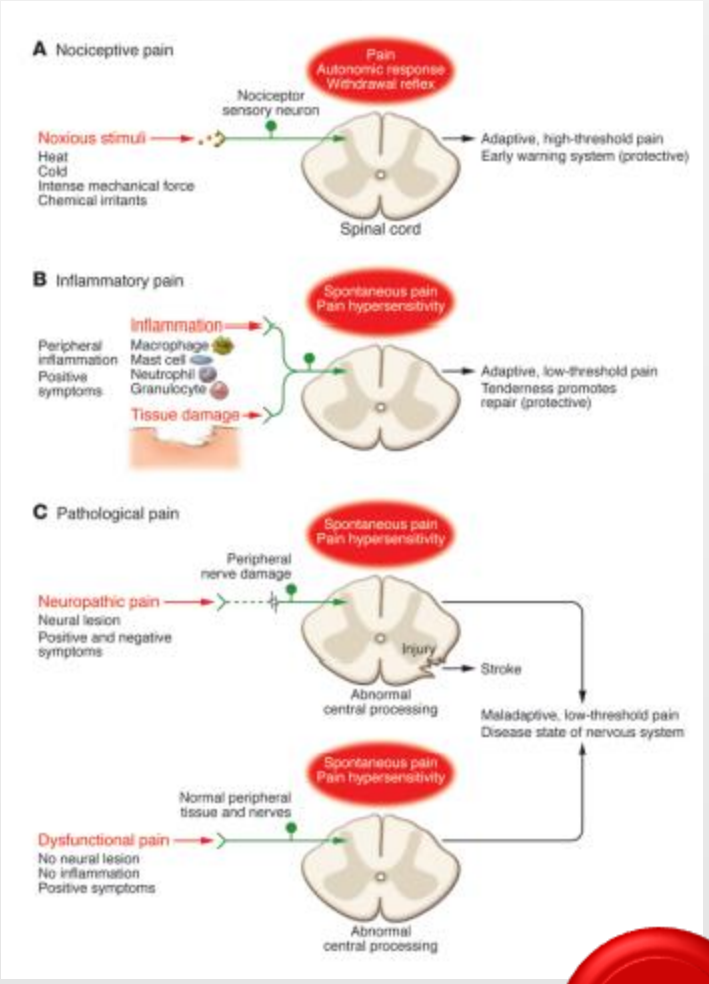
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

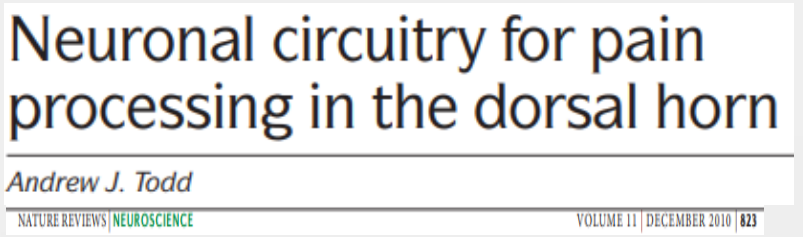
CERTAIN ANSWER!

There are two (2) main classes of nociceptors. The A-Delta are myelinated and of medium diameter, and are responsible for the transmission of rapid pain (5 to 30 m/s); are mechanical and thermal (unimodal;) there is also a division into two functional groups; the first group A-Delta1 respond to mechanical and chemical stimuli and have a high heat threshold, sensitizing in the context of tissue injury; is a pathway that probably respond to the first pain to mechanical stimuli, the fibers of group A-Delta2 have a lower heat threshold and high mechanical threshold involved in rapid pain threshold for example to heat; Type C are amyelinic and small in diameter, which transmit nociception slowly (less than 1 m/s); they are mechanical, thermal and chemical (polymodal); they are classified not functionally, but rather molecular condition based on the receptors and the neurochemistry underlying their expression; thus leading to a wide range of markers studied with the aim of defining neuronal subpopulations and correlating them with the response property of these receptors.


What is this thing called pain?

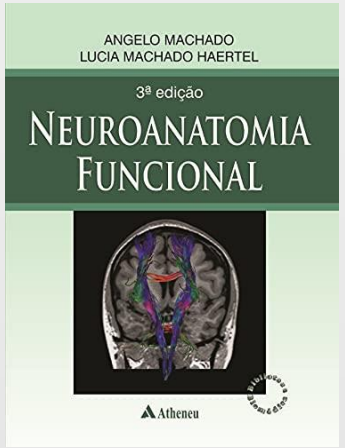
Clifford J. Woolf

Program in Neurobiology and Department of Neurology, Children's Hospital Boston, and Department of Neurobiology, Harvard Medical School, Boston, Massachusetts, USA.


Neuronal circuitry for pain processing in the dorsal horn

Andrew J. Todd

NATURE REVIEWS | NEUROSCIENCE VOLUME 11 | DECEMBER 2010 | 823



continue

Menu

3 – Acute pain and chronic pain are characterized by a condition of a temporal character, where:

A — acute pain has a protective role and chronic pain is a disease;

B — chronic pain is an uncured acute pain;

C — Acute and chronic pain have a protective role;

D — are the result of the byproduct of the activation of free nerve endings.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

ICD-11

Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the *International Classification of Diseases* (*ICD-11*)

Rolf-Detlef Treede[a,*], Winfried Rief[b], Antonia Barke[b], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h,i], Michael B. First[j], Maria Adele Giamberardino[k], Stein Kaasa[l,m,n], Beatrice Korwisi[b], Eva Kosek[o], Patricia Lavand'homme[p], Michael Nicholas[q], Serge Perrot[r], Joachim Scholz[s], Stephan Schug[t,u], Blair H. Smith[v], Peter Svensson[w,x], Johan W.S. Vlaeyen[y,z,aa], Shuu-Jiun Wang[bb,cc]

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

PAIN

When does acute pain become chronic?

C. Voscopoulos and M. Lema*

Department of Anesthesiology, Critical Care, and Pain Medicine, University at Buffalo, Buffalo, NY, USA

* Corresponding author. E-mail: mlema@buffalo.edu

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

ICD-11

Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the *International Classification of Diseases* (*ICD-11*)

Rolf-Detlef Treede[a,*], Winfried Rief[b], Antonia Barke[b], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h,i], Michael B. First[j], Maria Adele Giamberardino[k], Stein Kaasa[l,m,n], Beatrice Korwisi[b], Eva Kosek[o], Patricia Lavand'homme[p], Michael Nicholas[q], Serge Perrot[r], Joachim Scholz[s], Stephan Schug[t,u], Blair H. Smith[v], Peter Svensson[w,x], Johan W.S. Vlaeyen[y,z,aa], Shuu-Jiun Wang[bb,cc]

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

PAIN

When does acute pain become chronic?

C. Voscopoulos and M. Lema*

Department of Anesthesiology, Critical Care, and Pain Medicine, University at Buffalo, Buffalo, NY, USA

* Corresponding author. E-mail: mlema@buffalo.edu

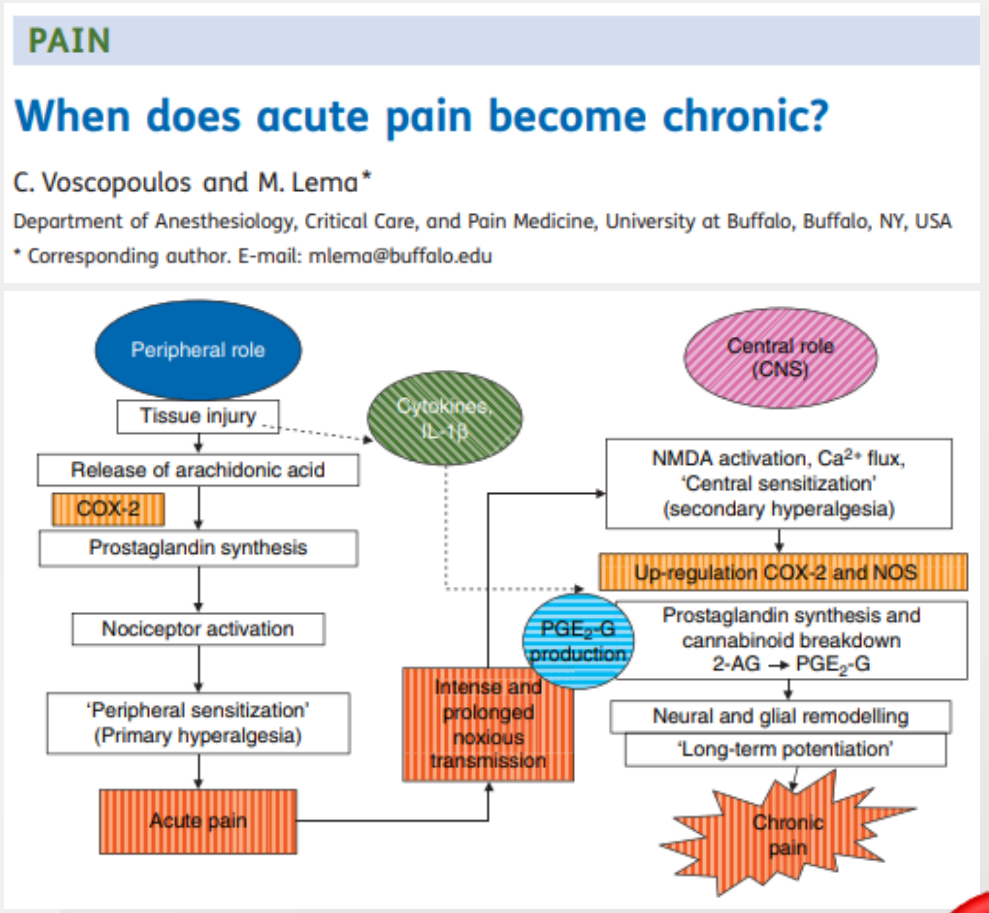
return

Menu

WRONG ANSWER! CLICK ON THE AUDIO, INTERACT AND TRY AGAIN!

According to Apkarian, in a publication in Neuroscience in 2019, the terminology used in pain research has strong implications for the conduct of science, as well as the way that scientists, clinicians and society interpret these new discoveries.

QUESTIONADOR PODCAST



return

Menu

CERTAIN ANSWER!

According to the IASP task force, in the publication: The International Association for the Study of Pain's Review, Definition of Pain: Concepts, Challenges, and Commitments, it was argued that pain is more than a symptom, that chronic pain can be a disease with its own clinical course, and hence the definition should reflect this perspective. Acute pain has a protective role of fundamental importance for our homeostasis and survival, alerting us and boosting the preservation of the integrity of our body.

Narrative Review

PAIN

ICD-11

Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the *International Classification of Diseases* (*ICD-11*)

Rolf-Detlef Treede[a,*], Winfried Rief[b], Antonia Barke[b], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h,i], Michael B. First[j], Maria Adele Giamberardino[k], Stein Kaasa[l,m,n], Beatrice Korwisi[b], Eva Kosek[o], Patricia Lavand'homme[p], Michael Nicholas[q], Serge Perrot[r], Joachim Scholz[s], Stephan Schug[t,u], Blair H. Smith[v], Peter Svensson[w,x], Johan W.S. Vlaeyen[y,z,aa], Shuu-Jiun Wang[bb,cc]

PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

PAIN

When does acute pain become chronic?

C. Voscopoulos and M. Lema*

Department of Anesthesiology, Critical Care, and Pain Medicine, University at Buffalo, Buffalo, NY, USA

* Corresponding author. E-mail: mlema@buffalo.edu

continue

Menu

4 - Pain is a complex and unique experience for each individual, so:

A

The pain may or may not occur as a result of tissue damage or potential damage to tissue innervated by nociceptors;

B

Pain occurs as a result of tissue damage innervated by nociceptors;

C

The impact of pain does not extend beyond your perception, and does not affect the emotional and social state of the individual;

D

There is no significant impact on the activities of people with acute and chronic pain, both recreational and of daily living.

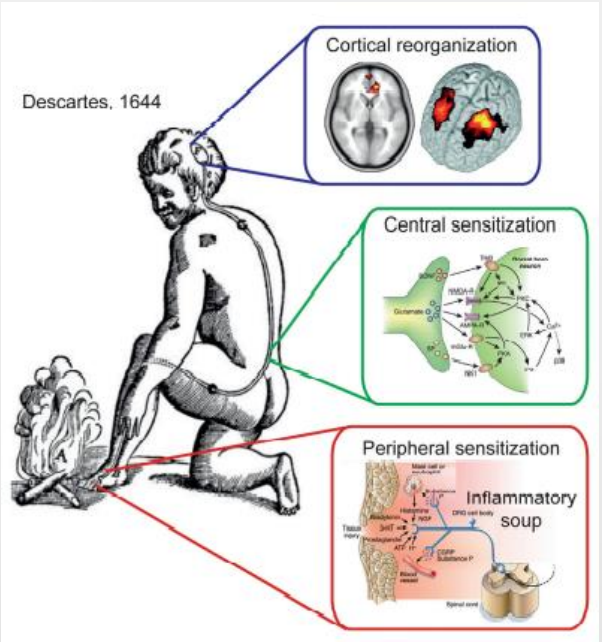
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Nociception, Pain, Negative Moods, and Behavior Selection

Marwan N. Baliki[1,*] and A. Vania Apkarian[1,2,3,*]
[1]Department of Physiology
[2]Department of Anesthesia
[3]Department of Physical Medicine and Rehabilitation

CellPress



return

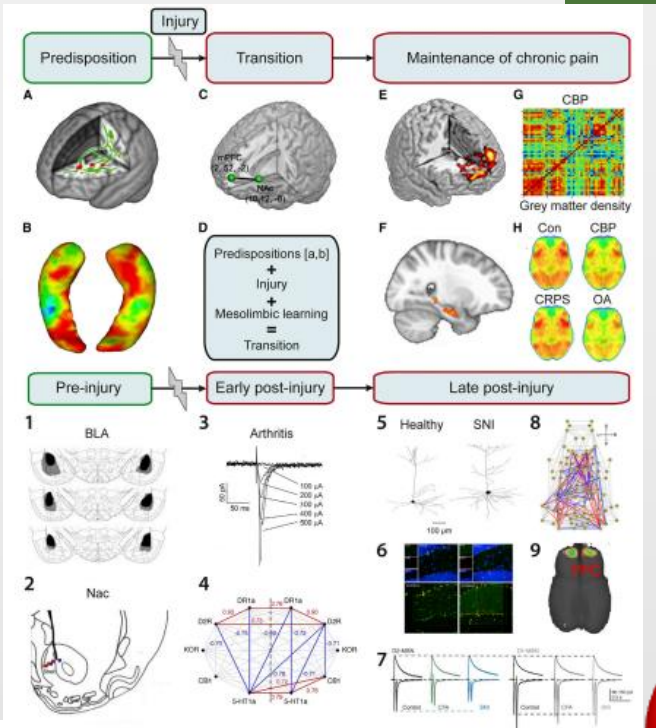
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Nociception, Pain, Negative Moods, and Behavior Selection

Marwan N. Baliki[1,*] and A. Vania Apkarian[1,2,3,*]
[1]Department of Physiology
[2]Department of Anesthesia
[3]Department of Physical Medicine and Rehabilitation

CellPress



return

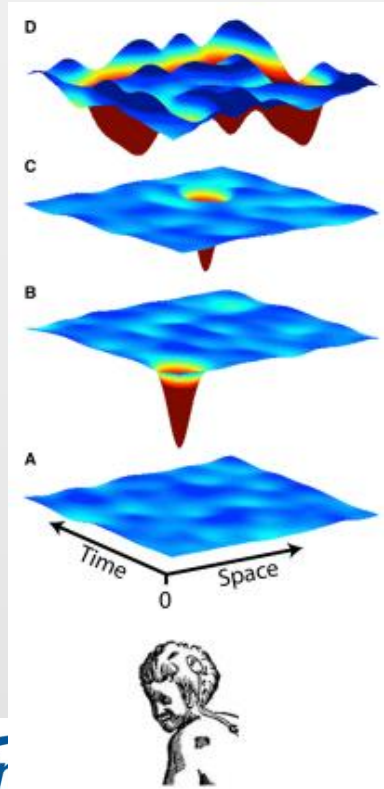
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Nociception, Pain, Negative Moods, and Behavior Selection

Marwan N. Baliki[1,*] and A. Vania Apkarian[1,2,3,*]
[1]Department of Physiology
[2]Department of Anesthesia
[3]Department of Physical Medicine and Rehabilitation

CellPress



return

Menu

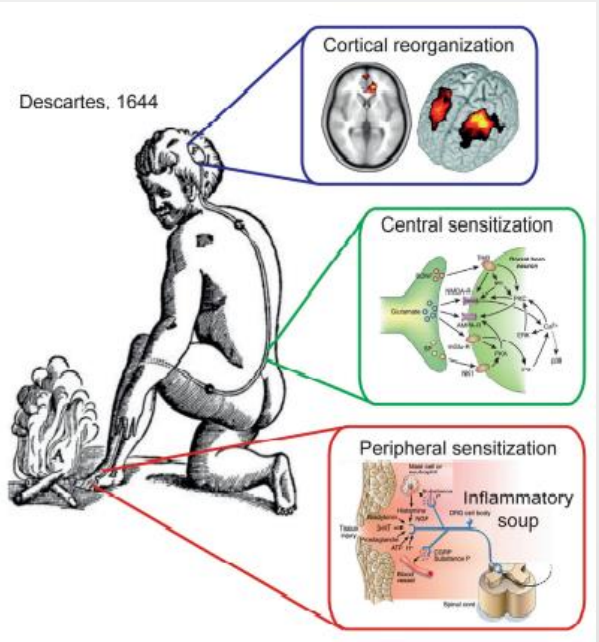
CERTAIN ANSWER!

Pain is a complex and unique experience for each individual, so it relates to teaching-learning throughout their experiences during their life. The impact of pain extends beyond your perception and can affect your emotional state and social relationships. Pain is the main reason why a person seeks medical attention. Chronic pain affects 1/3 of the world's population and 20% of these individuals report moderate to severe pain.

Nociception, Pain, Negative Moods, and Behavior Selection

Marwan N. Baliki[1,*] and A. Vania Apkarian[1,2,3,*]
[1]Department of Physiology
[2]Department of Anesthesia
[3]Department of Physical Medicine and Rehabilitation

CellPress



continue

Menu

5 - Pain Neuroscience Education (PNE) uses contemporary pain science to educate patients about the biopsychosocial nature of their pain experience. The main outcomes that are modified with PNE are:

A. Catastrophization, anxiety, disability and movement restrictions;

B. Strength, flexibility and epicritical touch;

C. Hypervigilance, balance, motor coordination and movement restrictions;

D. Stative and dynamic balance, gait and sensory-discriminative touch.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

MORTEN HOEGH, MSc, PhD, PT, EDPP, RISPT[1]

Pain Science in Practice: What Is *Pain Neuroscience*? Part 1

MORTEN HOEGH, MSc, PhD, PT, EDPP, RISPT[1]

Pain Science in Practice: What Is *Pain Neuroscience*? Part 2

SYNOPSIS: Biomechanical explanations for musculoskeletal pain are abundant and have been used for many years; however, researchers and clinicians are moving toward neuroscience-based explanations to study and explain them. This article discusses some specific mechanisms, commonly used in pain medicine, and their somewhat less specific but equally important role in nonpharmacological management of musculoskeletal pain. The article also explains the role of different receptors and how they relate to clinical conditions.
J Orthop Sports Phys Ther 2022;52(4):166-168. doi:10.2519/jospt.2022.10994

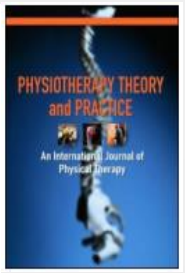
KEY WORDS: *musculoskeletal pain, neuroscience, pain, pain education, pain neurobiology education*

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Clinical biopsychosocial physiotherapy assessment of patients with chronic pain: The first step in pain neuroscience education



Amarins J. Wijma PT, PhD, C. Paul van Wilgen PT, PhD, Mira Meeus PT, PhD & Jo Nijs PT, PhD

Revisiting the Provision of Pain Neuroscience Education: An Adjunct Intervention for Patients but a Primary Focus of Clinician Education

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

American Pain Society
RESEARCH
EDUCATION
TREATMENT
ADVOCACY
PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 16, No 9 (September), 2015: pp 807-813
Available online at www.jpain.org and www.sciencedirect.com

Critical Review

Fifteen Years of Explaining Pain: The Past, Present, and Future

G. Lorimer Moseley*,† and David S. Butler*,‡

Table 1. Suggested Common Misconceptions and the Accurate Conceptions About EP

Misconception	*Accurate Conception*
EP is teaching people how to manage their pain, similar to, for example, coping skills training, relaxation training, goal setting, or problem solving skills	EP is teaching people about the biological processes underpinning pain. EP does not include instruction on strategies or skills with which to reduce the impact of pain on one's life. EP draws on instructional design and multimedia principles to present pain biology information
EP is advising people to move despite their pain	EP is teaching people that pain can be overprotective
EP is advising people that pain messages are turned up and down at the spinal cord	EP is teaching people that danger messages are turned up and down at the spinal cord
EP is describing the pain gate control theory	EP is teaching people that the brain can turn down the danger message at the spinal cord
EP is explaining that central sensitization is causing their pain, and there are no known cures for central sensitization	EP is teaching people that their danger transmission system can become very sensitive, which can lead to more danger messages, but it is always the brain that decides whether or not to produce pain
EP is reassuring people that the pain they perceive to be there is not really there at all	EP is reassuring people that their pain is completely real even although the tissue may not be in danger
EP is a discrete intervention that can be delivered effectively alongside treatments based on a structural pathology model	EP can be effectively provided only under a biopsychosocial paradigm, which integrates treatment of peripheral and central nociceptive drivers
EP relates only to chronic pain, not acute pain	EP relates to pain
EP throws out biology and biomedical models to focus only on the psychosocial	EP is a pragmatic application of the biopsychosocial model of pain, which integrates treatment of peripheral and central nociceptive drivers alongside other contributions to pain

return

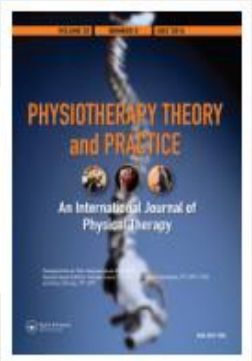
Menu

CERTAIN ANSWER!

The objectives of the PNE are to diminish beliefs, fears, biases and myths about pain. Reduce the limitations of the activities and provide adequate knowledge about pain, facilitating coping and engagement strategies. It is indicated as a tool that can modify the knowledge of patients about their painful state, change and propose new concepts about the neuroscience of pain.

Teaching patients about pain: It works, but what should we call it?

Adriaan Louw, Emilio "Louie" J. Puentedura & Kory Zimney



Therapeutic Neuroscience Education, Pain, Physiotherapy and the Pain Neuromatrix

Adriaan Louw[1] & Emilio J Puentedura[2]

IJHS
INTERAMERICAN JOURNAL OF HEALTH SCIENCES

return

Menu

6 - In the classic book "Explaining Pain," David Butler, in section 5 titled "Dealing with Life and Pain," the author considers that "active wrestlers" treat pain and many other health issues better than "passive wrestlers." Considering this proposal of the author, they are active strategies in the engagement of the treatment:

A. Learn about the problem, make plans, explore ways to move around, and explore and poke at the "edges" of pain;

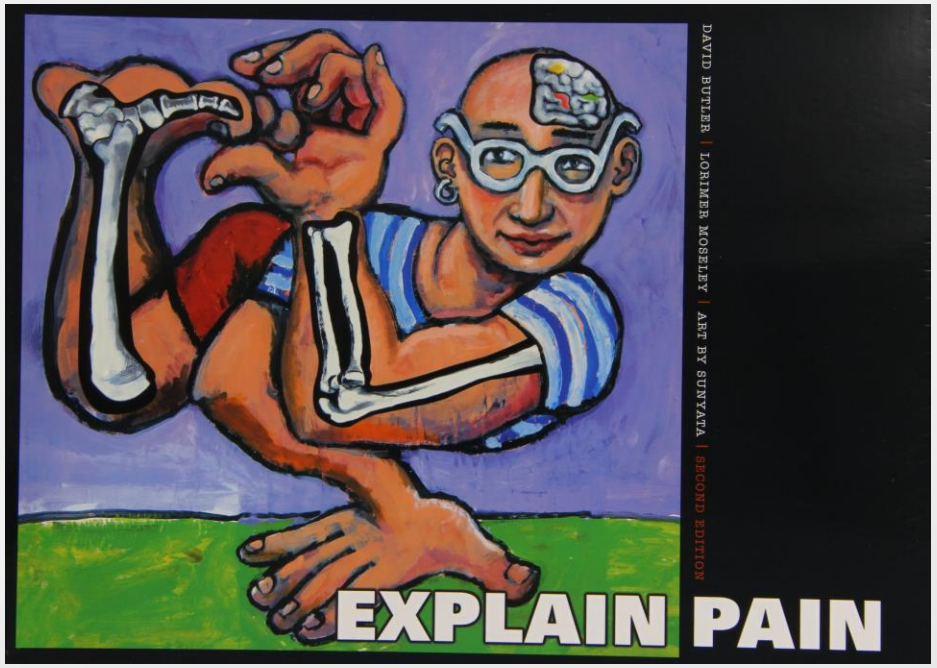
B. Exploring ways to move, believing that other people have the answers, waiting for things to happen, and doing nothing;

C. Draw plans, rest, avoid movements that poke at the "edges of pain" and seek out a movement professional to begin the exercises;

D. Learn about the problem, set goals, explore ways to move around, and avoid exploring and poking at the "edges" of pain.

Menu

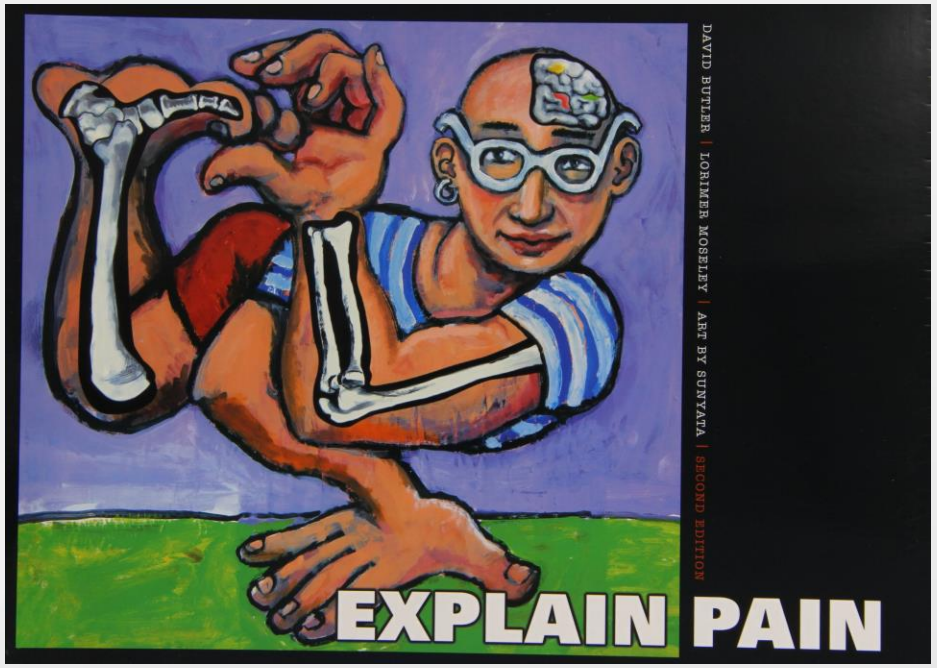
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

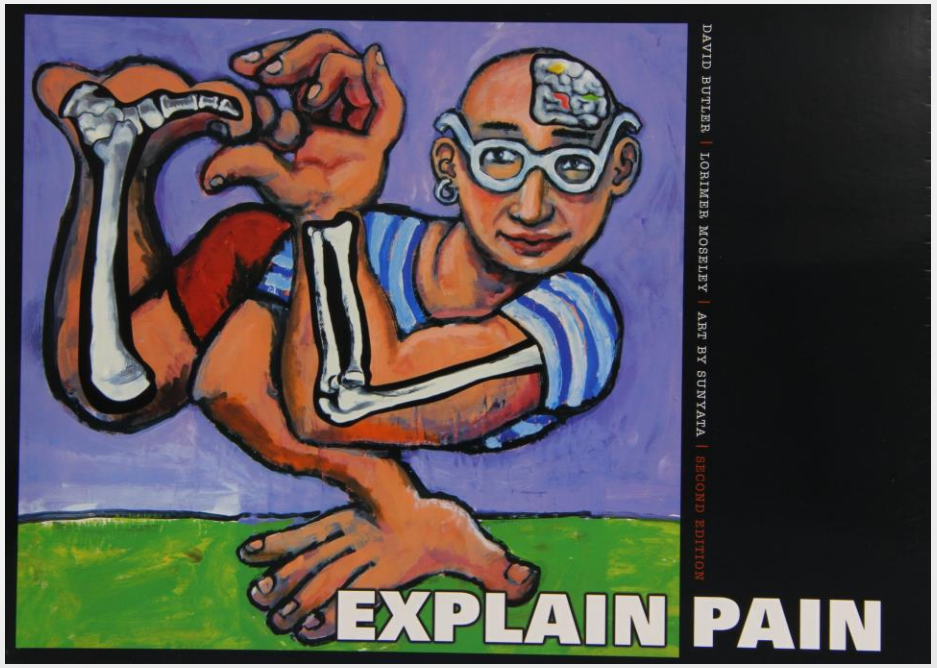
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

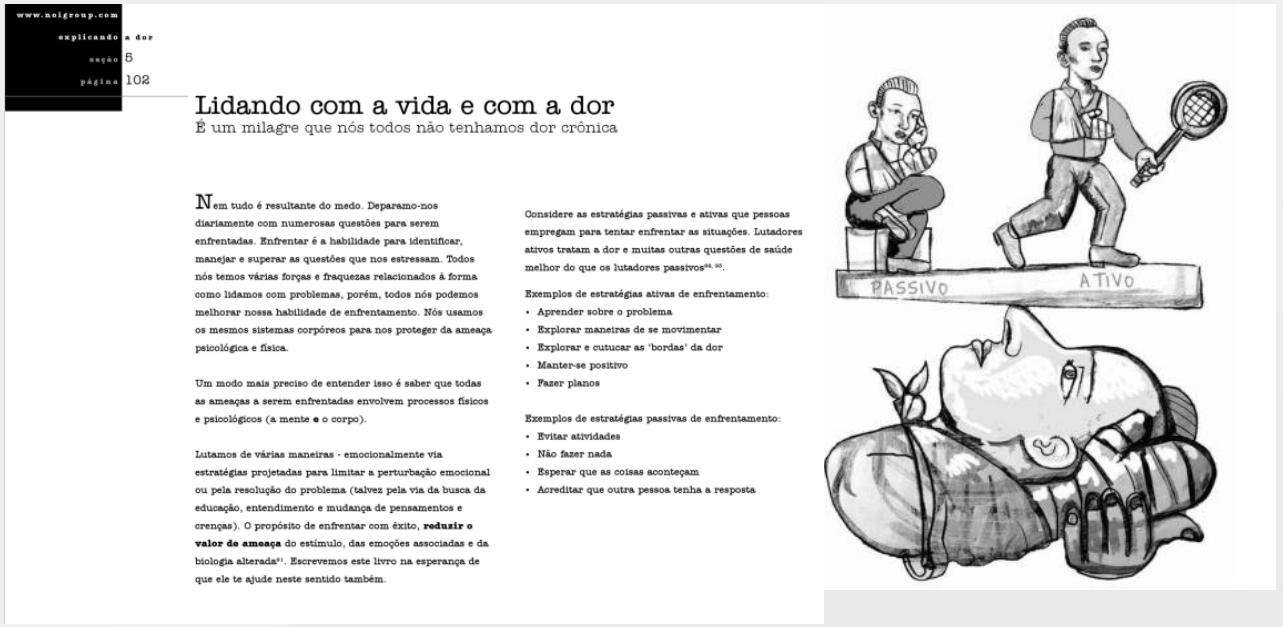


return

Menu

CERTAIN ANSWER!

"Coping is the ability to identify, manage and overcome the issues that stress us out. We all have various strengths and weaknesses related to how we deal with problems, but we can all improve our coping skills. We use the same bodily systems to protect ourselves from psychological and physical threat. A more accurate way to understand this is to know that all threats to be faced involve physical and psychological processes (the mind and body). Consider the passive and active strategies that people employ to try to cope with situations. Active wrestlers treat pain and many other health issues better than passive wrestlers."



www.noigroup.com
explicando a dor
seção 5
página 102

Lidando com a vida e com a dor

É um milagre que nós todos não tenhamos dor crônica

Nem tudo é resultante do medo. Deparamo-nos diariamente com numerosas questões para serem enfrentadas. Enfrentar é a habilidade para identificar, manejar e superar as questões que nos estressam. Todos nós temos várias forças e fraquezas relacionados à forma como lidamos com problemas, porém, todos nós podemos melhorar nossa habilidade de enfrentamento. Nós usamos os mesmos sistemas corpóreos para nos proteger da ameaça psicológica e física.

Um modo mais preciso de entender isso é saber que todas as ameaças a serem enfrentadas envolvem processos físicos e psicológicos (a mente **e** o corpo).

Lutamos de várias maneiras - emocionalmente via estratégias projetadas para limitar a perturbação emocional ou pela resolução do problema (talvez pela via da busca da educação, entendimento e mudança de pensamentos e crenças). O propósito de enfrentar com êxito, **reduzir o valor de ameaça** do estímulo, das emoções associadas e da biologia alterada[81]. Escrevemos este livro na esperança de que ele te ajude neste sentido também.

Considere as estratégias passivas e ativas que pessoas empregam para tentar enfrentar as situações. Lutadores ativos tratam a dor e muitas outras questões de saúde melhor do que os lutadores passivos[84, 85].

Exemplos de estratégias ativas de enfrentamento:
- Aprender sobre o problema
- Explorar maneiras de se movimentar
- Explorar e cutucar as 'bordas' da dor
- Manter-se positivo
- Fazer planos

Exemplos de estratégias passivas de enfrentamento:
- Evitar atividades
- Não fazer nada
- Esperar que as coisas aconteçam
- Acreditar que outra pessoa tenha a resposta

return

Menu

7 - Still based on the book "Explaining pain" and according to the PNE (Pain Neuroscience Education) understand as much as you can about what is causing your pain, and not only about what you could do about it are recommendations proposed by the authors to the IASP. So:

A

People without any training in the health professions or biology can understand the physiology of pain and understand about its physiology pain reduces the threat value of pain;

B

People with a background in health or biology should understand the physiology of pain and understanding about the physiology of pain can lead to hypervigilance increasing the value of pain softening.;

C

Physicians and physiotherapists should understand about the physiology of pain and only these professionals should explain the conditions to the population, avoiding beliefs, biases, nocebos and hypervigilance about the threat of pain;

D

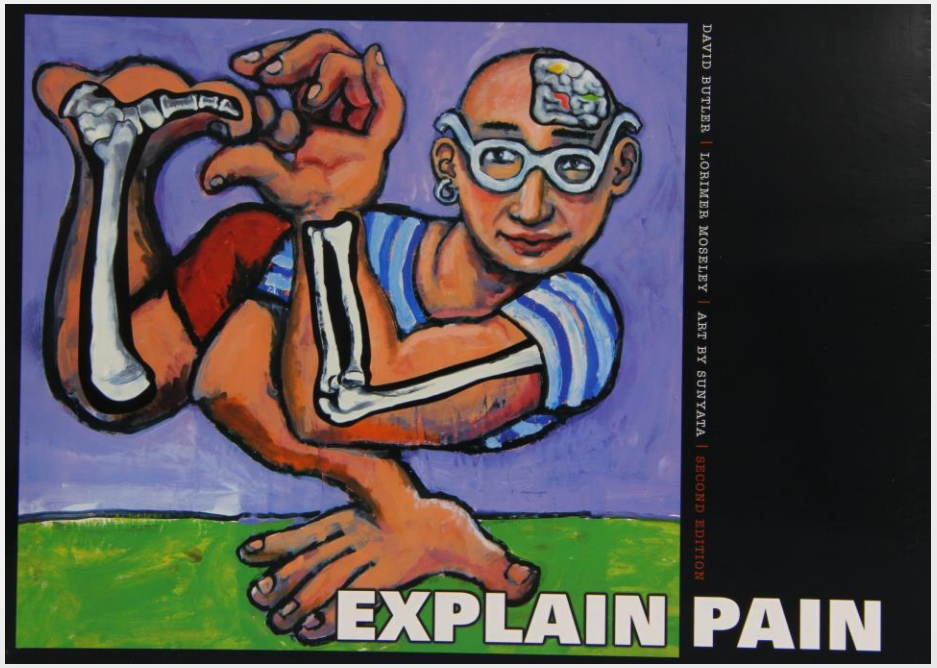
Physical therapists should understand about the physiology of pain and only these professionals should explain the conditions to the population, avoiding beliefs, biases, nocebos and hypervigilance about the threat of pain.

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

We might think that simply learning about what to do but not learning why is like superficial learning, which represents how much information is remembered but not understood by your attitudes and beliefs.

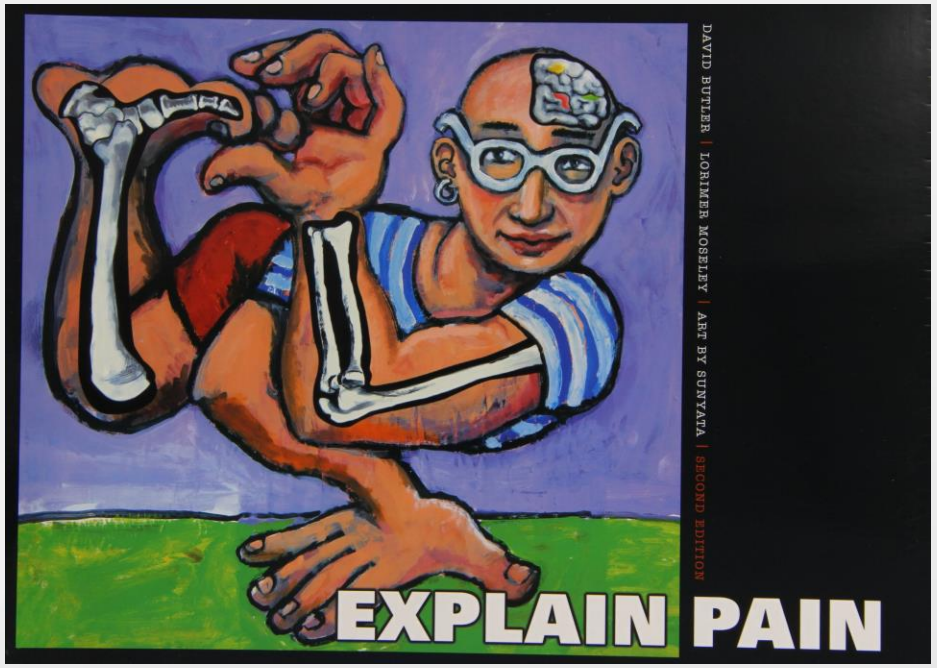
QUESTIONADOR PODCAST



return

Menu

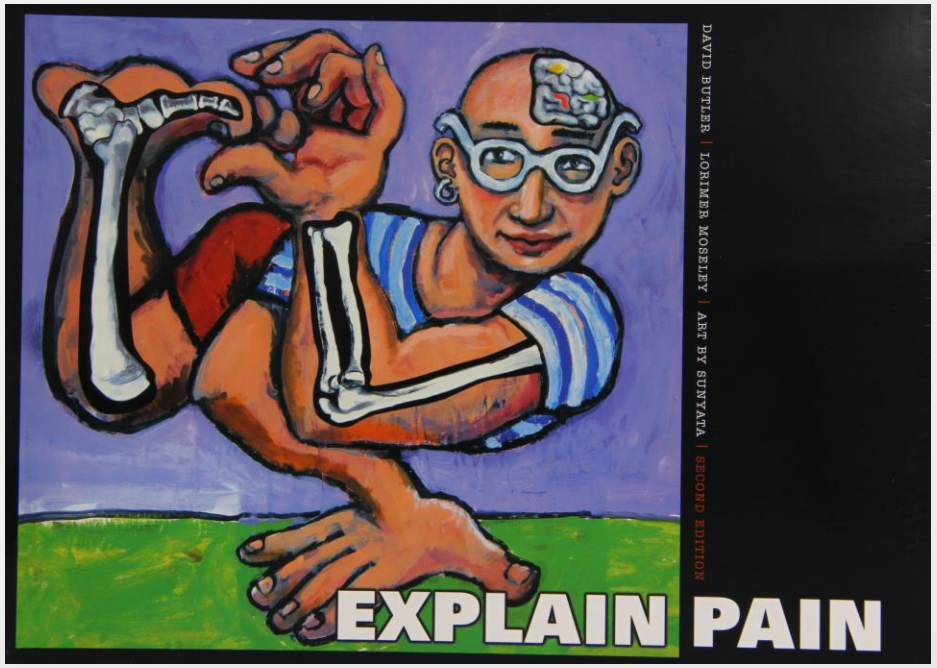
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

CERTAIN ANSWER!

People without any training in the health professions or biology can understand the physiology of pain. One of the goals of understanding the physiology of pain is to facilitate the process called "deep learning," in which information is retained, understood, and applied to available problems. One might think that simply learning about what to do, but not learning why, is like "superficial" learning, which represents when information is remembered but not understood or integrated into attitudes and beliefs.

Uso deste livro

Este livro tem quatro objetivos. Primeiro, ajudar a uma variedade de profissionais da saúde em saber como explicar a dor; queríamos fornecer uma conduta do mundo da neurociência básica a clínicos e seus pacientes. Segundo, capacitar pessoas com dor para que estas entendam mais sobre sua situação e sintam menos medo da sua dor. Sabemos que o valor de ameaça da dor contribui diretamente para a experiência de dor, e, ao informar as pessoas sobre o que realmente está acontecendo dentro delas, podemos reduzir esta ameaça. Terceiro, ajudar as pessoas com dor e aqueles que fazem parte da vida destas a fazer escolhas melhores em relação aos seus tratamentos. E por último, apresentar modelos modernos de tratamento e fornecer o tratamento essencial para superar a dor e retornar a uma vida normal.

O livro é planejado para ser usado como um manual por clínicos a fim de explicarem dor aos seus pacientes como um livro de consulta e pesquisa, para ser lido de forma conjunta pelo clínico e paciente; como parte de um programa de tratamento multidisciplinar cognitivo-comportamental da dor; ou para o paciente usar como um recurso domiciliar.

Você encontrará conforme lê, pequenos números espalhados no meio do texto. Estes estão relacionados às referências para leitura ou a fontes bibliográficas, nas quais encontramos a informação usada no texto. As referências estão listadas em ordem numérica na página 125.

Os princípios apresentados neste livro são particularmente condizentes com dores crônicas não-específicas (ex. lombalgia, dor no cotovelo). No entanto, podem ser estendidos aos estados de dor como aqueles provenientes da artrite reumatóide e serem usados em conjunto com outras estratégias de tratamento.

Achamos que uma das qualidades deste livro é que qualquer um que sofre de dor persistente, ou que tenha uma pessoa amada, um colega ou amigo que também sofra deste tipo de dor, possa beneficiar-se diretamente com o uso deste livro. O benefício será maior quando houver instrução de um clínico informado, quando necessário.

Finalmente, esperamos que os profissionais da saúde achem este livro, a visão da dor, o tratamento da mesma, e a forma como eles são apresentados, úteis, conforme tentam integrar a ciência moderna da dor na terapia. Todo esforço foi feito para utilizarmos referências científicas, relevantes e atualizadas. A literatura nesta área é vasta, por isso selecionamos a mais epresentativa. Existe também uma lista de livros relevantes 'de fácil leitura' no final do livro na página 129.

Lorimer e David

return

Menu

8 - The studies published by Barcellos et al. in 2017 and Aguiar et al. in 2021 showed that the epidemiology of chronic pain in Brazil has a predominance of gender, demographic region and neurophysiological mechanism. Being statistically presented:

A) 62.50% of adults female, southeast and neuropathic mechanism;

B) 45.59% of adult females, Midwest and nociceptive mechanism;

C) 42% adults without predominance of sex, Midwest and nociplastic mechanism;

D) 40% of adults and 12% of children and adolescents, northeast and neuropathic mechanism;

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Prevalência de dor crônica no Brasil: revisão sistemática

Prevalence of chronic pain in Brazil: systematic review

Débora Pinheiro Aguiar[1], Cleanis Pereira de Queiroz Souza[1], Wania Justina Miranda Barbosa[1], Francisco Fleury Uchoa Santos-Júnior[1,2], Anamaria Siriani de Oliveira[2]

Prevalence of Chronic Pain, Treatments, Perception, and Interference on Life Activities: Brazilian Population-Based Survey

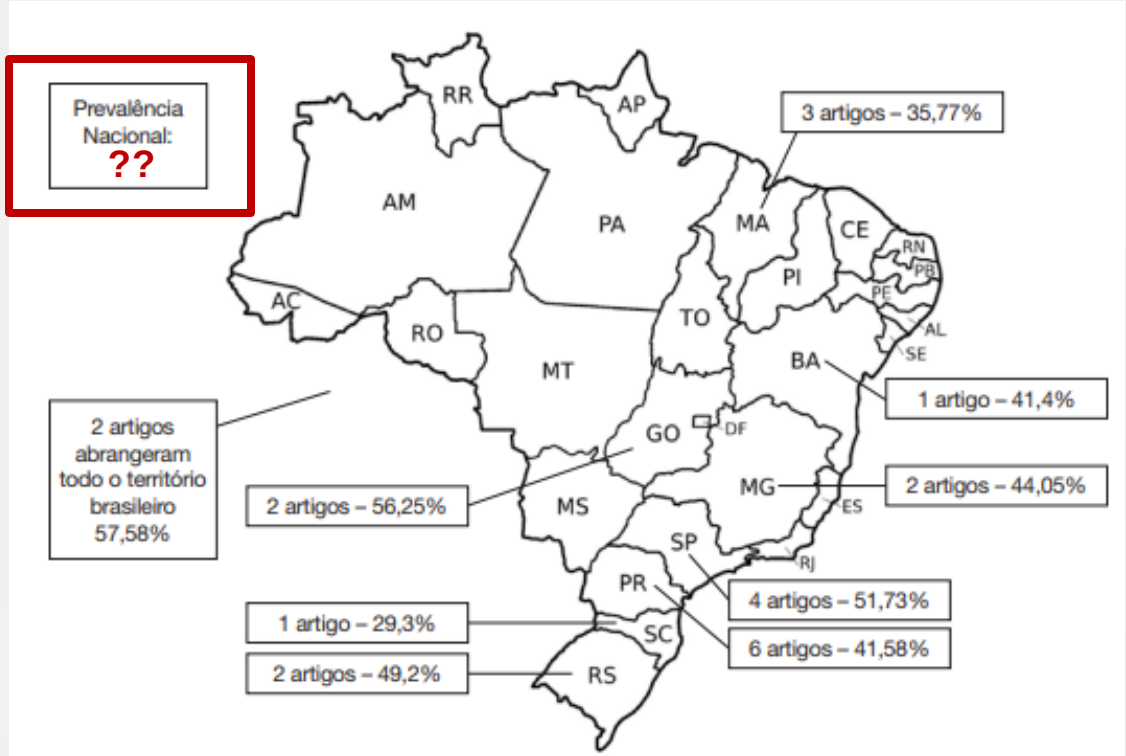
Juliana Barcellos de Souza,[1,2] Eduardo Grossmann,[2,3] Dirce Maria Navas Perissinotti,[2,4] Jose Oswaldo de Oliveira Junior,[2,5] Paulo Renato Barreiros da Fonseca,[2,6] and Irimar de Paula Posso[2,7]

Pain Research and Management
Volume 2017, Article ID 4643830, 9 pages
https://doi.org/10.1155/2017/4643830

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

BJPT

Prevalência de dor crônica no Brasil: revisão sistemática

Prevalence of chronic pain in Brazil: systematic review

Débora Pinheiro Aguiar[1], Cleanis Pereira de Queiroz Souza[1], Wania Justina Miranda Barbosa[1], Francisco Fleury Uchoa Santos-Júnior[1,2], Anamaria Siriani de Oliveira[2]

Prevalence of Chronic Pain, Treatments, Perception, and Interference on Life Activities: Brazilian Population-Based Survey

Juliana Barcellos de Souza,[1,2] Eduardo Grossmann,[2,3] Dirce Maria Navas Perissinotti,[2,4] Jose Oswaldo de Oliveira Junior,[2,5] Paulo Renato Barreiros da Fonseca,[2,6] and Irimar de Paula Posso[2,7]

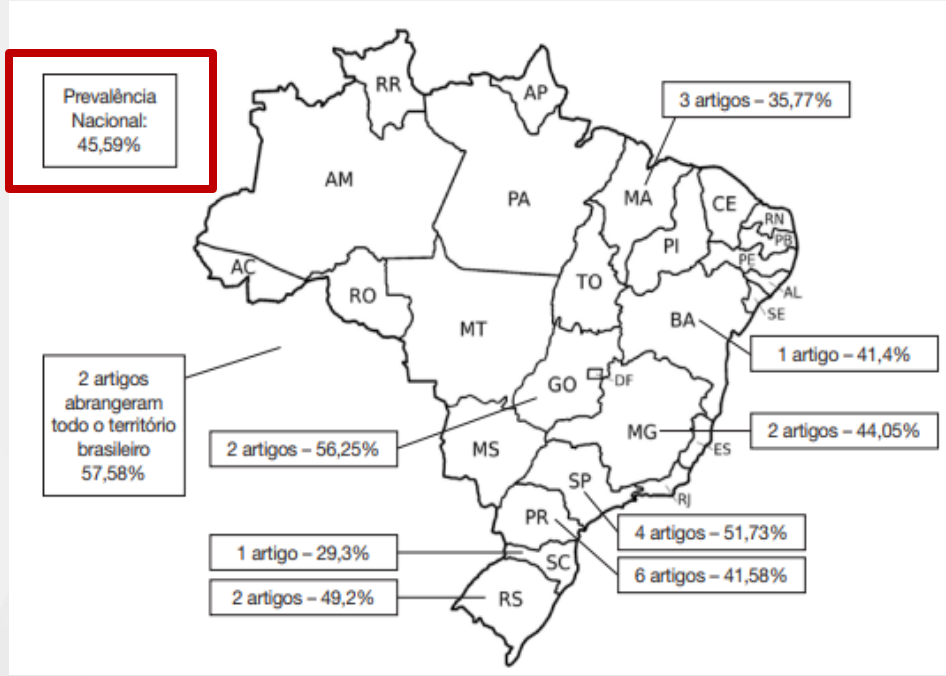
Pain Research and Management
Volume 2017, Article ID 4643830, 9 pages
https://doi.org/10.1155/2017/4643830

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

These studies aimed to estimate the prevalence of chronic pain in Brazil, describe and compare the differences between types and characteristics of pain, identifying the types of therapies adopted and the impact of pain on the daily life of Brazilians. Most people turn to specialists in the fields of orthopedics, rheumatology and neurology. They also have a broad prescription of analgesics and anti-inflammatory drugs for pain management. The study showed that chronic pain in Brazil affects 45.59% of adults, predominantly female, with a predominance of a possible nociceptive neurophysiological mechanism and a higher prevalence of involvement in the lumbar spine. The Brazilian Society for the Study of Pain (SBED) proposes a national campaign for the treatment and control of acute and chronic pain. The Brazil without pain project of SBED, aims at an educational management due to the high prevalence of disease in our territory.



return

Menu

9 – IASP defines pain as an unpleasant sensory and emotional experience associated with actual or potential harm in terms of such harm. Melzack and Casey, in 1968, proposed 3 (three) dimensions of pain, they are:

A — Sensory, discriminative and interpretive;

B — Evaluative, discriminative and perspective;

C — Affective-motivational, cognitive-evaluative and sensory-discriminative;

D — Sensory-discriminative, evaluative-interpretive and affective-interpretive.

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

If the injury or any other noxious input evokes an aversive drive, the experience cannot be labeled as pain. the authors consider that pain is a function of the interaction of the 3 dimensions and cannot be attributed to any of them alone.

QUESTIONADOR PODCAST

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Chapter 20

SENSORY, MOTIVATIONAL, AND CENTRAL CONTROL DETERMINANTS OF PAIN

A New Conceptual Model*

R. Melzack and K. L. Casey†

THE PROBLEM OF PAIN, since the beginning of the century, has been dominated by the concept that pain is a sensory experience. Yet pain has a unique, distinctly unpleasant, affective quality that differentiates it from sensory experiences such as sight, hearing, or touch. It becomes overwhelming, demands immediate attention, and disrupts ongoing behavior and thought. It motivates or drives the organism into activity aimed at stopping the pain as quickly as possible. To consider only the sensory feature pain, and ignore its motivational and affective properties, is to l part of the problem, and not even the most important part and Livingston, 1963; Chapman, Dingman, and theory that pain is a sensory modality is relati theory of pain in the 19th century (Marsh that it is an affective *quale* — the op tion, and emphasized the unpl organism into action. Th dimensions was clea rarely, probabl is, without amot

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

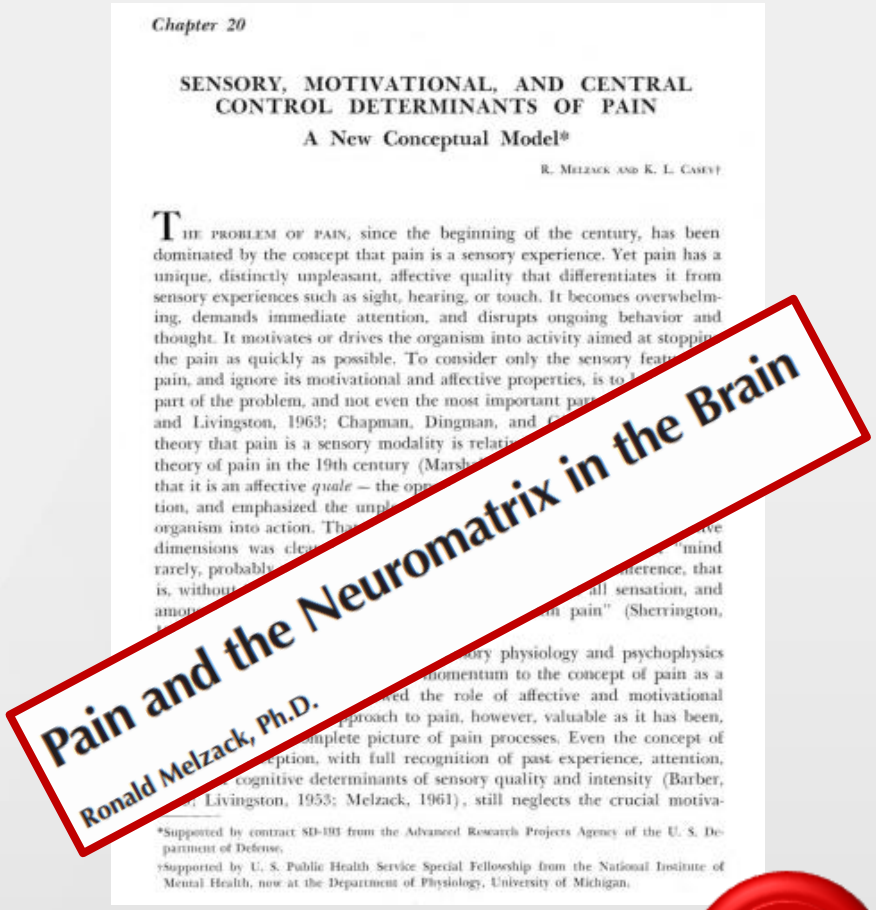
*Supported by contract SD-193 from the Advanced Research Projects Agency of the U. S. Department of Defense.

†Supported by U. S. Public Health Service Special Fellowship from the National Institute of Mental Health, now at the Department of Physiology, University of Michigan.

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

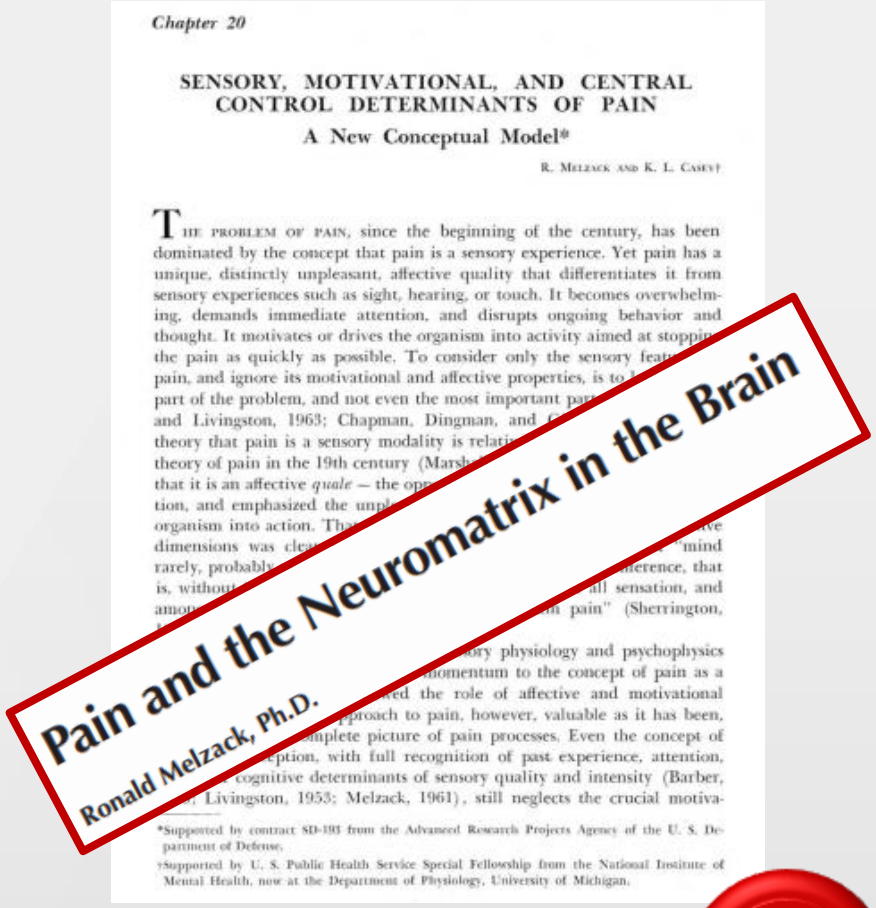


return

Menu

CERTAIN ANSWER!

Melzack and Casey in 1968 related 3 dimensions to the universe of pain. The word "pain" is a label, a category, meaning a multitude of unique and different experiences. Pain varies throughout life, both in the sensitive-discriminative dimension and in the affective-motivational dimensions. The magnitude or intensity along these dimensions is influenced by cognitive activities. If the injury or any other harmful entry fails to evoke the aversive impulse, the experience cannot be labeled as pain. The authors consider that pain is an interaction function of the 3 dimensions and cannot be attributed to any of them alone. Clearly, each of these areas of the central nervous system involved in the total experience of pain has specialized functions. Within this model, this "function" does not reside in any area. Instead, each specialized portion of the brain contributes to the experience and response as a whole.


Chapter 20

SENSORY, MOTIVATIONAL, AND CENTRAL CONTROL DETERMINANTS OF PAIN

A New Conceptual Model*

R. Melzack and K. L. Casey†

THE PROBLEM OF PAIN, since the beginning of the century, has been dominated by the concept that pain is a sensory experience. Yet pain has a unique, distinctly unpleasant, affective quality that differentiates it from sensory experiences such as sight, hearing, or touch. It becomes overwhelming, demands immediate attention, and disrupts ongoing behavior and thought. It motivates or drives the organism into activity aimed at stopping the pain as quickly as possible. To consider only the sensory feature of pain, and ignore its motivational and affective properties, is to leave out part of the problem, and not even the most important part.

and Livingston, 1963; Chapman, Dingman, and

theory that pain is a sensory modality is relati

theory of pain in the 19th century (Marsh

that it is an affective *quale* — the op

tion, and emphasized the unp

organism into action. Th

dimensions was clea

rarely, probabl

is, without

amo

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

ory physiology and psychophysics momentum to the concept of pain as a ed the role of affective and motivational pproach to pain, however, valuable as it has been, mplete picture of pain processes. Even the concept of ception, with full recognition of past experience, attention, cognitive determinants of sensory quality and intensity (Barber, Livingston, 1953; Melzack, 1961), still neglects the crucial motiva-

*Supported by contract SD-193 from the Advanced Research Projects Agency of the U. S. Department of Defense.

†Supported by U. S. Public Health Service Special Fellowship from the National Institute of Mental Health, now at the Department of Physiology, University of Michigan.

return

Menu

10 - The sensory-discriminative component covers:

A — Pain intensity and quality;

B — Intensity and duration of pain;

C — Intensity, quality, impact and duration of pain;

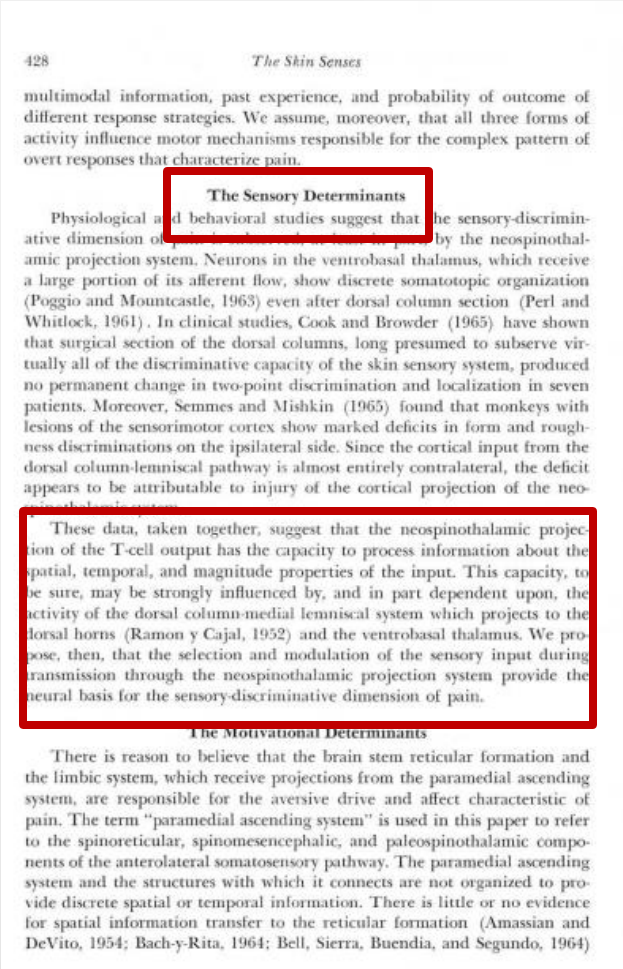
D — Intensity, location, quality and duration of pain.

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

Neurons of the thalamus, in their ventro-basal part, receive a large part of their afferent influx and present a discreet somatotopic organization. The authors then propose that the modulation of sensory input, during transmission through the neospinothalamic projection system, provides the neural basis for the sensitive-discriminative dimension of pain;

QUESTIONADOR PODCAST



428 *The Skin Senses*

multimodal information, past experience, and probability of outcome of different response strategies. We assume, moreover, that all three forms of activity influence motor mechanisms responsible for the complex pattern of overt responses that characterize pain.

The Sensory Determinants

Physiological and behavioral studies suggest that the sensory-discriminative dimension of pain is subserved in part by the neospinothalamic projection system. Neurons in the ventrobasal thalamus, which receive a large portion of its afferent flow, show discrete somatotopic organization (Poggio and Mountcastle, 1963) even after dorsal column section (Perl and Whitlock, 1961). In clinical studies, Cook and Browder (1965) have shown that surgical section of the dorsal columns, long presumed to subserve virtually all of the discriminative capacity of the skin sensory system, produced no permanent change in two-point discrimination and localization in seven patients. Moreover, Semmes and Mishkin (1965) found that monkeys with lesions of the sensorimotor cortex show marked deficits in form and roughness discriminations on the ipsilateral side. Since the cortical input from the dorsal column-lemniscal pathway is almost entirely contralateral, the deficit appears to be attributable to injury of the cortical projection of the neospinothalamic system.

These data, taken together, suggest that the neospinothalamic projection of the T-cell output has the capacity to process information about the spatial, temporal, and magnitude properties of the input. This capacity, to be sure, may be strongly influenced by, and in part dependent upon, the activity of the dorsal column-medial lemniscal system which projects to the dorsal horns (Ramon y Cajal, 1952) and the ventrobasal thalamus. We propose, then, that the selection and modulation of the sensory input during transmission through the neospinothalamic projection system provide the neural basis for the sensory-discriminative dimension of pain.

The Motivational Determinants

There is reason to believe that the brain stem reticular formation and the limbic system, which receive projections from the paramedial ascending system, are responsible for the aversive drive and affect characteristic of pain. The term "paramedial ascending system" is used in this paper to refer to the spinoreticular, spinomesencephalic, and paleospinothalamic components of the anterolateral somatosensory pathway. The paramedial ascending system and the structures with which it connects are not organized to provide discrete spatial or temporal information. There is little or no evidence for spatial information transfer to the reticular formation (Amassian and DeVito, 1954; Bach-y-Rita, 1964; Bell, Sierra, Buendia, and Segundo, 1964)

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

multimodal information, past experience, and probability of outcome of different response strategies. We assume, moreover, that all three forms of activity influence motor mechanisms responsible for the complex pattern of overt responses that characterize pain.

The Sensory Determinants

Physiological and behavioral studies suggest that the sensory-discriminative dimension of pain is subserved, at least in part, by the neospinothalamic projection system. Neurons in the ventrobasal thalamus, which receive [illegible] (Poggio and Mountcastle, 1963) even after dorsal column section (Perl and Whitlock, 1961). In clinical studies, Cook and Browder (1965) have shown that surgical section of the dorsal columns, long presumed to subserve virtually all of the discriminative capacity of the skin sensory system, produced no permanent change in two-point discrimination and localization in seven patients. Moreover, Semmes and Mishkin (1965) found that monkeys with lesions of the sensorimotor cortex show marked deficits in form and roughness discriminations on the ipsilateral side. Since the cortical input from the dorsal column-lemniscal pathway is almost entirely contralateral, the deficit appears to be attributable to injury of the cortical projection of the neospinothalamic system.

These data, taken together, suggest that the neospinothalamic projection of the T-cell output has the capacity to process information about the spatial, temporal, and magnitude properties of the input. This capacity, to be sure, may be strongly influenced by, and in part dependent upon, the activity of the dorsal column-medial lemniscal system which projects to the dorsal horns (Ramon y Cajal, 1952) and the ventrobasal thalamus. We propose, then, that the selection and modulation of the sensory input during transmission through the neospinothalamic projection system provide the neural basis for the sensory-discriminative dimension of pain.

The Motivational Determinants

There is reason to believe that the brain stem reticular formation and the limbic system, which receive projections from the paramedial ascending system, are responsible for the aversive drive and affect characteristic of pain. The term "paramedial ascending system" is used in this paper to refer to the spinoreticular, spinomesencephalic, and paleospinothalamic components of the anterolateral somatosensory pathway. The paramedial ascending system and the structures with which it connects are not organized to provide discrete spatial or temporal information. There is little or no evidence for spatial information transfer to the reticular formation (Amassian and DeVito, 1954; Bach-y-Rita, 1964; Bell, Sierra, Buendia, and Segundo, 196[illegible]

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

multimodal information, past experience, and probability of outcome of different response strategies. We assume, moreover, that all three forms of activity influence motor mechanisms responsible for the complex pattern of overt responses that characterize pain.

The Sensory Determinants

Physiological and behavioral studies suggest that the sensory-discriminative dimension of pain is subserved, at least in part, by the neospinothalamic projection system. Neurons in the ventrobasal thalamus, which receive a large portion of its afferent flow, show discrete somatotopic organization (Poggio and Mountcastle, 1963) even after dorsal column section (Perl and Whitlock, 1961). In clinical studies, Cook and Browder (1965) have shown that surgical section of the dorsal columns, long presumed to subserve virtually all of the discriminative capacity of the skin sensory system, produced no permanent change in two-point discrimination and localization in seven patients. Moreover, Semmes and Mishkin (1965) found that monkeys with lesions of the sensorimotor cortex show marked deficits in form and roughness discriminations on the ipsilateral side. Since the cortical input from the dorsal column-lemniscal pathway is almost entirely contralateral, the deficit appears to be attributable to injury of the cortical projection of the neospinothalamic system.

These data, taken together, suggest that the neospinothalamic projection of the T-cell output has the capacity to process information about the spatial, temporal, and magnitude properties of the input. This capacity, to be sure, may be strongly influenced by, and in part dependent upon, the activity of the dorsal column-medial lemniscal system which projects to the dorsal horns (Ramon y Cajal, 1952) and the ventrobasal thalamus. We propose, then, that the selection and modulation of the sensory input during transmission through the neospinothalamic projection system provide the neural basis for the sensory-discriminative dimension of pain.

The Motivational Determinants

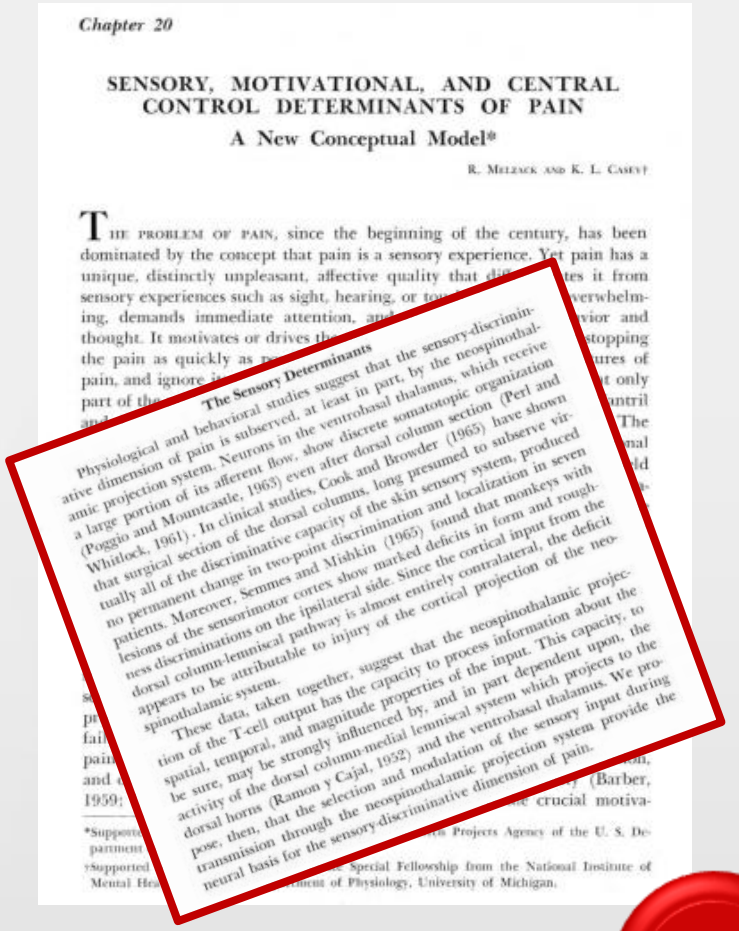
There is reason to believe that the brain stem reticular formation and the limbic system, which receive projections from the paramedial ascending system, are responsible for the aversive drive and affect characteristic of pain. The term "paramedial ascending system" is used in this paper to refer to the spinoreticular, spinomesencephalic, and paleospinothalamic components of the anterolateral somatosensory pathway. The paramedial ascending system and the structures with which it connects are not organized to provide discrete spatial or temporal information. There is little or no evidence for spatial information transfer to the reticular formation (Amassian and DeVito, 1954; Bach-y-Rita, 1964; Bell, Sierra, Buendia, and Segundo, 1964)

return

Menu

CERTAIN ANSWER!

Physiological and behavioral studies suggest that the sensory-discriminative dimension of pain is met, at least in part by the projection of the neospinothalamic pathway. Neurons in the thalamus, in its ventrobasal part, largely receive their afferent flow and present discrete somatotopic organization. The authors propose, then, that the selection and modulation of sensory input during transmission through the neospinothalamic projection system provide the neural basis for the sensory-discriminative dimension of pain.



Chapter 20

SENSORY, MOTIVATIONAL, AND CENTRAL CONTROL DETERMINANTS OF PAIN

A New Conceptual Model*

R. Melzack and K. L. Casey†

The Sensory Determinants

Physiological and behavioral studies suggest that the sensory-discriminative dimension of pain is subserved, at least in part, by the neospinothalamic projection system. Neurons in the ventrobasal thalamus, which receive a large portion of its afferent flow, show discrete somatotopic organization (Poggio and Mountcastle, 1963) even after dorsal column section (Perl and Whitlock, 1961). In clinical studies, Cook and Browder (1965) have shown that surgical section of the dorsal columns, long presumed to subserve virtually all of the discriminative capacity of the skin sensory system, produced no permanent change in two-point discrimination and localization in seven patients. Moreover, Semmes and Mishkin (1965) found that monkeys with lesions of the sensorimotor cortex show marked deficits in form and roughness discriminations on the ipsilateral side. Since the cortical input from the dorsal column-lemniscal pathway is almost entirely contralateral, the deficit appears to be attributable to injury of the cortical projection of the neospinothalamic system.

These data, taken together, suggest that the neospinothalamic projection of the T-cell output has the capacity to process information about the spatial, temporal, and magnitude properties of the input. This capacity, to be sure, may be strongly influenced by, and in part dependent upon, the activity of the dorsal column-medial lemniscal system which projects to the dorsal horns (Ramon y Cajal, 1952) and the ventrobasal thalamus. We propose, then, that the selection and modulation of the sensory input during transmission through the neospinothalamic projection system provide the neural basis for the sensory-discriminative dimension of pain.

return

Menu

11 - The affective-motivational component covers:

A. The suffering of the individual related to pain;

B. The impact of pleasant, unpleasant and creative experience on the individual;

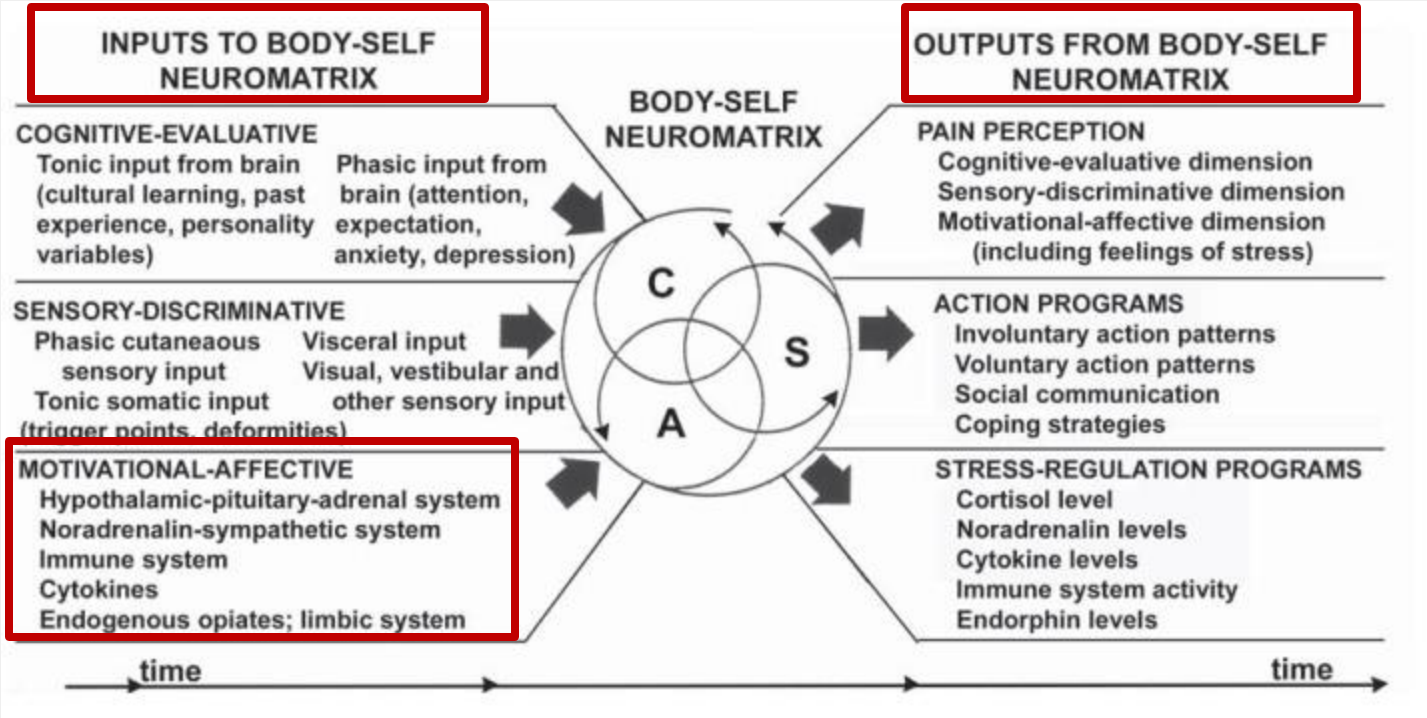
C. The impact of the emotional and psychological relationship of pain on the individual;

D. The impact of the complex psychophysical relationship on willpower in the individual.

Menu

WRONG ANSWER, INTERACT WITH THE SCHEME AND TRY AGAIN!

Factors that contribute to patterns of activities generated by the neuromatrix, which comprises the sensory, affective and cognitive areas. Neuromatrix output patterns produce multiple dimensions of pain experience, as well as concomitant homeostatic and behavioral responses.



return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

Melzack, also proposes in the study “Pain and the NeuroMatrix in the Brain” in 2001, that the expansion of the field of pain, to include the endocrinological, immunological and neurohormonal part, can lead to new paths in the universe of evaluation and treatment from the pain.

QUESTIONADOR PODCAST

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

Abstract: The neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic “neurosignature” patterns of nerve impulses generated by a widely distributed neural network—the “body-self neuromatrix”—in the brain. These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Acute pains evoked by brief noxious inputs have been meticulously investigated by neuroscientists, and their sensory transmission mechanisms are generally well understood. In contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, remain a mystery. Furthermore, chronic psychological or physical stress is often associated with chronic pain, but the relationship is poorly understood. The neuromatrix theory of pain provides a new conceptual framework to examine these problems. It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioral programs after injury, pathology, or chronic stress. Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology. The neuromatrix, which is genetically determined and modified by sensory experience, is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.

Dr. Melzack is Professor Emeritus, Department of Psychology, McGill University. Direct correspondence and requests for reprints to him at Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada H3A 1B1; 514-398-6084 phone; 514-398-4896 fax; rmelzack@ego.psych.mcgill.ca.

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

Abstract: The neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses generated by a widely distributed neural network—the "body-self neuromatrix"—in the brain. These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Acute pains evoked by brief noxious inputs have been meticulously investigated by neuroscientists, and their sensory transmission mechanisms are generally well understood. In contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, remain a mystery. Furthermore, chronic psychological or physical stress is often associated with chronic pain, but the relationship is poorly understood. The neuromatrix theory of pain provides a new conceptual framework to examine these problems. It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioral programs after injury, pathology, or chronic stress. Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology. The neuromatrix, which is genetically determined and modified by sensory experience, is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.

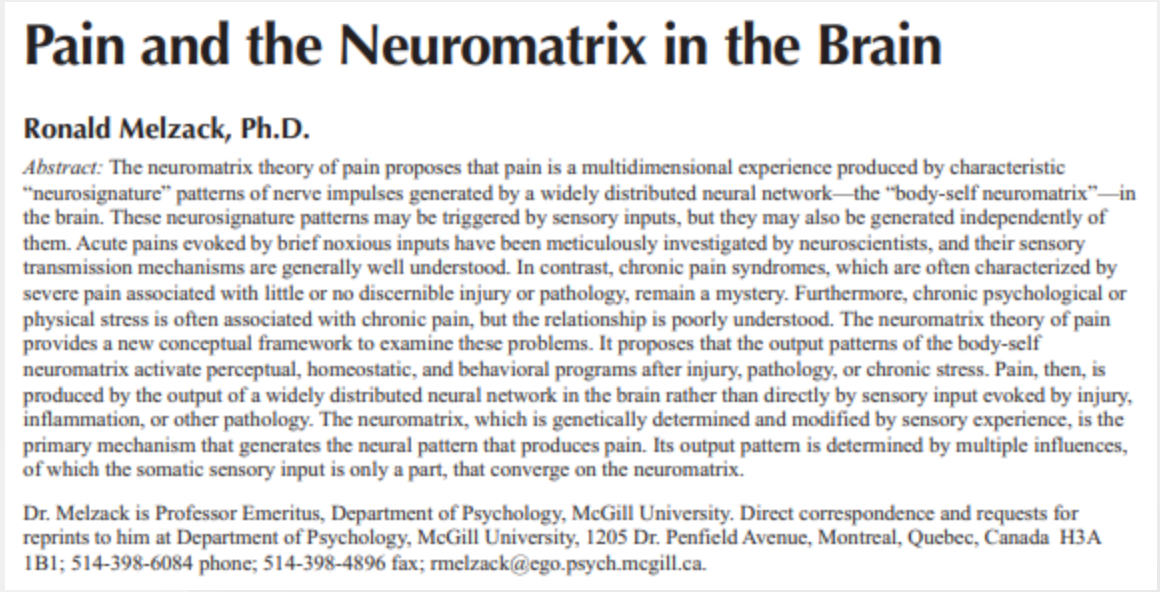
Dr. Melzack is Professor Emeritus, Department of Psychology, McGill University. Direct correspondence and requests for reprints to him at Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada H3A 1B1; 514-398-6084 phone; 514-398-4896 fax; rmelzack@ego.psych.mcgill.ca.

return

Menu

CERTAIN ANSWER!

The affective-motivational component is related to suffering related to pain. They interact with the limbic system areas of the nervous system. The multiple determinants of pain include the stress regulation system, with its complex and delicate balance, is an integral part of the multiple conditions that give rise to chronic pain. Melzack also proposes in the study "Pain and the Neuromatrix in the Brain" that the expansion of the pain field to include the neurohormonal, endocrinological and immunological part, may lead to new paths in the universe of pain assessment and treatment.



Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

Abstract: The neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses generated by a widely distributed neural network—the "body-self neuromatrix"—in the brain. These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Acute pains evoked by brief noxious inputs have been meticulously investigated by neuroscientists, and their sensory transmission mechanisms are generally well understood. In contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, remain a mystery. Furthermore, chronic psychological or physical stress is often associated with chronic pain, but the relationship is poorly understood. The neuromatrix theory of pain provides a new conceptual framework to examine these problems. It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioral programs after injury, pathology, or chronic stress. Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology. The neuromatrix, which is genetically determined and modified by sensory experience, is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.

Dr. Melzack is Professor Emeritus, Department of Psychology, McGill University. Direct correspondence and requests for reprints to him at Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada H3A 1B1; 514-398-6084 phone; 514-398-4896 fax; rmelzack@ego.psych.mcgill.ca.

return

Menu

12 - The cognitive-evaluative component covers:

A — Contextualization of pain related to the current moment of teaching-learning of sensitivity;

B — Contextualization of pain from current and past experiences;

C — Evaluation of pain from a coherent judgment of the process;

D — Pain assessment from a disproportionate interpretation of the process.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

Abstract: The neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses generated by a widely distributed neural network—the "body-self neuromatrix"—in the brain. These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Acute pains evoked by brief noxious inputs have been meticulously investigated by neuroscientists, and their sensory transmission mechanisms are generally well understood. In contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, remain a mystery. Furthermore, chronic psychological or physical stress is often associated with chronic pain, but the relationship is poorly understood. The neuromatrix theory of pain provides a new conceptual framework to examine these problems. It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioral programs after injury, pathology, or chronic stress. Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology. The neuromatrix, which is genetically determined and modified by sensory experience, is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.

Dr. Melzack is Professor Emeritus, Department of Psychology, McGill University. Direct correspondence and requests for reprints to him at Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada H3A 1B1; 514-398-6084 phone; 514-398-4896 fax; rmelzack@ego.psych.mcgill.ca.

return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

The cognitive-evaluative dimension is related to areas of the frontal cortex that play a very important role in mediating between the cognitive activities of pain. Since it receives information from intracortical fibers of virtually all sensory and associative systems. And they project to the reticular formation and limbic system.

QUESTIONADOR PODCAST

Pain and the Neuromatrix in the Brain

Ronald Melzack, Ph.D.

Abstract: The neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses generated by a widely distributed neural network—the "body-self neuromatrix"—in the brain. These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them. Acute pains evoked by brief noxious inputs have been meticulously investigated by neuroscientists, and their sensory transmission mechanisms are generally well understood. In contrast, chronic pain syndromes, which are often characterized by severe pain associated with little or no discernible injury or pathology, remain a mystery. Furthermore, chronic psychological or physical stress is often associated with chronic pain, but the relationship is poorly understood. The neuromatrix theory of pain provides a new conceptual framework to examine these problems. It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioral programs after injury, pathology, or chronic stress. Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology. The neuromatrix, which is genetically determined and modified by sensory experience, is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.

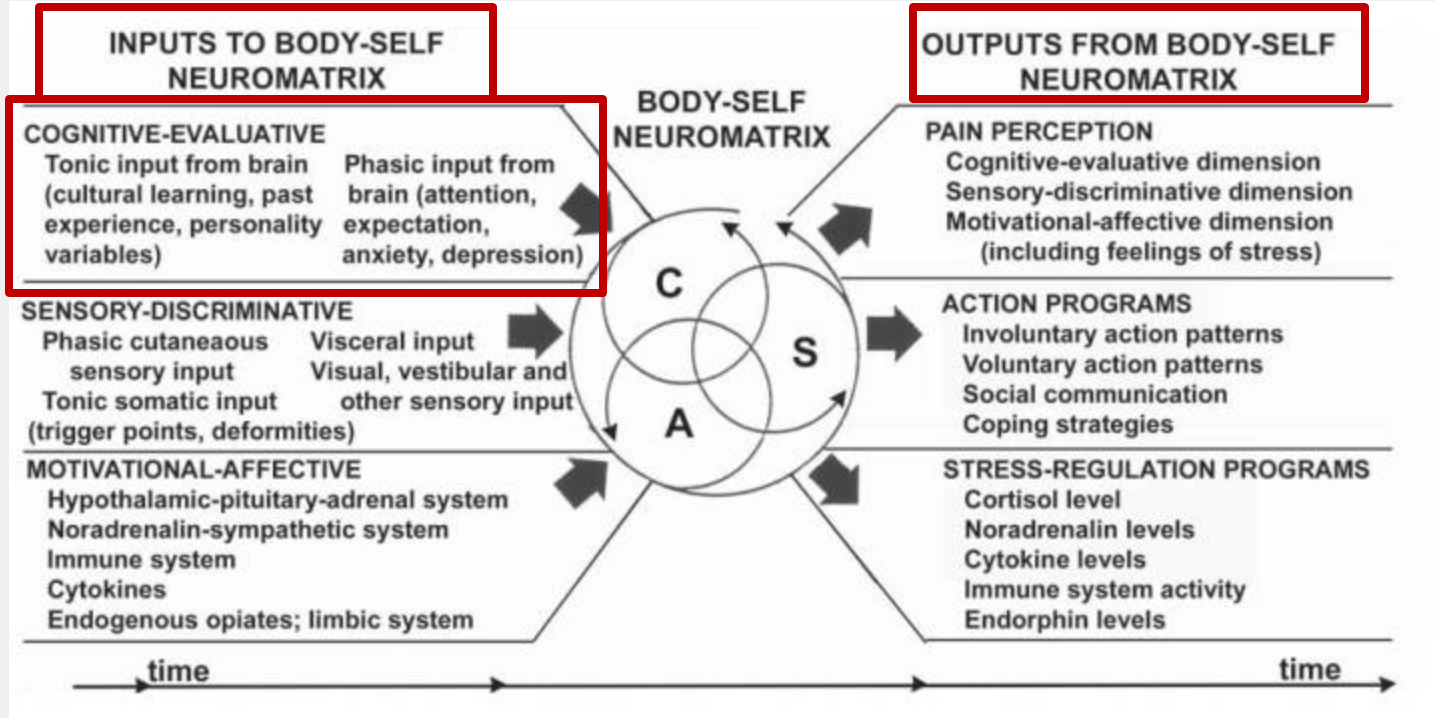
Dr. Melzack is Professor Emeritus, Department of Psychology, McGill University. Direct correspondence and requests for reprints to him at Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec, Canada H3A 1B1; 514-398-6084 phone; 514-398-4896 fax; rmelzack@ego.psych.mcgill.ca.

retornar

Menu

WRONG ANSWER, INTERACT WITH THE SCHEME AND TRY AGAIN!

Factors that contribute to patterns of activities generated by the neuromatrix, which comprises the sensory, affective and cognitive areas. Neuromatrix output patterns produce multiple dimensions of pain experience, as well as concomitant homeostatic and behavioral responses.

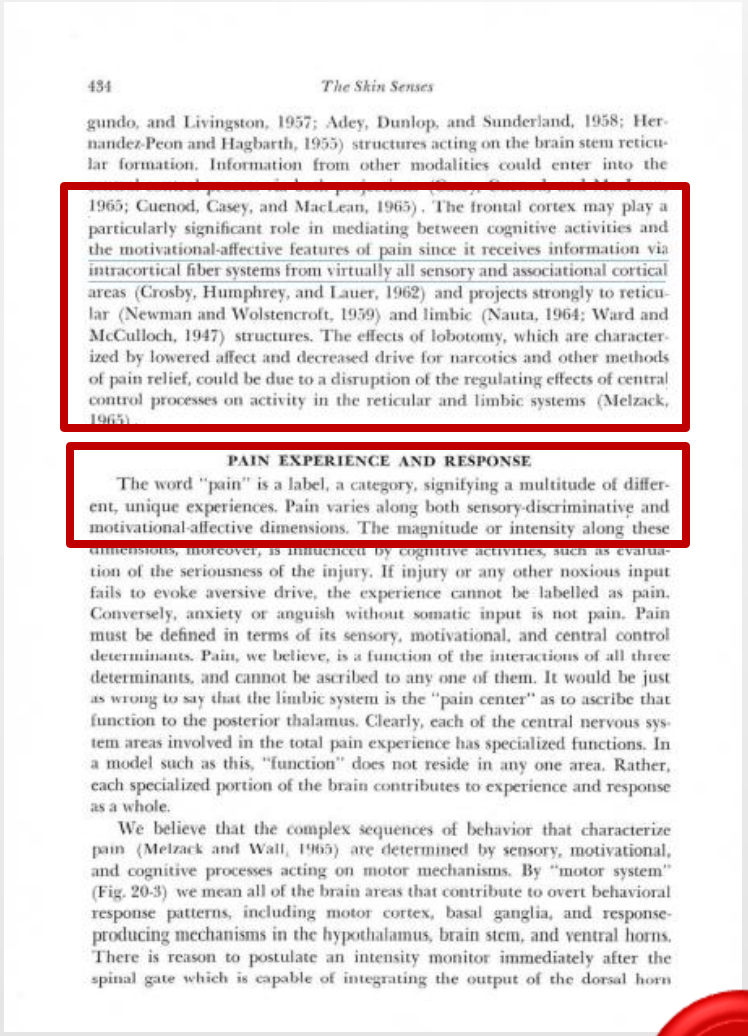


return

Menu

CERTAIN ANSWER!

The cognitive-evaluative component contextualizes pain from current and past experiences. Therefore, it is related to areas of the frontal cortex that plays a very important role in mediating between cognitive activities of pain, since it receives information from intracortical fibers of practically all sensory and associative systems and projects to reticular formation and limbic system. These activities that involve the neocortex and that also act in the reticular formation, can affect both the sensory experience in its physical properties, evaluated in terms of past and present experience and modified before influencing the sensory or motivational systems.



434 *The Skin Senses*

gundo, and Livingston, 1957; Adey, Dunlop, and Sunderland, 1958; Hernandez-Peon and Hagbarth, 1955) structures acting on the brain stem reticular formation. Information from other modalities could enter into the [illegible] 1965; Guenod, Casey, and MacLean, 1965). The frontal cortex may play a particularly significant role in mediating between cognitive activities and the motivational-affective features of pain since it receives information via intracortical fiber systems from virtually all sensory and associational cortical areas (Crosby, Humphrey, and Lauer, 1962) and projects strongly to reticular (Newman and Wolstencroft, 1959) and limbic (Nauta, 1964; Ward and McCulloch, 1947) structures. The effects of lobotomy, which are characterized by lowered affect and decreased drive for narcotics and other methods of pain relief, could be due to a disruption of the regulating effects of central control processes on activity in the reticular and limbic systems (Melzack, 1965).

PAIN EXPERIENCE AND RESPONSE

The word "pain" is a label, a category, signifying a multitude of different, unique experiences. Pain varies along both sensory-discriminative and motivational-affective dimensions. The magnitude or intensity along these dimensions, moreover, is influenced by cognitive activities, such as evaluation of the seriousness of the injury. If injury or any other noxious input fails to evoke aversive drive, the experience cannot be labelled as pain. Conversely, anxiety or anguish without somatic input is not pain. Pain must be defined in terms of its sensory, motivational, and central control determinants. Pain, we believe, is a function of the interactions of all three determinants, and cannot be ascribed to any one of them. It would be just as wrong to say that the limbic system is the "pain center" as to ascribe that function to the posterior thalamus. Clearly, each of the central nervous system areas involved in the total pain experience has specialized functions. In a model such as this, "function" does not reside in any one area. Rather, each specialized portion of the brain contributes to experience and response as a whole.

We believe that the complex sequences of behavior that characterize pain (Melzack and Wall, 1965) are determined by sensory, motivational, and cognitive processes acting on motor mechanisms. By "motor system" (Fig. 20-3) we mean all of the brain areas that contribute to overt behavioral response patterns, including motor cortex, basal ganglia, and response-producing mechanisms in the hypothalamus, brain stem, and ventral horns. There is reason to postulate an intensity monitor immediately after the spinal gate which is capable of integrating the output of the dorsal horn

return

Menu

13 - From the experience of acute pain, the individual begins to learn about pain. This experience is learned by 3 processes:

A — Learning, cognition-emotions and behavior;

B — Learning, pain intensity, and pain localization;

C — Learning, emotional state and focused attention;

D — Learning, individual intellectual level and pain intensity.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Cognitive and emotional control of pain and its disruption in chronic pain

M. Catherine Bushnell, Marta Čeko and Lucie A. Low NATURE REVIEWS | NEUROSCIENCE

REVIEW

Deconstructing the sensation of pain: The influence of cognitive processes on pain perception

Katja Wiech[1,2]* SCIENCE sciencemag.org

SPECIAL SECTION PAIN RESEARCH

Topical Review

PAIN

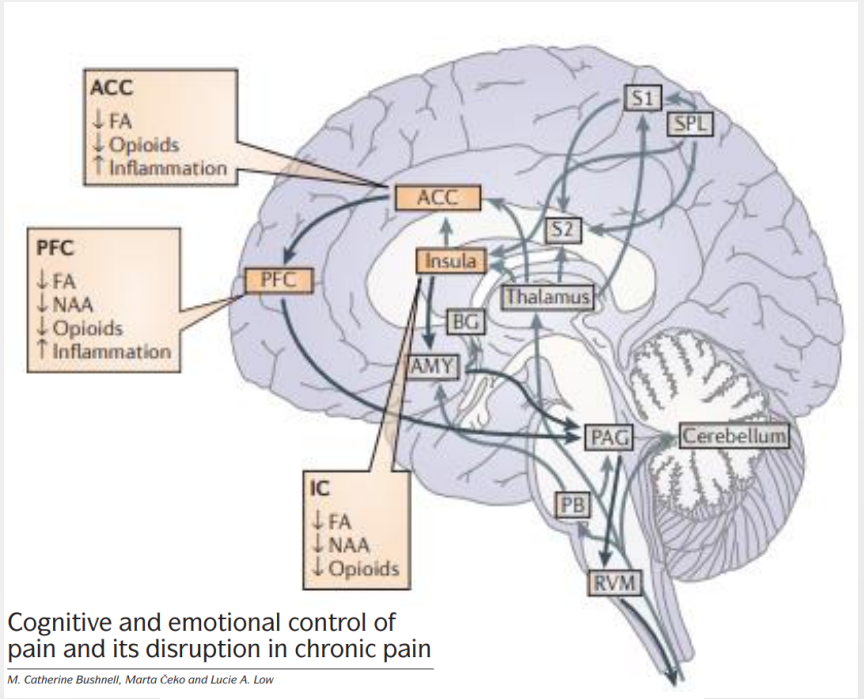
Habituation to pain: a motivational-ethological perspective

Annick L. De Paepe[a,*], Amanda C. de C. Williams[b], Geert Crombez[a]

return

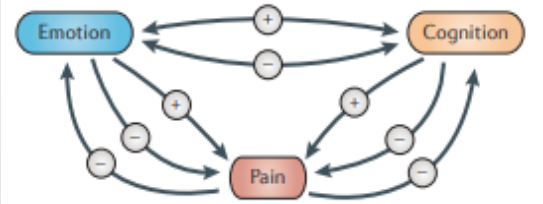
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Cognitive and emotional control of pain and its disruption in chronic pain

M. Catherine Bushnell, Marta Čeko and Lucie A. Low



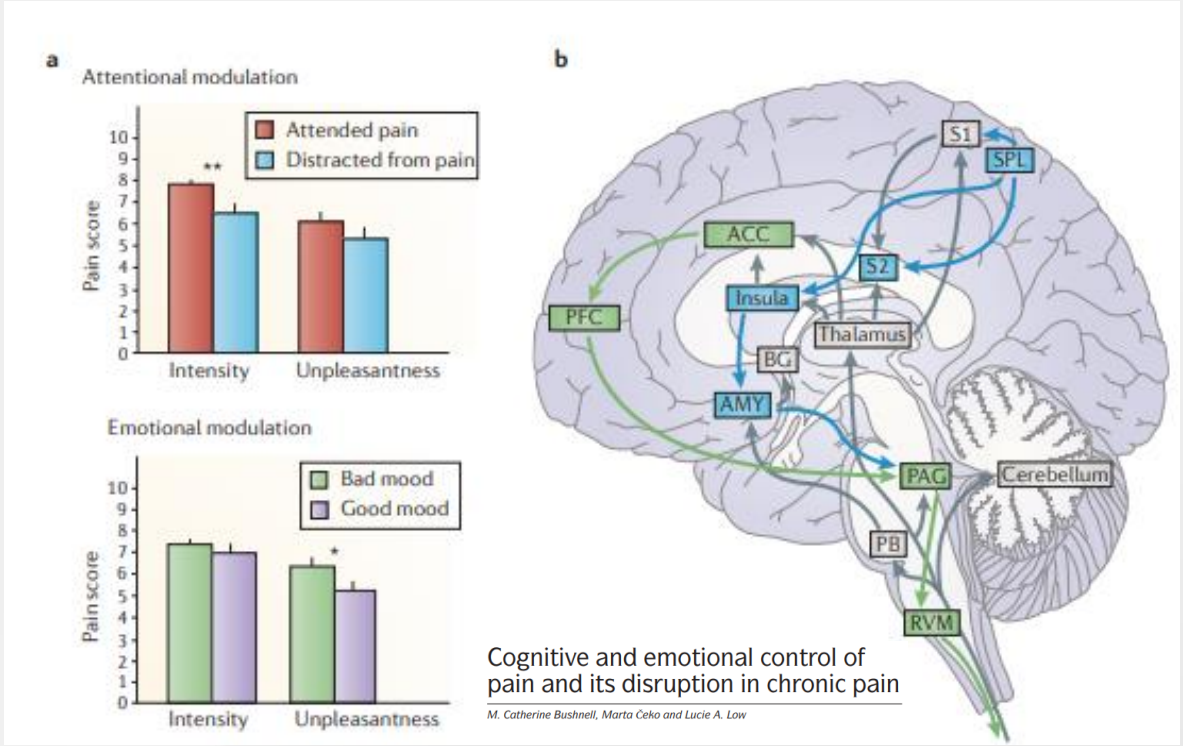
return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

These studies show decreased binding of endogenous opioids and their receptors in patients with chronic pain, in 3 cortical regions. They are: the anterior cingulate cortex, the prefrontal cortex and the insula.

QUESTIONADOR PODCAST



return

Menu

CERTAIN ANSWER!

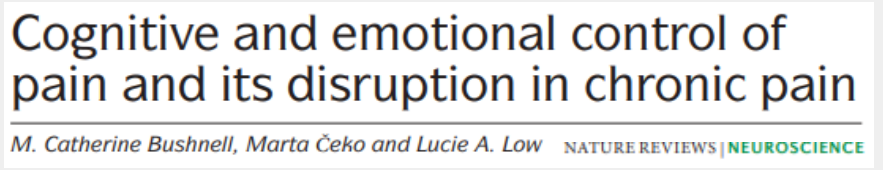
The modulation of this experience/symptom of pain occurs through 3 (three) main pathways:

1 - learning: sensitivity of the nociceptive pathway (the non-associative learning that involves the modulation of the sensitivity of the nociceptive pathway, once the individual comes into contact with the stimulus – processes of sensitization and habituation; this life is unconscious and implicit);

2 - cognition and emotions: measures that will try to help the individual to potentially threatening future situations (predict threatening events or sensations); and

3 - Behavior: how the attempt to modulate symptoms can influence the behavior of the individual, leading to disability (control the symptom).

Molecular imaging studies show decreased binding between endogenous opioids and their receptors in patients with chronic pain, in 3 cortical regions which are the anterior cingulate cortex, prefrontal cortex and the insula.


Cognitive and emotional control of pain and its disruption in chronic pain

M. Catherine Bushnell, Marta Čeko and Lucie A. Low NATURE REVIEWS | NEUROSCIENCE


REVIEW

Deconstructing the sensation of pain: The influence of cognitive processes on pain perception

Katja Wiech[1,2]* SCIENCE sciencemag.org

SPECIAL SECTION PAIN RESEARCH


Topical Review

PAIN

Habituation to pain: a motivational-ethological perspective

Annick L. De Paepe[a,*], Amanda C. de C. Williams[b], Geert Crombez[a]

return

Menu

14 - According to the latest IASP update along with ICD-11, chronic pain is classified as:

A

A sharp pain that has not healed over the course of 12 weeks (3 months);

B

A disease with 7 classifications and subclassifications;

C

A persistent pain for more than 12 weeks (3 months);

D

A persistent pain for more than 12 weeks (3 months) with no signs on imaging tests of tissue healing.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

International Association for the Study of Pain
IASP
Working together for pain relief

FOR IMMEDIATE RELEASE

World Health Assembly of the WHO Approves 11th Version of the International Classification of Diseases (ICD-11), Including New Diagnostic Codes for Chronic Pain

IASP Task Force worked closely with World Health Organization to develop new classification system of chronic pain for improved patient care and research

WASHINGTON, DC – June 3, 2019 – The World Health Organization (WHO) has adopted ICD-11, the latest revision of its International Classification of Diseases, including a new classification system for chronic pain. The decision was made at the World Health Assembly on 25 May 2019.

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

PAIN

OPEN

A classification of chronic pain for *ICD-11*

Rolf-Detlef Treede[a], Winfried Rief[b], Antonia Barke[b,*], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h], Michael B. First[i], Maria Adele Giamberardino[j], Stein Kaasa[k], Eva Kosek[l], Patricia Lavand'homme[m], Michael Nicholas[n], Serge Perrot[o], Joachim Scholz[p], Stephan Schug[q], Blair H. Smith[r], Peter Svensson[s,t], Johan W.S. Vlaeyen[u,v], Shuu-Jiun Wang[w]

Table 1

Glossary of *ICD-11* terms.

WHO term	Explanation
(Diagnostic) entity	The unit of classification, eg, individual diagnoses and diagnostic chapters
Content model	A structured framework that contains all information required to describe an entity within the *ICD*. A content model contains information on an entity's name, its definition, the affected body system or structure, the disease course, its etiology, treatment, and limitations in physical, emotional, or social functioning associated with the entity
Parent/child	Entities are arranged in a hierarchical order, with a "parent" entity at the top, eg, "chronic pain," and child entities subsumed underneath, eg, "chronic neuropathic pain". Child entities can be parent to the next level, eg, "chronic neuropathic pain" is a parent relative to "chronic peripheral neuropathic pain"
Multiple parenting	Entities can have more than 1 parent. An entity such as "chronic chemotherapy-induced pain" has, eg, "chronic cancer pain" and "chronic neuropathic pain" as parents. One of them is designated as the "primary" parent, but the entity can be found under either heading. Multiple parenting thus allows 1 entity to be included in 2 or more diagnostic categories

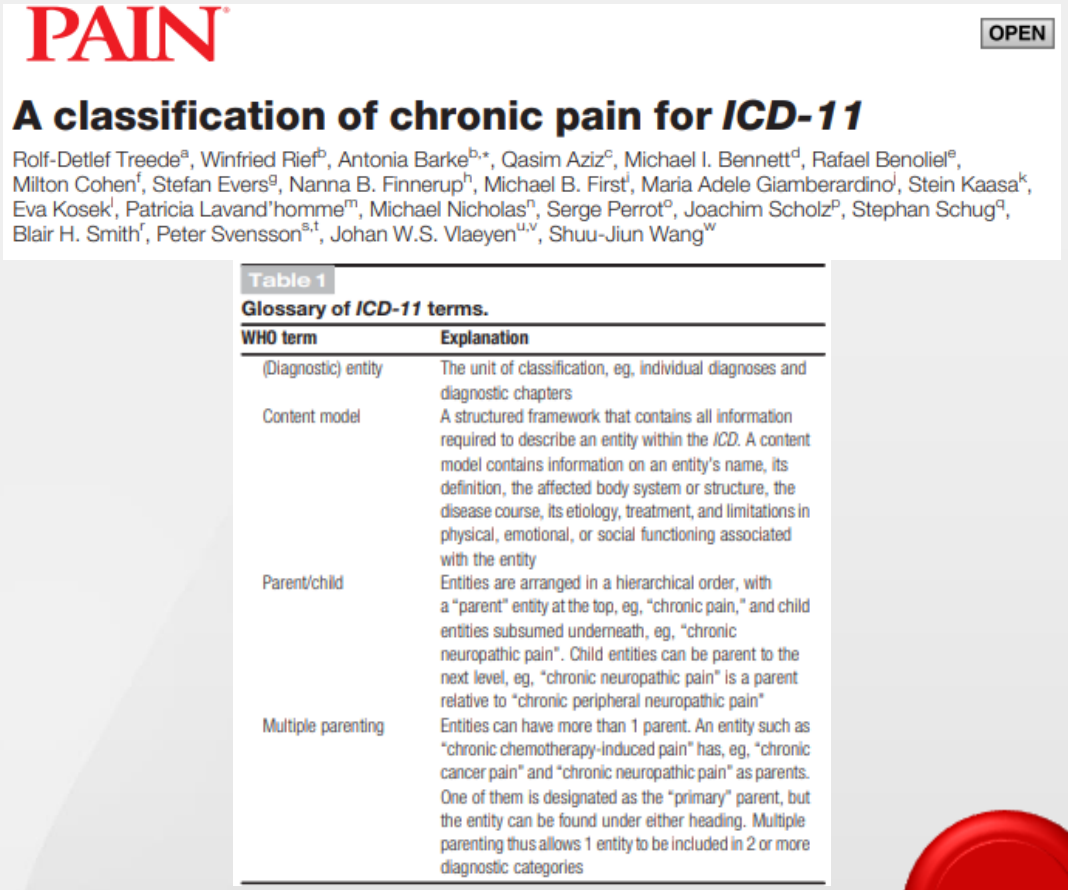
return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

In the coming years, the International Classification of Diseases (ICD-11) will be adopted in several countries around the world. Thus, a revised definition of pain is very timely, and is in line with this and other current efforts to advance the otologic structures within which pain resides.

QUESTIONADOR PODCAST



PAIN

OPEN

A classification of chronic pain for *ICD-11*

Rolf-Detlef Treede[a], Winfried Rief[b], Antonia Barke[b,*], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h], Michael B. First[i], Maria Adele Giamberardino[j], Stein Kaasa[k], Eva Kosek[l], Patricia Lavand'homme[m], Michael Nicholas[n], Serge Perrot[o], Joachim Scholz[p], Stephan Schug[q], Blair H. Smith[r], Peter Svensson[s,t], Johan W.S. Vlaeyen[u,v], Shuu-Jiun Wang[w]

Table 1

Glossary of *ICD-11* terms.

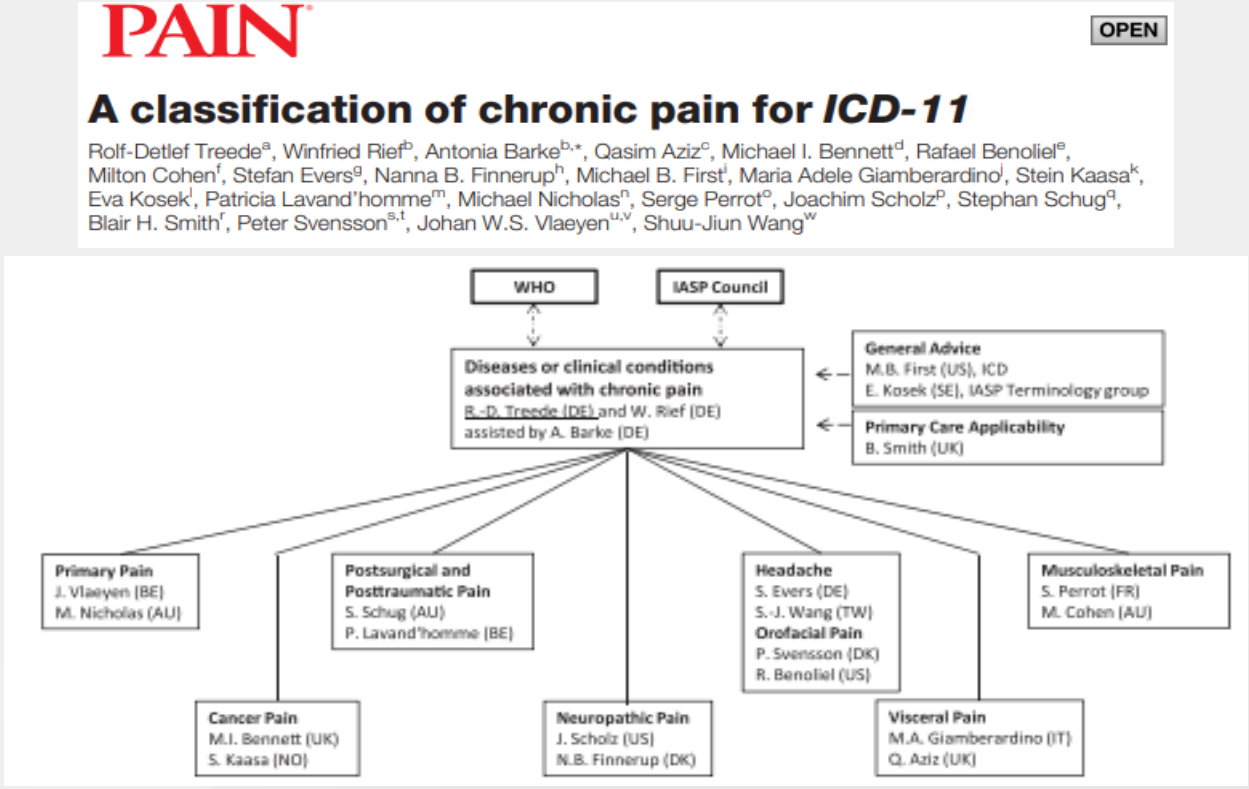
WHO term	Explanation
(Diagnostic) entity	The unit of classification, eg, individual diagnoses and diagnostic chapters
Content model	A structured framework that contains all information required to describe an entity within the *ICD*. A content model contains information on an entity's name, its definition, the affected body system or structure, the disease course, its etiology, treatment, and limitations in physical, emotional, or social functioning associated with the entity
Parent/child	Entities are arranged in a hierarchical order, with a "parent" entity at the top, eg, "chronic pain," and child entities subsumed underneath, eg, "chronic neuropathic pain". Child entities can be parent to the next level, eg, "chronic neuropathic pain" is a parent relative to "chronic peripheral neuropathic pain"
Multiple parenting	Entities can have more than 1 parent. An entity such as "chronic chemotherapy-induced pain" has, eg, "chronic cancer pain" and "chronic neuropathic pain" as parents. One of them is designated as the "primary" parent, but the entity can be found under either heading. Multiple parenting thus allows 1 entity to be included in 2 or more diagnostic categories

return

Menu

CERTAIN ANSWER!

In 2013, the IASP formed a task force to produce and update a classification of painful diseases for international use. As a result of this work, the new edition of the International Classification of Diseases (ICD-11) that WHO adopted in 2019 included a classification of chronic pain for the first time. In the coming years, ICD-11 will be adopted in several countries. Thus, a revised definition of pain is very timely and aligns with this and other current efforts to advance ontological structures within which pain resides. These combined IASP efforts are important steps toward recognizing pain as an important health condition, transforming pain research and the care of pain people around the world.



return

Menu

15 - Acute pain happens as a direct result of a potential or actual injury to a tissue and is a symptom. Its onset is well defined and relates to a known condition. Chronic pain, on the other hand, does not protect tissues and does not have a clear biological function. Pain can be considered chronic if:

A. The weakness is incompatibly greater than the physical findings, persists beyond the normal recovery time and occurs even in the absence of tissue injury found;

B. Persists beyond normal recovery time and diagnosis is made by imaging;

C. Tissue healing did not occur chronologically within the time established by the IASP of acute pain (3-6 months);

D. When the acute injury has not been adequately treated during its time period.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

ICD-11

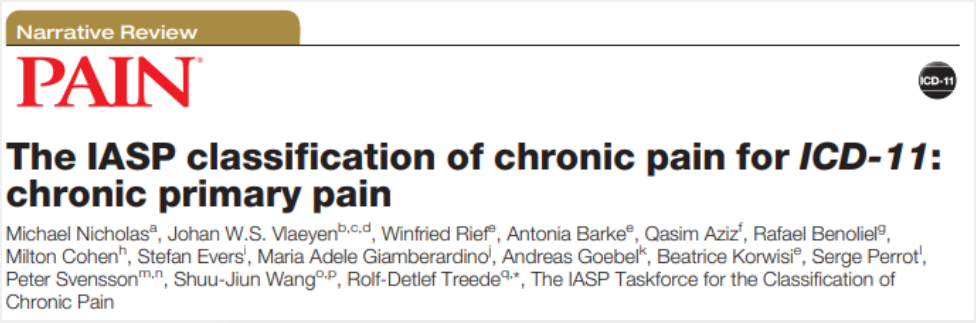
Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the *International Classification of Diseases* (*ICD-11*)

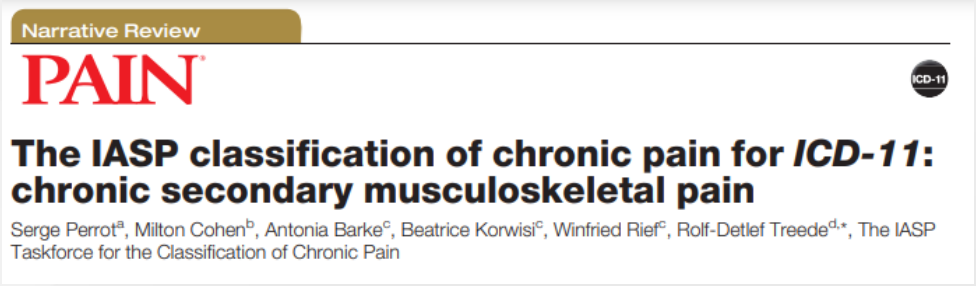
Rolf-Detlef Treede[a,*], Winfried Rief[b], Antonia Barke[b], Qasim Aziz[c], Michael I. Bennett[d], Rafael Benoliel[e], Milton Cohen[f], Stefan Evers[g], Nanna B. Finnerup[h,i], Michael B. First[j], Maria Adele Giamberardino[k], Stein Kaasa[l,m,n], Beatrice Korwisi[b], Eva Kosek[o], Patricia Lavand'homme[p], Michael Nicholas[q], Serge Perrot[r], Joachim Scholz[s], Stephan Schug[t,u], Blair H. Smith[v], Peter Svensson[w,x], Johan W.S. Vlaeyen[y,z,aa], Shuu-Jiun Wang[bb,cc]

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



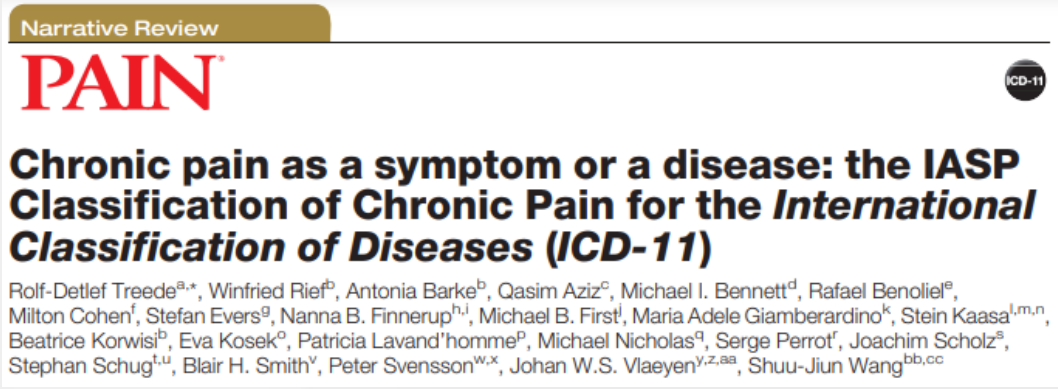


return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

The inclusion of chronic pain in the latest update of the International Classification of Diseases to ICD-11 proposes to lead to better classification and diagnostic coding. Thus, advancing the recognition of chronic pain as a prevalent health condition.



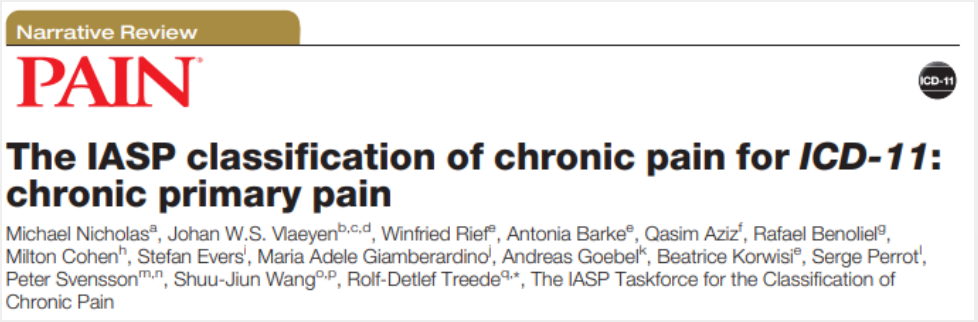
QUESTIONADOR PODCAST

return

Menu

CERTAIN ANSWER!

Chronic pain does not have a clear biological function, it is not considered a symptom, but the disease itself. It has 3 temporal subcategories and 2 associated with origin. The temporal ones are: continuous chronic pain, recurrent chronic pain and chronic pain associated with flare-up. And the two categories are classified into primary and secondary chronic pain. The treatment of chronic pain is complex and the best responses occur when the approach is interdisciplinary. . The implementation of chronic pain in ICD-11 proposes to lead to better classification and diagnostic coding, thus advancing the recognition of chronic pain as a prevalent health condition.

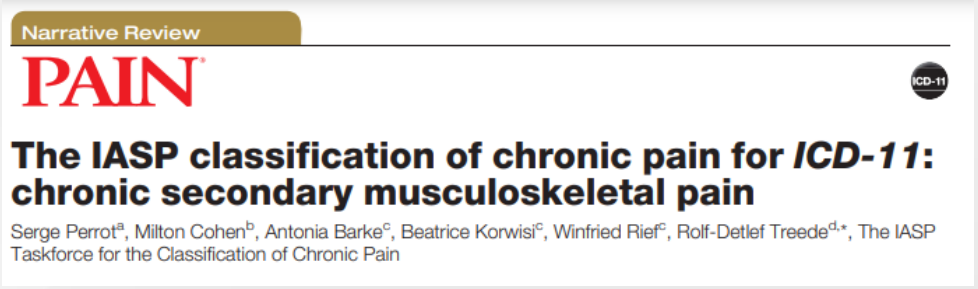

Narrative Review

PAIN

ICD-11

The IASP classification of chronic pain for *ICD-11*: chronic primary pain

Michael Nicholas[a], Johan W.S. Vlaeyen[b,c,d], Winfried Rief[e], Antonia Barke[e], Qasim Aziz[f], Rafael Benoliel[g], Milton Cohen[h], Stefan Evers[i], Maria Adele Giamberardino[j], Andreas Goebel[k], Beatrice Korwisi[e], Serge Perrot[l], Peter Svensson[m,n], Shuu-Jiun Wang[o,p], Rolf-Detlef Treede[q,*], The IASP Taskforce for the Classification of Chronic Pain


Narrative Review

PAIN

ICD-11

The IASP classification of chronic pain for *ICD-11*: chronic secondary musculoskeletal pain

Serge Perrot[a], Milton Cohen[b], Antonia Barke[c], Beatrice Korwisi[c], Winfried Rief[c], Rolf-Detlef Treede[d,*], The IASP Taskforce for the Classification of Chronic Pain

return

Menu

16 - The biopsychial social model proposed by Engle and widely adopted in the world when we talk about the neurophysiology of pain, describes that the initiation, maintenance and perception of pain is influenced by biological, psychosocial factors and factors of the movement system. When we talk about an updated approach to pain, the idea of using neurophysiological mechanisms is more likely to help the patient compared to the action based on signs and symptoms, according to Cliford Wolff and Mitchell Max. What are the neurophysiological mechanisms currently proposed by the IASP?

A — Central sensitization, peripheral sensitization and medullary facilitation;

B — Nociceptive, neuropathic and central sensitization;

C — Nociplastic, neuropathic and peripheral sensitization;

D — Neuropathic, nociceptive and nociplastic.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

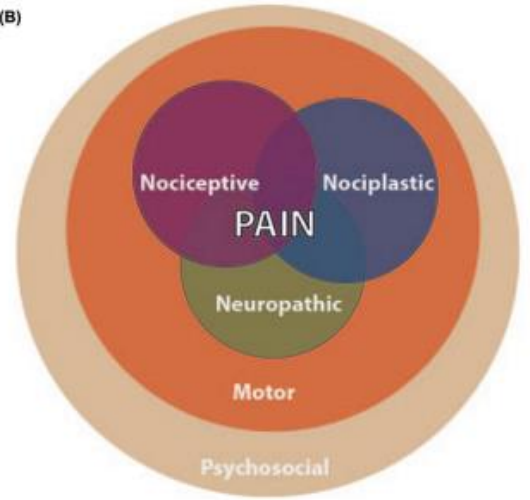
A Mechanism-Based Approach to Physical Therapist Management of Pain

Ruth L. Chimenti, Laura A. Frey-Law, Kathleen A. Sluka

(A)

Nociceptive	Nociplastic	Neuropathic
•Due to activation of nociceptors ·Inflammation ·Mechanical irritant ·Injury •Examples ·Osteoarthritis ·Ankle sprain ·Rheumatoid arthritis	•Due to disturbance in central pain processing ·↑excitability ·↓inhibition •Examples ·Fibromyalgia ·Temporomandibular disorder ·Nonspecific low back pain	•Due to lesion or disease of the somatosensory system •Examples ·Diabetic neuropathy ·Carpal tunnel syndrome ·Complex regional pain syndrome

(B)



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Table 2

Proposed taxonomy for the classification of pain compared with the existing IASP taxonomy from 2011 (http://www.iasp-pain.org/Taxonomy), changes highlighted.

Descriptor	Definition	Notes
Nociceptive pain	Pain that arises from actual or threatened damage to nonneural tissue and is due to the activation of nociceptors	***The term is used to describe pain occurring with a normally functioning somatosensory nervous system***
Neuropathic pain	Pain caused by a lesion or disease of the somatosensory nervous system	Neuropathic pain is a clinical description (and not a diagnosis) that requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria. The term *lesion* is commonly used when diagnostic investigations (eg, imaging, neurophysiology, biopsies, laboratory tests) reveal an abnormality or when there was obvious trauma. The term *disease* is commonly used when the underlying cause of the lesion is known (eg, stroke, vasculitis, diabetes mellitus, genetic abnormality). *Somatosensory* refers to information about the body per se including visceral organs, rather than information about the external world (eg, vision, hearing, or olfaction). The presence of symptoms or signs (eg, touch-evoked pain) alone does not justify the use of the term *neuropathic*. Some disease entities, such as trigeminal neuralgia, are currently defined by their clinical presentation rather than by objective diagnostic testing. Other diagnoses such as postherpetic neuralgia are normally based on the history. It is common when investigating neuropathic pain that diagnostic testing may yield inconclusive or even inconsistent data. In such instances, clinical judgment is required to reduce the totality of findings in a patient into one putative diagnosis or concise group of diagnoses
Nociplastic/algopathic/nocipathic pain	***Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain***	***Patients can have a combination of nociceptive and nociplastic/algopathic/nocipathic pain***
Pain of unknown origin (previously idiopathic pain)	***Pain of unknown cause and origin***	***Pain that cannot be classified as neuropathic, nociceptive or nociplastic/algopathic/nocipathic***

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Contents lists available at ScienceDirect

Musculoskeletal Science and Practice

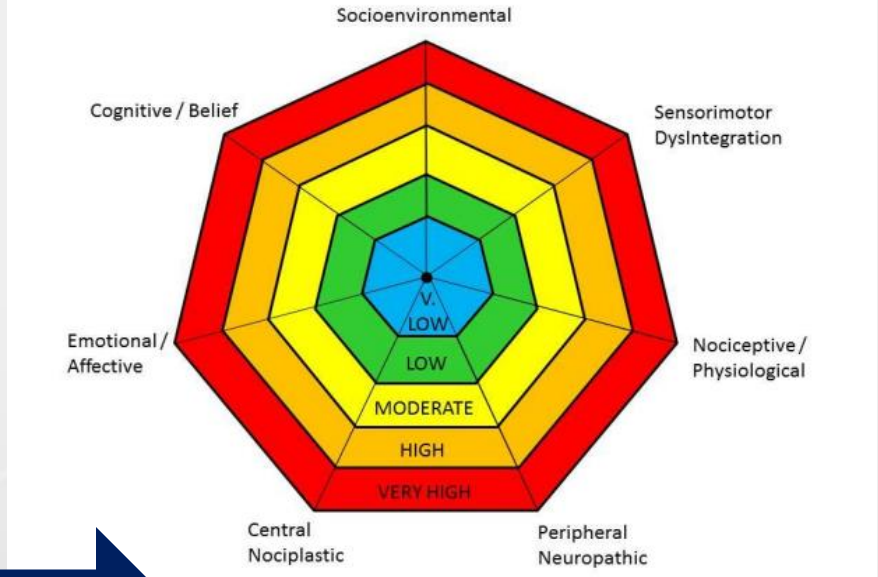
journal homepage: www.elsevier.com/locate/msksp

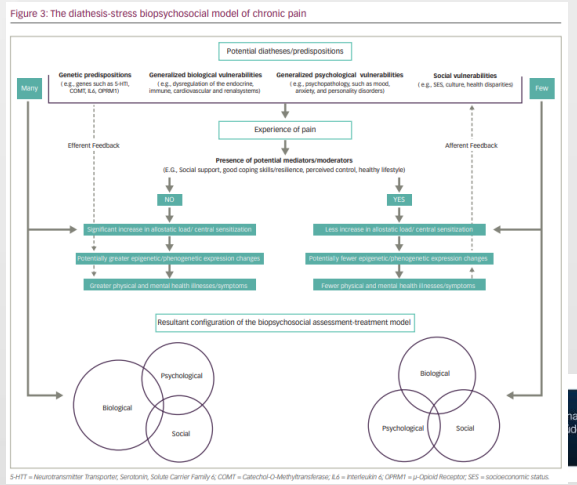
Original article

A new clinical model for facilitating the development of pattern recognition skills in clinical pain assessment*

David M. Walton[a,*], James M. Elliott[b]

[a] Faculty of Health Science, Western University Canada, Canada
[b] Faculty of Health Sciences, The University of Sydney, and the Kolling Institute, Royal North Shore Hospital, NSW, Australia





return

Menu

CERTAIN ANSWER!

The neurophysiological mechanisms present a probability of greater help to the patient when compared to the model of signs and symptoms only. They were updated in 2016 by the IASP, and the last mechanism to compose the described framework was the nociplastic mechanism. The object of this review in 2016 was to propose a debate through a third mechanism aimed at characterizing chronic pain through an altered nociceptive function. They are categorized into 3 classes. They are: nociceptive: pain due to actual or potential damage to non-neural tissue from the activation of nociceptors; nociplastic: pain due to changes in nociception despite absence of evidence of actual or potential tissue damage, causing activation of peripheral nociceptors or evidence of disease or injury of the somatosensory system causing the pain; and neuropathic: pain caused by an injury or disease of the somatosensory system.

return

Menu

17 - The nociceptive mechanism is composed of 3 (three) subclassifications, they are:

A. Chemistry, non-inflammatory chemistry and tissue maintenance;

B. Chemical, thermal and mechanical;

C. Mechanics, chemistry and motor;

D. Chemistry, central and peripheral.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Research Paper

PAIN®

Features and methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: a Delphi expert consensus study

Muath A. Shraim[a], Kathleen A. Sluka[b], Michele Sterling[c], Lars Arendt-Nielsen[d], Charles Argoff[e], Karl S. Bagraith[f], Ralf Baron[g], Helena Brisby[h], Daniel B. Carr[i], Ruth L. Chimenti[j], Carol A. Courtney[k], Michele Curatolo[l], Beth D. Darnall[m], Jon J. Ford[n], Thomas Graven-Nielsen[o], Melissa C. Kolski[p], Eva Kosek[q,r], Richard E. Liebano[s], Shannon L. Merkle[t], Romy Parker[u], Felipe J. J. Reis[v,w], Keith Smart[x], Rob J. E. M. Smeets[y,z], Peter Svensson[aa], Bronwyn L. Thompson[ab], Rolf-Detlef Treede[ac], Takahiro Ushida[ad], Owen D. Williamson[ae], Paul W. Hodges[a,*]

The Discriminative Validity of "Nociceptive," "Peripheral Neuropathic," and "Central Sensitization" as Mechanisms-based Classifications of Musculoskeletal Pain

Keith M. Smart, PhD, Catherine Blake, PhD,† Anthony Staines, PhD,‡ and Catherine Doody, PhD†*

Delivering transformative action in paediatric pain: a *Lancet Child & Adolescent Health* Commission

Christopher Eccleston, Emma Fisher, Richard F Howard, Rebeccah Slater, Paula Forgeron, Tonya M Palermo, Kathryn A Birnie, Brian J Anderson, Christine T Chambers, Geert Crombez, Gustaf Ljungman, Isabel Jordan, Zachary Jordan, Caitriona Roberts, Neil Schechter, Christine B Sieberg, Dick Tibboel, Suellen M Walker, Dominic Wilkinson, Chantal Wood

www.thelancet.com/child-adolescent **Published online October 13, 2020** https://doi.org/10.1016/S2352-4642(20

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Delivering transformative action in paediatric pain: a *Lancet Child & Adolescent Health* Commission

Christopher Eccleston, Emma Fisher, Richard F Howard, Rebeccah Slater, Paula Forgeron, Tonya M Palermo, Kathryn A Birnie, Brian J Anderson, Christine T Chambers, Geert Crombez, Gustaf Ljungman, Isabel Jordan, Zachary Jordan, Caitriona Roberts, Neil Schechter, Christine B Sieberg, Dick Tibboel. Suellen M Walker. Dominic Wilkinson. Chantal Wood

***Panel 3*: Pain definition and classifications**

In 2020, a new International Association for the Study of Pain task force proposed an updated definition of pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage",[86] with added text to recognise that, in many circumstances, pain could not be verbally mediated: "Verbal description is only one of several behaviours to express pain; inability to communicate does not negate the possibility that a human or a non-human animal experiences pain".[86] Pain can be classified or described in multiple ways, some of the most frequently used include:

By somatosensory mechanism

- Nociceptive pain: pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors (ie, pain-detecting nerves). Nociceptive pain is the mechanism operating in most everyday painful experiences and, when it results from an injury or a damage, it should resolve when healing has occurred. In infants, children, and throughout later development, the mechanisms of nociceptive pain change with age.
- Neuropathic pain: pain caused by a lesion or disease of the somatosensory nervous system. When the system that detects pain is itself damaged, it can generate pain, although it might not respond to a previously painful stimulus. Cellular and molecular mechanisms of neuropathic pain are different from those of nociceptive pain, and are less likely to resolve with the healing process. During development and maturation, the mechanisms and clinical presentations of neuropathic pain differ with age and depend on the underlying cause of damage.
- Nociplastic pain: pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain. Changes in nociceptive processing mechanisms can be shown in some individuals for whom a clear underlying cause is not detectable by currently available methods.

By time

- Acute pain: pain that lasts ≤3 months (eg, acute postoperative pain and vaccination pain). Mechanisms of acute pain are mostly nociceptive and resolution is normally expected when healing occurs.
- Chronic pain: pain that lasts or recurs for ≥3 months (eg, chronic musculoskeletal pain and chronic disease-related pain). Chronic pain can involve nociceptive, neuropathic, and nociplastic mechanisms.
- In clinical situations, pain might also be described as continuous (ie, background pain) or intermittent (ie, episodic pain), or as either predictable (ie, incident) or unpredictable (ie, spontaneous).

By context or location

- Disease-related pain: pain that is associated with specific diagnoses or conditions (eg, juvenile inflammatory arthritis and cancer pain).
- Tissue or organ-dependent pain: pain arising from specific tissues or organs (eg, visceral, musculoskeletal [associated with bone, joint, and muscle], headaches, and pelvic pain).
- Iatrogenic pain: pain associated with or following medical treatments (eg, procedure pain including vaccination, surgical, or medical [eg, chemotherapy-induced neuropathy] interventions).
- Idiopathic pain (also known as functional or primary pain): pain for which there is no clear identified cause (eg, chronic primary abdominal pain)

When pain is described in terms of context, mechanisms might be nociceptive, neuropathic, or nociplastic, and could also be acute or chronic.

www.thelancet.com/child-adolescent **Published online October 13, 2020** https://doi.org/10.1016/S2352-4642(20

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Topical Review

PAIN

Updating the definition of pain

Amanda C. de C. Williams[a,*], Kenneth D. Craig[b]

1. Introduction

The definition of pain[24] promulgated by the IASP (Box 1) has provided a powerful conceptual anchor for scientific and health care professional advances in understanding the nature and treatment of acute and chronic pain. Based on work by Merskey in 1964,[22] it has been widely endorsed and even more widely used,[27] with the primary text unchanged since first published in 1979.[15] Since then, there have been substantial advances in our understanding of pain, in assessment and treatment, using a multidisciplinary perspective, and emergence of chronic disease models. These advances instantiate the biopsychosocial perspective on pain that was required to capture evidence-based understanding and the evolution of pain care. While the IASP definition was under development, Melzack and Wall[21] (1965) published "Pain Mechanisms: A New Theory" in *Science*, generating a revolution in our understanding of pain mechanisms and management.[16]

In light of these advances, a review of the definition of pain seems warranted. We provide a rationale explaining why a revised definition better captures the essence of what we presently understand to be pain and how it would better equip those who try to control pain. The following definition is proposed:

Pain is a distressing experience associated with actual or potential tissue damage with sensory, emotional, cognitive, and social components.

Text box 1

Pain definition.

As updated from "Part III: Pain Terms, A Current List with Definitions and Notes on Usage" (pp 209–214), *Classification of Chronic Pain, Second Edition, IASP Task Force on Taxonomy*, edited by H. Merskey and N. Bogduk, IASP Press, Seattle, ©1994. http://www.iasp-pain.org/Taxonomy#Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note: The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment. Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli that cause pain are liable to damage tissue. Accordingly, pain is that experience we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant, and therefore also an emotional experience. Experiences that resemble pain but are not unpleasant, eg, pricking, should not be called pain. Unpleasant abnormal experiences (dysesthesias) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain. Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain, and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, although we may well appreciate that pain most often has a proximate physical cause.

return

Menu

CERTAIN ANSWER!

It is a pain due to actual or potential damage to non-neural tissue from the activation of nociceptors; there are a number of drivers (generating sources) of predominantly nociceptive pain. It has 3 (three) subclassifications described by the IASP currently, they are: chemistry, non-inflammatory chemistry and tissue maintenance.

Narrative Review

PAIN

The IASP classification of chronic pain for *ICD-11*: functioning properties of chronic pain

Boya Nugraha[a], Christoph Gutenbrunner[a], Antonia Barke[b], Matthias Karst[c], Jörg Schiller[a], Peter Schäfer[a], Silke Falter[a,d], Beatrice Korwisi[b], Winfried Rief[b], Rolf-Detlef Treede[e,*], The IASP Taskforce for the Classification of Chronic Pain

American Pain Society — RESEARCH EDUCATION TREATMENT ADVOCACY

PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 15, No 3 (March), 2014: pp 241-249
Available online at www.jpain.org and www.sciencedirect.com

Focus Article

The ACTTION-American Pain Society Pain Taxonomy (AAPT): An Evidence-Based and Multidimensional Approach to Classifying Chronic Pain Conditions

Roger B. Fillingim,* Stephen Bruehl,† Robert H. Dworkin,‡ Samuel F. Dworkin,§ John D. Loeser,¶ Dennis C. Turk,‖ Eva Widerstrom-Noga,# Lesley Arnold,** Robert Bennett,†† Robert R. Edwards,‡‡ Roy Freeman,§§ Jennifer Gewandter,¶¶ Sharon Hertz,‖‖ Marc Hochberg,## Elliot Krane,*** Patrick W. Mantyh,††† John Markman,‡‡‡ Tuhina Neogi,§§§ Richard Ohrbach,¶¶¶ Judith A. Paice,‖‖‖ Frank Porreca,### Bob A. Rappaport,**** Shannon M. Smith,†††† Thomas J. Smith,‡‡‡‡ Mark D. Sullivan,§§§§ G. Nicholas Verne,¶¶¶¶ Ajay D. Wasan,‖‖‖‖ and Ursula Wesselmann####

return

Menu

18 - The neuropathic mechanism is composed of 2 (two) subclassifications, they are:

A — Localized and diffuse;

B — Diffuse and generalized;

C — Central and peripheral;

D — Peripheral and somatic.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Physiol Rev 101: 259–301, 2021
First published June 25, 2020; doi: 10.1152/physrev.00045.2019

NEUROPATHIC PAIN: FROM MECHANISMS TO TREATMENT

Nanna Brix Finnerup, Rohini Kuner, and Troels Staehelin Jensen

Danish Pain Research Center, Department of Clinical Medicine, Aarhus University, Aarhus, Denmark; Department of Neurology, Aarhus University Hospital, Aarhus, Denmark; and Department of Pharmacology, Heidelberg University, Heidelberg, Germany

Possible neuropathic pain

History of relevant neurological lesion or disease[a]
Pain distribution neuroanatomically plausible[b]

Probable neuropathic pain

Pain is associated with sensory signs in the same neuroanatomically plausible distribution on clinical examination[c]

Confirmed neuropathic pain

Diagnostic test confirming a lesion or disease of the somatosensory nervous system explaining the pain[d]

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

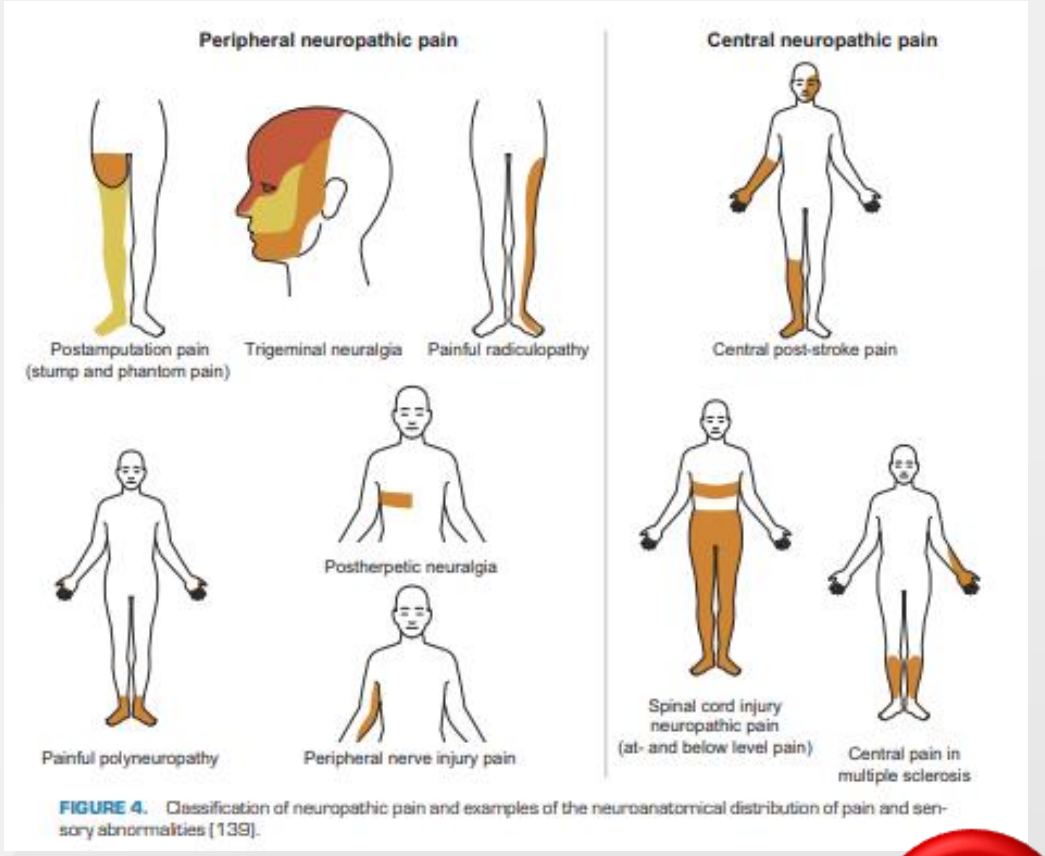


FIGURE 4. Classification of neuropathic pain and examples of the neuroanatomical distribution of pain and sensory abnormalities [139].

return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

The signs and symptoms presented by the patient, such as decrease or loss of sensitivity, indicating possible involvement of the somatosensory system, must be compatible with the territory of innervation of the affected nervous structure.

QUESTIONADOR PODCAST

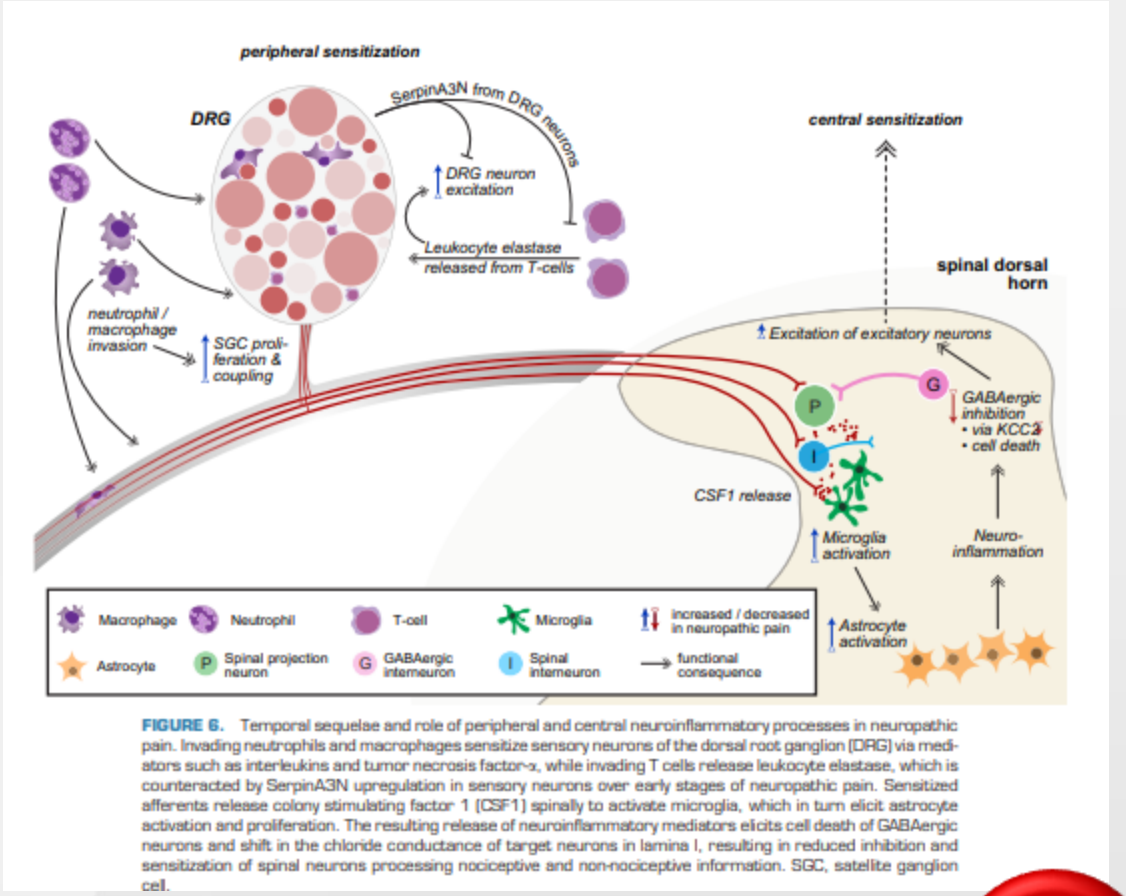


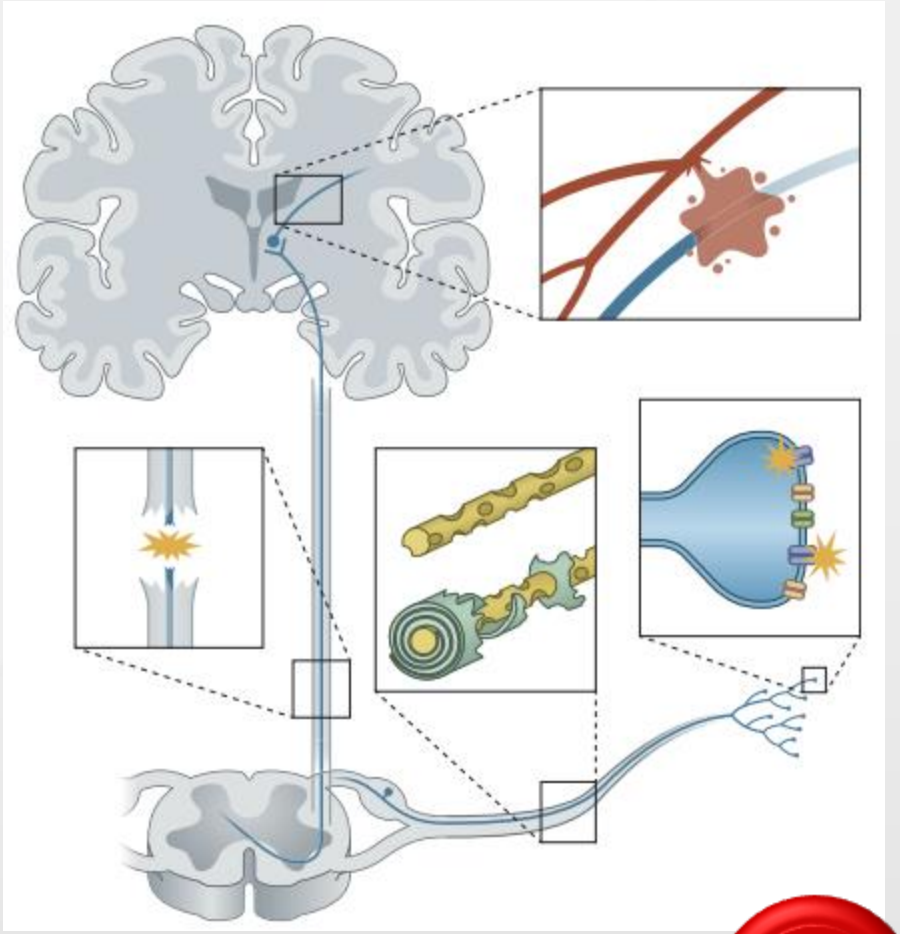
FIGURE 6. Temporal sequelae and role of peripheral and central neuroinflammatory processes in neuropathic pain. Invading neutrophils and macrophages sensitize sensory neurons of the dorsal root ganglion (DRG) via mediators such as interleukins and tumor necrosis factor-α, while invading T cells release leukocyte elastase, which is counteracted by SerpinA3N upregulation in sensory neurons over early stages of neuropathic pain. Sensitized afferents release colony stimulating factor 1 (CSF1) spinally to activate microglia, which in turn elicit astrocyte activation and proliferation. The resulting release of neuroinflammatory mediators elicits cell death of GABAergic neurons and shift in the chloride conductance of target neurons in lamina I, resulting in reduced inhibition and sensitization of spinal neurons processing nociceptive and non-nociceptive information. SGC, satellite ganglion cell.

return

Menu

CERTAIN ANSWER!

Pain caused by an injury or disease of the somatosensory nervous system; even if the underlying condition has been treated or cured. Neuropathic pain may be spontaneous or evoked as an increased response to a painful stimulus. Diagnosis requires a history of injury or disease of the nervous system and a neuroanatomically plausible distribution of pain; Negative symptoms and signs such as decreased or lost sensation, and positive ones such as hyperalgesia and allodynia, indicating the involvement of the somatosensory nervous system should be compatible with the territory of innervation of the affected nerve structure.



return

Menu

19 - The nociplastic mechanism consists of 2 (two) subclassifications, they are:

A — True and augmented;

B — True and false;

C — Intense and disproportionate;

D — Real and imaginary.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Topical Review

PAIN

Do we need a third mechanistic descriptor for chronic pain states?

Eva Kosek[a,*], Milton Cohen[b], Ralf Baron[c], Gerald F. Gebhart[d], Juan-Antonio Mico[e], Andrew S.C. Rice[f], Winfried Rief[g], A. Kathleen Sluka[h]

Table 1

Historical overview of mechanistic pain terminology.

	Nociceptive	Neuropathic
1994*	Not defined	Pain initiated or caused by a primary lesion or dysfunction in the nervous system
2005*	Pain due to stimulation of primary nociceptive nerve endings	Pain due to lesion or dysfunction of the nervous system
2007-2010	Pain due to activation of primary nociceptors Pain arising from activation of nociceptors Pain resulting from noxious stimulation of normal tissue with a normal somatosensory nervous system	
2011*	Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors	Pain caused by a lesion or disease of the somatosensory nervous system

* Adopted by IASP council in those years

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Table 2

Proposed taxonomy for the classification of pain compared with the existing IASP taxonomy from 2011 (http://www.iasp-pain.org/Taxonomy), changes highlighted.

Descriptor	Definition	Notes
Nociceptive pain	Pain that arises from actual or threatened damage to nonneural tissue and is due to the activation of nociceptors	***The term is used to describe pain occurring with a normally functioning somatosensory nervous system***
Neuropathic pain	Pain caused by a lesion or disease of the somatosensory nervous system	Neuropathic pain is a clinical description (and not a diagnosis) that requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria. The term *lesion* is commonly used when diagnostic investigations (eg, imaging, neurophysiology, biopsies, laboratory tests) reveal an abnormality or when there was obvious trauma. The term *disease* is commonly used when the underlying cause of the lesion is known (eg, stroke, vasculitis, diabetes mellitus, genetic abnormality). *Somatosensory* refers to information about the body per se including visceral organs, rather than information about the external world (eg, vision, hearing, or olfaction). The presence of symptoms or signs (eg, touch-evoked pain) alone does not justify the use of the term *neuropathic*. Some disease entities, such as trigeminal neuralgia, are currently defined by their clinical presentation rather than by objective diagnostic testing. Other diagnoses such as postherpetic neuralgia are normally based on the history. It is common when investigating neuropathic pain that diagnostic testing may yield inconclusive or even inconsistent data. In such instances, clinical judgment is required to reduce the totality of findings in a patient into one putative diagnosis or concise group of diagnoses
Nociplastic/algopathic/nocipathic pain	***Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain***	***Patients can have a combination of nociceptive and nociplastic/algopathic/nocipathic pain***
Pain of unknown origin (previously idiopathic pain)	***Pain of unknown cause and origin***	***Pain that cannot be classified as neuropathic, nociceptive or nociplastic/algopathic/nocipathic***

return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

Nociplastic relates to nociceptive plasticity and has been described to reflect a change in the function of nociceptive pathways.

QUESTIONADOR PODCAST

Topical Review

PAIN

Do we need a third mechanistic descriptor for chronic pain states?

Eva Kosek[a,*], Milton Cohen[b], Ralf Baron[c], Gerald F. Gebhart[d], Juan-Antonio Mico[e], Andrew S.C. Rice[f], Winfried Rief[g], A. Kathleen Sluka[h]

1. Introduction

The redefinition of neuropathic pain,[2,3] which specifically excludes the concept of "dysfunction," has left a large group of patients without a valid pathophysiological descriptor for their experience of pain. This group comprises people who have neither obvious activation of nociceptors nor neuropathy (defined as disease or damage of the somatosensory system) but in whom clinical and psychophysical findings suggest altered nociceptive function. Typical such patient groups include those labelled as having fibromyalgia, complex regional pain syndrome (CRPS) type 1, other instances of "musculoskeletal" pain (such as "nonspecific" chronic low-back pain), and "functional" visceral pain disorders (such as irritable bowel syndrome, bladder pain syndrome). The aim of this topical review was to propose, for debate, a third mechanistic descriptor intended for chronic pain characterized by altered nociceptive function.

1.1. Historical review

Before developing any argument for a third descriptor to accommodate these patients, it is worthwhile reviewing the history of pain terminology. Traditionally, pain mechanisms have been divided into "nociceptive" and "neuropathic" categories. See **Table 1** for the historical overview of these definitions.

1.2. Implications of the changed definition of "neuropathic pain"

In the 2005 iteration, "nociceptive" pain was the norm, the "default" or common sense experience of injury = damage ≤pain, familiar to humans. But it evolved that *any* pain that was not "nociceptive" might be termed "neuropathic" because the latter descriptor included "dysfunction," which was taken to include any inferred change in nociceptive function. Although it has always been possible to invoke another category, such as "unknown" or "idiopathic," that strategy runs a poor third to the other 2, as there is no implication of a putative mechanism.

The 2011 redefinition of neuropathic pain makes biological and etymological sense. The note that accompanies this definition is stringent: *Neuropathic pain is a clinical description (and not a diagnosis)*, which requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria. This robust definition is not being challenged.

However, the note that accompanies the 2011 redefinition of *nociceptive* pain—pain that arises from actual or threatened damage to nonneural tissue and is due to activation of nociceptors—states: *This term* ***is designed to contrast with neuropathic pain****. The term is used to describe pain occurring with a normally functioning somatosensory nervous system to contrast with the abnormal function seen in neuropathic pain* (emphasis added). This perpetuates the "nocieptive–neuropathic" dichotomy as above, except that now the "default" position is neuropathic pain, so that any pain condition that is not characterized by damage to neuronal tissue may attract the term "nociceptive." This is not only counterintuitive, as surely "a normally functioning somatosensory nervous system" should be taken as the basis for any contrast, but also it fails to accommodate a large group of patients in whom "activation of nociceptors" cannot be confidently established.

2. Proposals

This situation requires clarification. The prop
here, as presented in **Table 2**, include:

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

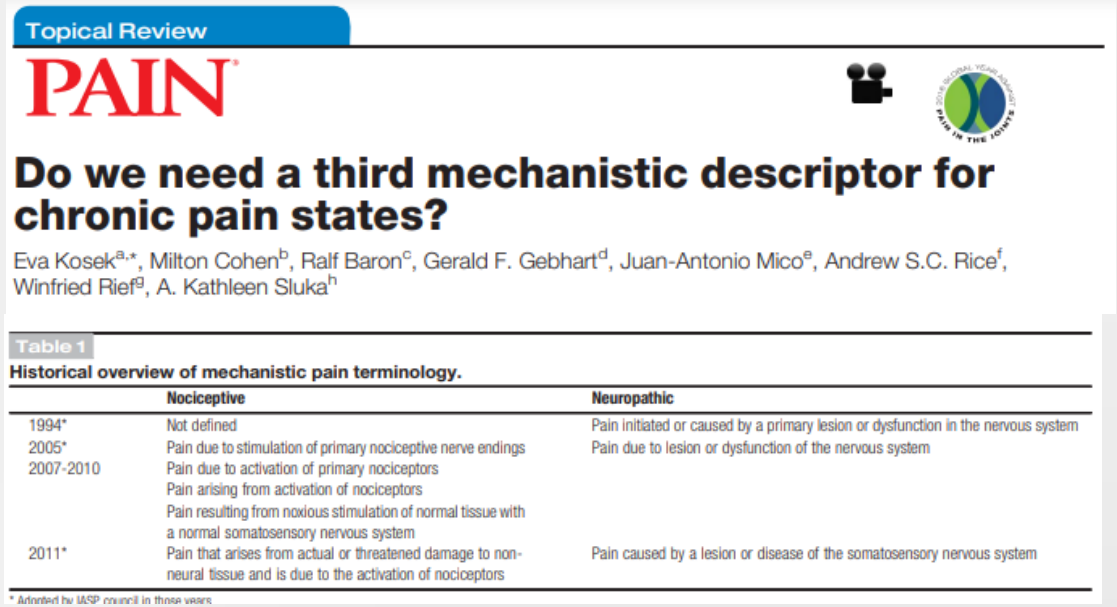
[a] *Department of Clinical Neuroscience, Karolinska Institutet and Stockholm Spine Center, Stockholm, Sweden,* [b] *St Vincent's Clinical School, UNSW Australia, Sydney, Australia,* [c] *Division of Neurological Pain Research and Therapy, Department of Neurology, Universitaetsklinikum Schleswig-Holstein, Campus Kiel, Kiel, Germany,* [d] *Department of Anesthesiology, Center for Pain Research, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA,* [e] *Department of Neuroscience, CIBER of Mental Health, CIBERSAM, University of Cadiz, Cadiz, Spain,* [f] *Department of Surgery and Cancer, Pain Research, Imperial College*

return

Menu

CERTAIN ANSWER!

It refers to a physiologically-based category, which is particularly applicable to chronic primary pain conditions, described in ICD-11 in 2019 with the WHO, that is, it is pain that arises from abnormal pain processing without any clear evidence of tissue damage or mild pathological, involving the somatosensory system; Interventional procedures are related to worse outcomes in individuals with nociplastic pain when compared to individuals with nociceptive pain.



Topical Review

PAIN

Do we need a third mechanistic descriptor for chronic pain states?

Eva Kosek[a,*], Milton Cohen[b], Ralf Baron[c], Gerald F. Gebhart[d], Juan-Antonio Mico[e], Andrew S.C. Rice[f], Winfried Rief[g], A. Kathleen Sluka[h]

Table 1

Historical overview of mechanistic pain terminology.

	Nociceptive	Neuropathic
1994*	Not defined	Pain initiated or caused by a primary lesion or dysfunction in the nervous system
2005*	Pain due to stimulation of primary nociceptive nerve endings	Pain due to lesion or dysfunction of the nervous system
2007-2010	Pain due to activation of primary nociceptors Pain arising from activation of nociceptors Pain resulting from noxious stimulation of normal tissue with a normal somatosensory nervous system	
2011*	Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors	Pain caused by a lesion or disease of the somatosensory nervous system

* Adopted by IASP council in those years

return

Menu

20 - Chronic pain can be divided into primary and secondary. Primary chronic pain is defined as pain in one or more anatomical regions that persists or recurs for more than three months and is associated with significant emotional distress or functional disability (interference with activities of daily living and participation in social roles) and that cannot be better explained by another chronic pain condition. They are sub-classified into:

A) generalized chronic pain; complex regional pain syndrome type 1; primary chronic orofacial or head pain; primary chronic visceral pain and primary chronic musculoskeletal pain;

B) sporadic chronic pain; visceral pain; orofacial or head pain and musculoskeletal pain;

C) diffuse chronic pain; generalized pain and somatic pain;

D) chronic musculoskeletal pain; chronic visceral pain; chronic orofacial or head pain and psychosomatic pain.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

The IASP classification of chronic pain for *ICD-11*: chronic primary pain

Michael Nicholas[a], Johan W.S. Vlaeyen[b,c,d], Winfried Rief[e], Antonia Barke[e], Qasim Aziz[f], Rafael Benoliel[g], Milton Cohen[h], Stefan Evers[i], Maria Adele Giamberardino[j], Andreas Goebel[k], Beatrice Korwisi[e], Serge Perrot[l], Peter Svensson[m,n], Shuu-Jiun Wang[o,p], Rolf-Detlef Treede[q,*], The IASP Taskforce for the Classification of Chronic Pain

Abstract

This article describes a proposal for the new diagnosis of chronic primary pain (CPP) in *ICD-11*. Chronic primary pain is chosen when pain has persisted for more than 3 months and is associated with significant emotional distress and/or functional disability, and the pain is not better accounted for by another condition. As with all pain, the article assumes a biopsychosocial framework for understanding CPP, which means all subtypes of the diagnosis are considered to be multifactorial in nature, with biological, psychological, and social factors contributing to each. Unlike the perspectives found in *DSM-5* and *ICD-10*, the diagnosis of CPP is considered to be appropriate independently of identified biological or psychological contributors, unless another diagnosis would better account for the presenting symptoms. Such other diagnoses are called "chronic secondary pain" where pain may at least initially be conceived as a symptom secondary to an underlying disease. The goal here is to create a classification that is useful in both primary care and specialized pain management settings for the development of individualized management plans, and to assist both clinicians and researchers by providing a more accurate description of each diagnostic category.

Keywords: *ICD-11*, Classification, Chronic pain, Chronic primary pain, CRPS, CWP, Fibromyalgia, Headache, Orofacial pain, Visceral pain, Musculoskeletal pain, Idiopathic pain, Functional pain

1. Background on chronic primary pain

There are 2 main diagnostic classification systems used internationally for chronic pain, apart from headaches: the *Diagnostic and Statistical Manual* (*DSM*) published by the American Psychiatric Association (APA), and the *International Classification of Diseases* (*ICD*) published by the World Health Organization (WHO). However, both have been found wanting in their accounts of chronic pain conditions. In particular, neither system reflects the developments in

ICD-10 refers to pain attributable exclusively to an underlying pathophysiological mechanism.[19] In the absence of a clear (pathophysiological) etiology, and when biological, psychological, and social factors seem to be contributing to a chronic pain presentation,[15] *ICD-10* offers only the option of "somatoform pain disorder." However, this classification cannot be used when pathophysiological factors are also considered to be contributing to the pain problem.[39]

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Narrative Review

PAIN

The IASP classification of chronic pain for *ICD-11*: chronic primary pain

Michael Nicholas[a], Johan W.S. Vlaeyen[b,c,d], Winfried Rief[e], Antonia Barke[e], Qasim Aziz[f], Rafael Benoliel[g], Milton Cohen[h], Stefan Evers[i], Maria Adele Giamberardino[j], Andreas Goebel[k], Beatrice Korwisi[e], Serge Perrot[l], Peter Svensson[m,n], Shuu-Jiun Wang[o,p], Rolf-Detlef Treede[q,*], The IASP Taskforce for the Classification of Chronic Pain

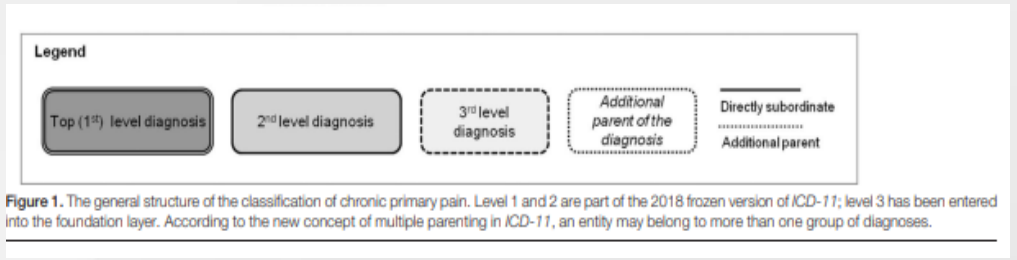


Figure 1. The general structure of the classification of chronic primary pain. Level 1 and 2 are part of the 2018 frozen version of *ICD-11*; level 3 has been entered into the foundation layer. According to the new concept of multiple parenting in *ICD-11*, an entity may belong to more than one group of diagnoses.

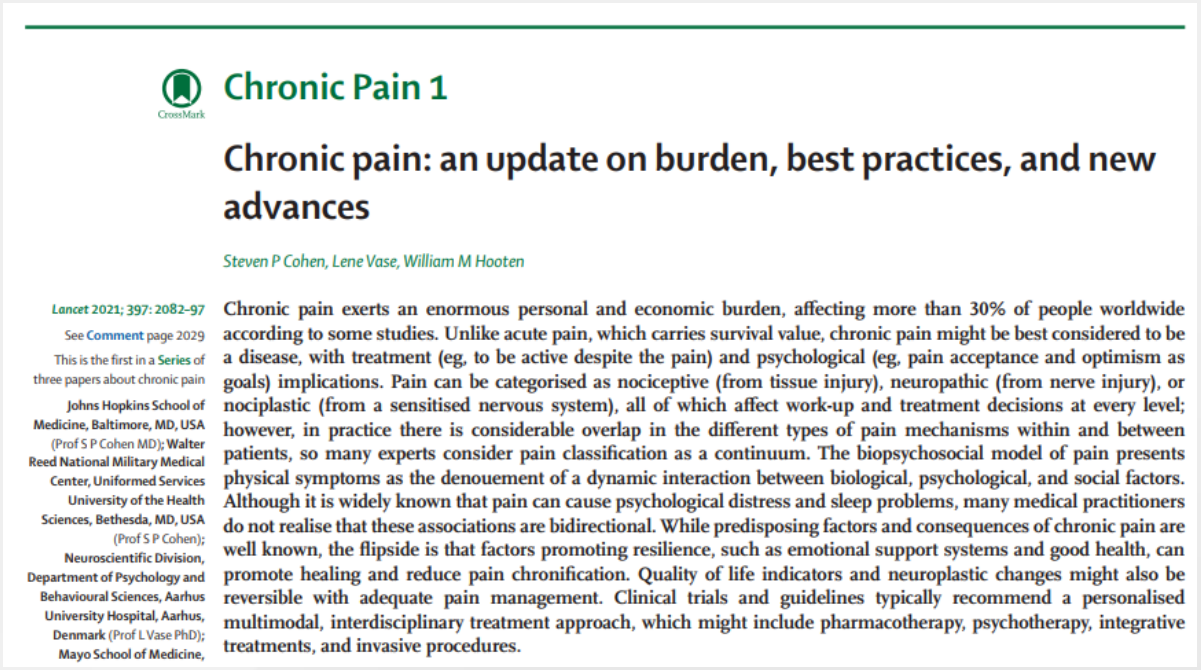
return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

In primary chronic pain, it is generally observed that there is no past history of the patient, that is, there is no detectable event or structural alteration that justifies their pain.

QUESTIONADOR PODCAST



Chronic Pain 1

Chronic pain: an update on burden, best practices, and new advances

Steven P Cohen, Lene Vase, William M Hooten

Lancet 2021; 397: 2082–97

See Comment page 2029

This is the first in a Series of three papers about chronic pain

Johns Hopkins School of Medicine, Baltimore, MD, USA (Prof S P Cohen MD); Walter Reed National Military Medical Center, Uniformed Services University of the Health Sciences, Bethesda, MD, USA (Prof S P Cohen); Neuroscientific Division, Department of Psychology and Behavioural Sciences, Aarhus University Hospital, Aarhus, Denmark (Prof L Vase PhD); Mayo School of Medicine,

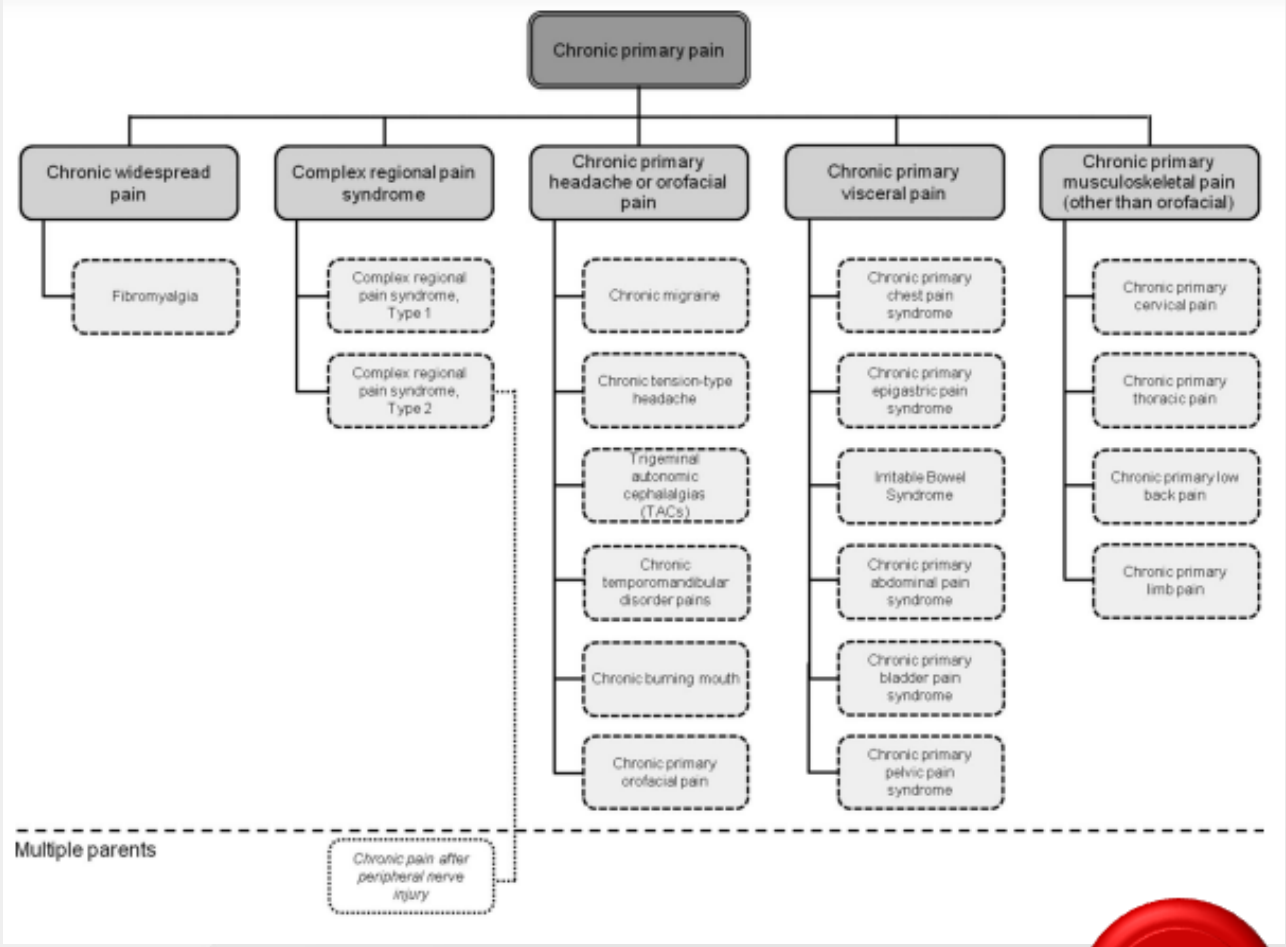
Chronic pain exerts an enormous personal and economic burden, affecting more than 30% of people worldwide according to some studies. Unlike acute pain, which carries survival value, chronic pain might be best considered to be a disease, with treatment (eg, to be active despite the pain) and psychological (eg, pain acceptance and optimism as goals) implications. Pain can be categorised as nociceptive (from tissue injury), neuropathic (from nerve injury), or nociplastic (from a sensitised nervous system), all of which affect work-up and treatment decisions at every level; however, in practice there is considerable overlap in the different types of pain mechanisms within and between patients, so many experts consider pain classification as a continuum. The biopsychosocial model of pain presents physical symptoms as the denouement of a dynamic interaction between biological, psychological, and social factors. Although it is widely known that pain can cause psychological distress and sleep problems, many medical practitioners do not realise that these associations are bidirectional. While predisposing factors and consequences of chronic pain are well known, the flipside is that factors promoting resilience, such as emotional support systems and good health, can promote healing and reduce pain chronification. Quality of life indicators and neuroplastic changes might also be reversible with adequate pain management. Clinical trials and guidelines typically recommend a personalised multimodal, interdisciplinary treatment approach, which might include pharmacotherapy, psychotherapy, integrative treatments, and invasive procedures.

return

Menu

CERTAIN ANSWER!

Primary chronic pain is the disease itself. Its predominant neurophysiological mechanism is nociplastic. It is usually observed when there is no previous history of the patient, that is, there is no detectable event or a structural change that justifies his pain.



return

Menu

21 - According to the inclusion of chronic pain in the ICD-11 and according to the IASP update, it is associated with an increase in the excitability of the nervous system and a decrease in its diffuse harmful inhibitory control. These are risk factors that increase the risk of developing chronic pain:

A. Sleep difficulties, fatigue, psychological changes and sedentary lifestyle;

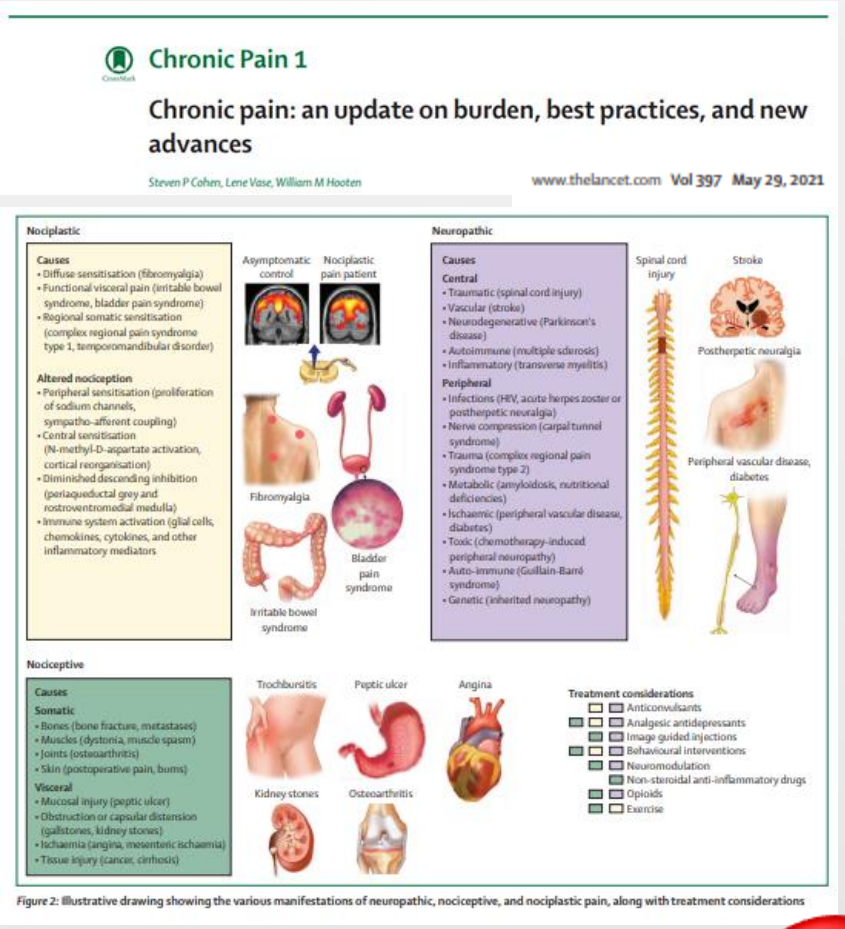
B. Psychological changes, genetic factor, aging and dehydration;

C. Sedentary lifestyle, previous surgery, active biological marker and carbohydrate-restrictive diet;

D. Acute pain not treated properly, active biological marker, sedentary lifestyle and fibromyalgia.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Chronic Pain 1

Chronic pain: an update on burden, best practices, and new advances

Steven P Cohen, Lene Vase, William M Hooten

www.thelancet.com Vol 397 May 29, 2021

Nociplastic

Causes
- Diffuse sensitisation (fibromyalgia)
- Functional visceral pain (irritable bowel syndrome, bladder pain syndrome)
- Regional somatic sensitisation (complex regional pain syndrome type 1, temporomandibular disorder)

Altered nociception
- Peripheral sensitisation (proliferation of sodium channels, sympatho-afferent coupling)
- Central sensitisation (N-methyl-D-aspartate activation, cortical reorganisation)
- Diminished descending inhibition (periaqueductal grey and rostroventromedial medulla)
- Immune system activation (glial cells, chemokines, cytokines, and other inflammatory mediators)

Asymptomatic control

Nociplastic pain patient

Fibromyalgia

Bladder pain syndrome

Irritable bowel syndrome

Neuropathic

Causes

Central
- Traumatic (spinal cord injury)
- Vascular (stroke)
- Neurodegenerative (Parkinson's disease)
- Autoimmune (multiple sclerosis)
- Inflammatory (transverse myelitis)

Peripheral
- Infections (HIV, acute herpes zoster or postherpetic neuralgia)
- Nerve compression (carpal tunnel syndrome)
- Trauma (complex regional pain syndrome type 2)
- Metabolic (amyloidosis, nutritional deficiencies)
- Ischaemic (peripheral vascular disease, diabetes)
- Toxic (chemotherapy-induced peripheral neuropathy)
- Auto-immune (Guillain-Barré syndrome)
- Genetic (inherited neuropathy)

Spinal cord injury

Stroke

Postherpetic neuralgia

Peripheral vascular disease, diabetes

Nociceptive

Causes

Somatic
- Bones (bone fracture, metastases)
- Muscles (dystonia, muscle spasm)
- Joints (osteoarthritis)
- Skin (postoperative pain, burns)

Visceral
- Mucosal injury (peptic ulcer)
- Obstruction or capsular distension (gallstones, kidney stones)
- Ischaemia (angina, mesenteric ischaemia)
- Tissue injury (cancer, cirrhosis)

Trochbursitis

Peptic ulcer

Angina

Kidney stones

Osteoarthritis

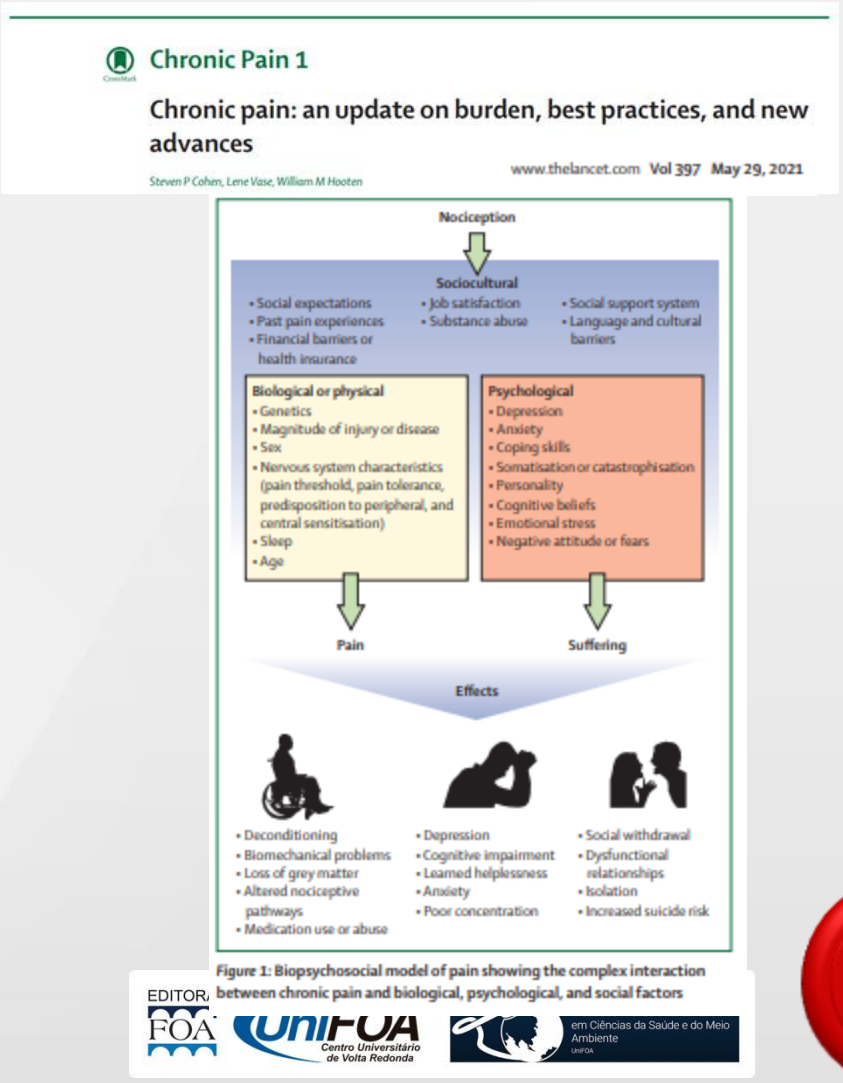
Treatment considerations
- Anticonvulsants
- Analgesic antidepressants
- Image guided injections
- Behavioural interventions
- Neuromodulation
- Non-steroidal anti-inflammatory drugs
- Opioids
- Exercise

Figure 2: Illustrative drawing showing the various manifestations of neuropathic, nociceptive, and nociplastic pain, along with treatment considerations

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

These systemic and social characteristics, within the multidimensional universe, in an adverse way, can be the result or trigger of chronic pain.

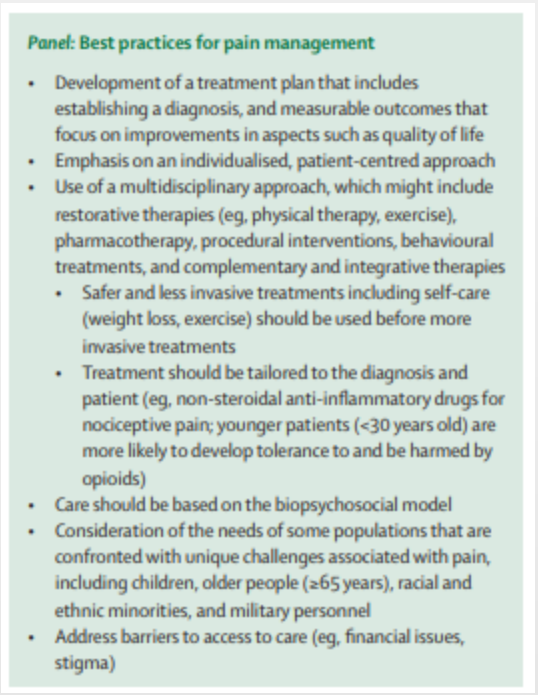
QUESTIONADOR PODCAST

Chronic Pain 1

Chronic pain: an update on burden, best practices, and new advances

Steven P Cohen, Lene Vase, William M Hooten

www.thelancet.com Vol 397 May 29, 2021



Panel: Best practices for pain management

- Development of a treatment plan that includes establishing a diagnosis, and measurable outcomes that focus on improvements in aspects such as quality of life
- Emphasis on an individualised, patient-centred approach
- Use of a multidisciplinary approach, which might include restorative therapies (eg, physical therapy, exercise), pharmacotherapy, procedural interventions, behavioural treatments, and complementary and integrative therapies
  - Safer and less invasive treatments including self-care (weight loss, exercise) should be used before more invasive treatments
  - Treatment should be tailored to the diagnosis and patient (eg, non-steroidal anti-inflammatory drugs for nociceptive pain; younger patients (<30 years old) are more likely to develop tolerance to and be harmed by opioids)
- Care should be based on the biopsychosocial model
- Consideration of the needs of some populations that are confronted with unique challenges associated with pain, including children, older people (≥65 years), racial and ethnic minorities, and military personnel
- Address barriers to access to care (eg, financial issues, stigma)

return

Menu

CERTAIN ANSWER!

The biopsychosocial model posits pain and disability as multidimensional and dynamic interactions between biological, psychological, and social factors that reciprocally influence each other. Features such as depression, anxiety, sleep, and adverse social conditions can be the result of chronic pain. It is less well known that these factors predispose individuals to chronic pain.

Series

Chronic Pain 1

Chronic pain: an update on burden, best practices, and new advances

Chronic pain exerts an enormous personal and economic burden, affecting more than 30% of people worldwide according to some studies. Unlike acute pain, which carries survival value, chronic pain might be best considered to be a disease, with treatment (eg, to be active despite the pain) and psychological (eg, pain acceptance and optimism as goals) implications. Pain can be categorised as nociceptive (from tissue injury), neuropathic (from nerve injury), or nociplastic (from a sensitised nervous system), all of which affect work-up and treatment decisions at every level; however, in practice there is considerable overlap in the different types of pain mechanisms within and between patients, so many experts consider pain classification as a continuum. The biopsychosocial model of pain presents physical symptoms as the downstream of a dynamic interaction between biological, psychological, and social factors. Although it is widely known that pain can cause psychological distress and sleep problems, many medical practitioners do not realise that these associations are bidirectional. While predisposing factors and consequences of chronic pain are well known, the flipside is that factors promoting resilience, such as emotional support systems and good health, can promote healing and reduce pain chronification. Quality of life indicators and neuroplastic changes might also be reversible with adequate pain management. Clinical trials and guidelines typically recommend a personalised multimodal, interdisciplinary treatment approach, which might include pharmacotherapy, psychotherapy, integrative treatments, and invasive procedures.

Introduction

It is difficult to overestimate the burden of chronic pain, which is defined by the International Association for the Study of Pain (IASP) as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.[1] Pain is the main reason why people seek medical care, with three of the top ten reasons being osteoarthritis, back pain, and headaches.[2] Among the four leading causes of years lost to disability, three of these (back pain, musculoskeletal disorders, and neck pain) are chronic pain conditions.[3] Prevalence rates of chronic pain vary between 11% and 40%, with a study by the US Centers for Disease Control and Prevention (CDC) estimating the point prevalence at 20·4%.[4] A systematic review comprising studies done in the UK reported a pooled chronic pain prevalence rate of 43·5%, with the rate of moderate-to-severe disabling pain ranging from 10·4% to 14·3%.[5] A large-scale 4-year longitudinal study, also done in the UK, found the annual incidence rate for chronic pain to be 8·3%, with a recovery rate of 5·4%.[6]

This paper is the first in a Series of three papers about chronic pain, and aims to provide an overview of chronic pain for a non-specialty audience, with emphasis on best practices and selected advances. The areas covered include epidemiology, the classification of pain, overarching models, and management, with the other articles focusing on nociplastic pain[7] and neuromodulation,[8] two areas that have witnessed substantial advances in the past several years but have not been adequately addressed in the general medicine literature.

Not all people are affected by chronic pain equally. Data from the CDC found higher prevalence rates in women, individuals from lower socioeconomic backgrounds, military veterans, and people residing in rural areas.[4] Regarding race and ethnicity, studies are mixed, with some reporting the highest rates among non-Hispanic White people than any other group,[4] whereas most have reported a higher prevalence in racial and ethnic minorities, such as African American people and indigenous populations.[9] Explanations for racial differences include enhanced physiological pain sensitivity, cultural differences, and reduced access to care. When controlling for income amount and adverse life events, differences in prevalence are attenuated but not eliminated.[9] The prevalence of chronic

Search strategy and selection criteria

From January to July, 2020, we searched databases on MEDLINE, Embase, Ovid, and Google using the key words "chronic pain", "neuropathic pain", "non-neuropathic pain", "nociceptive pain", "inflammatory pain", "diffuse pain", and "nociplastic pain", cross-referenced with key words tailored for individual sections (eg, "cost-effectiveness", "biopsychosocial", "cancer", etc) There were no restrictions on article types, date of publication, or language. For the pain management section, key words were chosen on the basis of the treatment(s) and conditions evaluated (eg, "gabapentin" and "neuropathic pain"). For this section, we prioritised systematic reviews, meta-analyses and large, randomised trials, but did not exclude any data sources including publicly available government documents.

2082

return

Menu

22 - In the chapter "Neurobiology of Pain and Analgesia," written by renowned researcher Kathleen A. Sluka, in the book "Electrotherapy Applied to Rehabilitation – From Foundations to Evidence" (Richard E. Liebano, 2021), the central nervous system balances excitatory or inhibitory activity through nociceptive structure and pathways. The goals of treatment are to rebalance the activity of the pathways involved in nociception and pain, therefore:

A) Increasing the stimulation of the direct motor pathway and inhibiting the stimulation of the indirect somesthetic pathway;

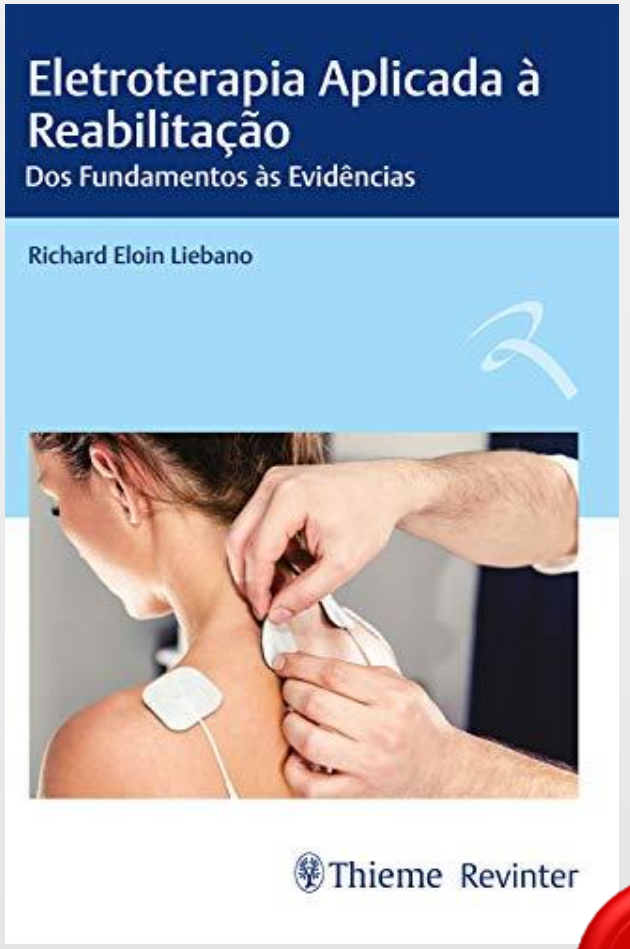
B) Stimulating the sensory pathway and motor pathway (sensorimotor pathway);

C) Reducing the activity of excitatory (facilitatory) pathways and increasing the activity of inhibitory structures;

D) Stimulating the activity of excitatory (facilitatory) pathways and reducing the activity of inhibitory structures.

Menu

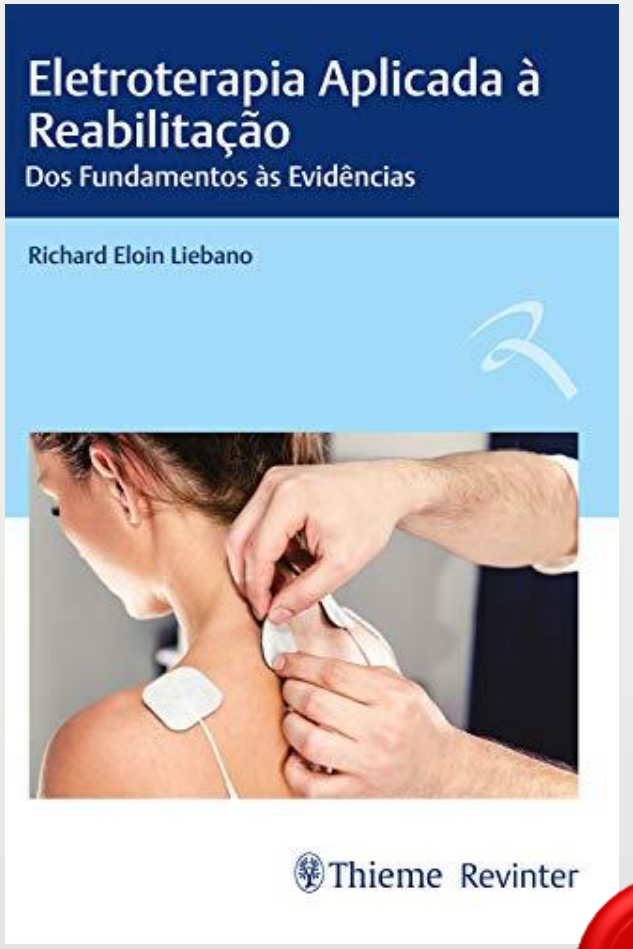
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



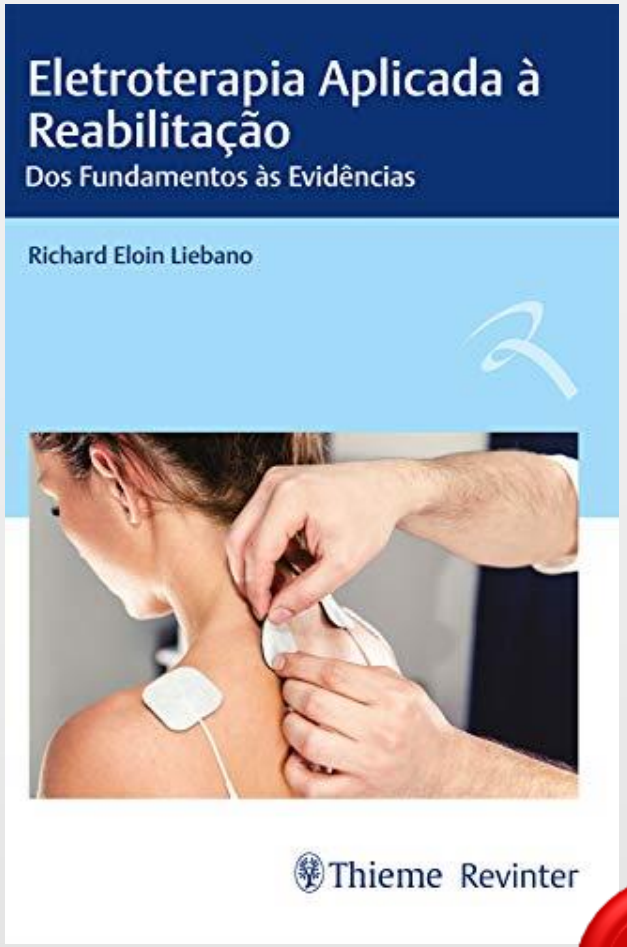
return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

These mechanisms are filtered by two forms of controls or paths. A TOP-DOWN descending path and a BOTTOW-UP ascending path. However, regardless of the system or path, they are entirely influenced and modulated by endogenous factors and also by exogenous factors, psychological and social aspects related to pain.

QUESTIONADOR PODCAST

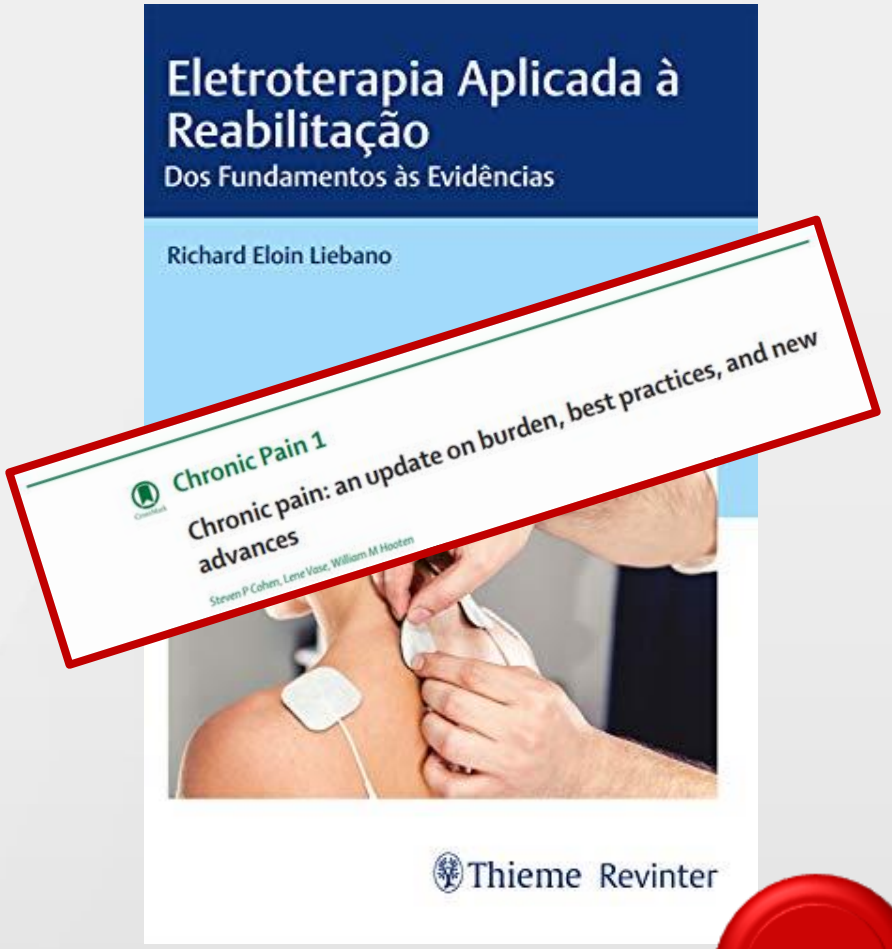


return

Menu

CERTAIN ANSWER!

Regardless of the site of pain (region of the body where it hurts), which system affected (somatic, visceral or neural), the tissue involved (nerves, muscles, cartilage, visceral tissue) even the passive stimuli of not being detected and the passive ones of detection. These mechanisms are filtered by two (2) control species or pathways. A top-down path and a bottow-up path. However, regardless of the system or pathway, they are in their entirety influenced and modulated by endogenous factors, but also by exogenous factors (psychological and social aspects of pain). However, the repercussion at the end is biochemical and neurophysiological, being at the molecular level and difficult to diagnose.



return

Menu

23 - These are questionnaires and scales used in the evaluation of patients with pain:

A. Escala Tampa de CineCentral Sensitization Inventory (CSI), Pain Detect, DASI, WOMAC.siofobia, Inventário de Sensibilização Central (CSI), Escala de Pensamentos Catrastóficos, Pain Detect, Douleur Neuropathique 4 Questions;

B. Ottawa Criteria, WOMAC, IKDC;

C. CADE-Q SV, ELSA, DASI;

D. Central Sensitization Inventory (CSI), Pain Detect, DASI, WOMAC.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

QUESTIONÁRIO PARA DIAGNÓSTICO DE DOR NEUROPÁTICA DN4
(VERSÃO BRASILEIRA 1.0)

Nome: _____

Data: ___/___/_____

Nas quatro perguntas abaixo, complete o questionário marcando uma resposta para cada número.

ENTREVISTA COM O PACIENTE		
Questão 1: A sua dor tem uma ou mais das seguintes características?		
1 – Queimação	( ) SIM	( ) NÃO
2 – Sensação de frio dolorosa	( ) SIM	( ) NÃO
3 – Choque elétrico	( ) SIM	( ) NÃO
Questão 2: Há presença de um ou mais dos seguintes sintomas na mesma área da sua dor?		
4 – Formigamento	( ) SIM	( ) NÃO
5 – Alfinetada e agulhada	( ) SIM	( ) NÃO
6 – Adormecimento	( ) SIM	( ) NÃO
7 – Coceira	( ) SIM	( ) NÃO
EXAME DO PACIENTE		
Questão 3: A dor está localizada numa área onde o exame físico pode revelar uma ou mais das seguintes características?		
8 – Hipoestesia ao toque	( ) SIM	( ) NÃO
9 – Hipoestesia à picada de agulha	( ) SIM	( ) NÃO
Questão 4: Na área dolorosa, a dor pode ser causada ou aumentada por:		
10 – Escovação	( ) SIM	( ) NÃO

Escore	Dor nociceptiva (<4) ( )	Dor neuropática (>=4) ( )

Referências Bibliográficas:

- Bouhassira D et al. Comparison of pain syndromes associated with nervous or somatic lesions and development a new neuropathic pain diagnostic questionnaire (DN4). Pain 2005 Mar; 114 (1-2): 29-36.
- Autores: Karine A. S. Leão Ferreira e Manoel J. Teixeira. Centro Multidisciplinar de Dor do Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo.
- Wiermann et al. Consenso Brasileiro sobre manejo da dor relacionado ao câncer. Rev Brasileira de Oncologia Clínica 2014 Outubro/Novembro/Dezembro; Vol. 10 (38): 132 – 1

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

DOR & NEUROMODULAÇAO – HCPA/CNPq (subárea 2.10.08.00 – 0)

Nome:_____

Sexo: F ( ) M ( ) Escolaridade:_____

Idade:_____ Data: ___/___/____ Testagem:_____

N° banco:_____ Entrevistador:_____

LABORATORY OF PAIN & NEUROMODULATION

Questionário de Sensibilização Central
Brazilian Portuguese Central Sensitization Inventory – BP-CSI

Os sintomas avaliados por este questionário se referem a sua presença diária ou na maioria dos dias dos últimos três meses.

Circule na coluna da direita a melhor resposta para cada questão.

PARTE A

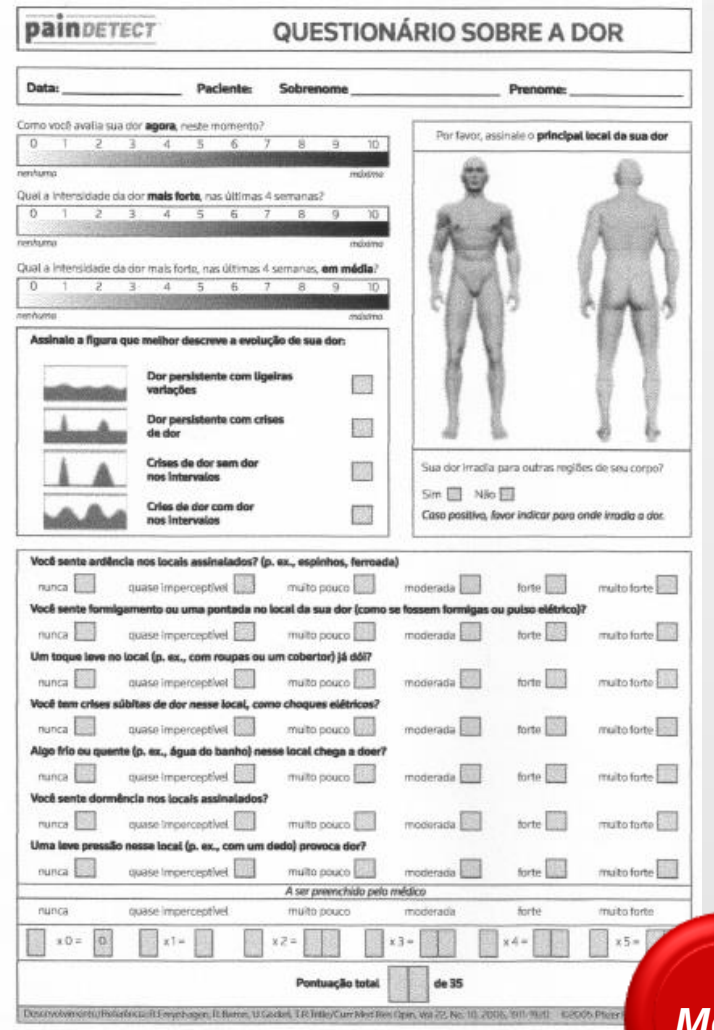
1. Sinto-me cansado (a) ao acordar pela manhã.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
2. Sinto que minha musculatura está enrijecida e dolorida.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
3. Tenho crises de ansiedade.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
4. Costumo apertar (ranger) os dentes.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
5. Tenho diarreia e/ou prisão de ventre.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
6. Preciso de ajuda para fazer as tarefas diárias.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
7. Sou sensível à luminosidade excessiva.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
8. Canso-me facilmente ao realizar atividades diárias que exigem algum esforço físico.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
9. Sinto dor em todo o corpo.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
10. Tenho dores de cabeça.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
11. Sinto desconforto e/ou ardência ao urinar.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
12. Durmo mal.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
13. Tenho dificuldade para me concentrar.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
14. Tenho problemas de pele como ressecamento, coceira e vermelhidão.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*
15. O estresse piora meus sintomas.	*0 Nunca*	*1 Raramente*	*2 Às vezes*	*3 Frequentemente*	*4 Sempre*

Mestrado Profissional em Ensino em Ciências da Saúde e do Meio Ambiente

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



painDETECT QUESTIONÁRIO SOBRE A DOR

Data: _____ Paciente: Sobrenome _____ Prenome: _____

Como você avalia sua dor **agora**, neste momento?

0 1 2 3 4 5 6 7 8 9 10

nenhuma máxima

Qual a intensidade da dor **mais forte**, nas últimas 4 semanas?

0 1 2 3 4 5 6 7 8 9 10

nenhuma máxima

Qual a intensidade da dor mais forte, nas últimas 4 semanas, **em média**?

0 1 2 3 4 5 6 7 8 9 10

nenhuma máxima

Assinale a figura que melhor descreve a evolução de sua dor:

- **Dor persistente com ligeiras variações**
- **Dor persistente com crises de dor**
- **Crises de dor sem dor nos intervalos**
- **Crises de dor com dor nos intervalos**

Por favor, assinale o **principal local da sua dor**

Sua dor irradia para outras regiões de seu corpo?

Sim ☐ Não ☐

Caso positivo, favor indicar para onde irradia a dor.

Você sente ardência nos locais assinalados? (p. ex., espinhos, ferroada)

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Você sente formigamento ou uma pontada no local da sua dor (como se fossem formigas ou pulso elétrico)?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Um toque leve no local (p. ex., com roupas ou um cobertor) já dói?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Você tem crises súbitas de dor nesse local, como choques elétricos?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Algo frio ou quente (p. ex., água do banho) nesse local chega a doer?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Você sente dormência nos locais assinalados?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

Uma leve pressão nesse local (p. ex., com um dedo) provoca dor?

nunca ☐ quase imperceptível ☐ muito pouco ☐ moderada ☐ forte ☐ muito forte ☐

A ser preenchido pelo médico

nunca quase imperceptível muito pouco moderada forte muito forte

x 0 = 0 x 1 = x 2 = x 3 = x 4 = x 5 =

Pontuação total ☐☐ **de 35**

Desenvolvimento: Pfleiderer B, Freyenhagen R, Bennett MI, Gockel HR, Tölle TR, Curr Med Res Opin. Vol 22, No. 10, 2006, 1911–1920. ©2005 Pfleiderer

return

Menu

CERTAIN ANSWER!

Pain is a subjective condition, therefore, difficult to measure. The numerous pain questionnaires are classified and oriented to allow a more objective and reproducible evaluation of the multiple factors that are associated with pain. The questionnaires are completed by the patients themselves and reveal the level and quality of pain, factors such as depression, anxiety, fear, insecurity, disability and catastrophism.

return

Menu

24 - O processamento da dor no cérebro envolve uma rede de estruturas que conferem à experiência dolorosa uma assinatura (neurotag), dentro de um caráter pessoal e individual. Três princípios se relacionam a intensidade dessas assinaturas:

A) Atenção, foco e persistência;

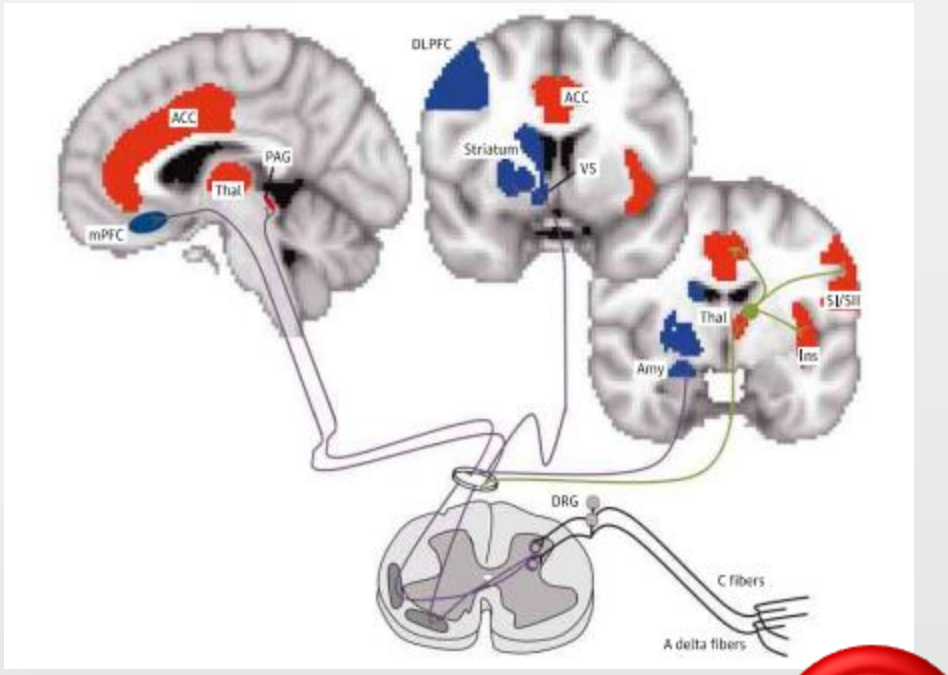
B) Massa neuronal, precisão neural e neuroplasticidade;

C) Neuroplasticidade, persistência e precisão neural;

D) Neuroplasticidade, cognição e neuroplasticidade.

Menu

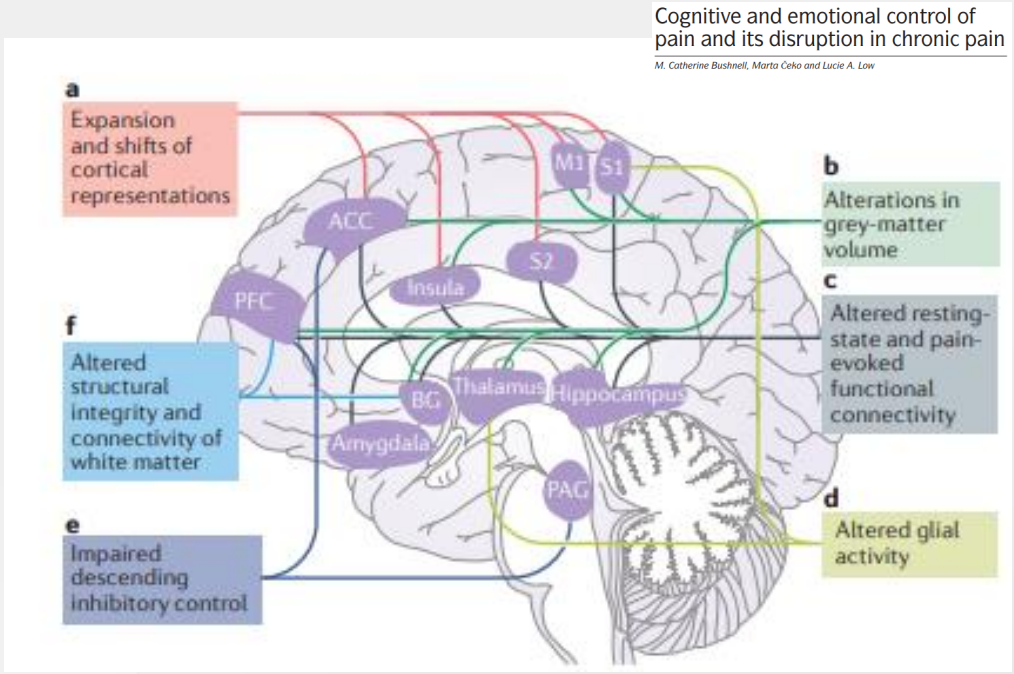
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

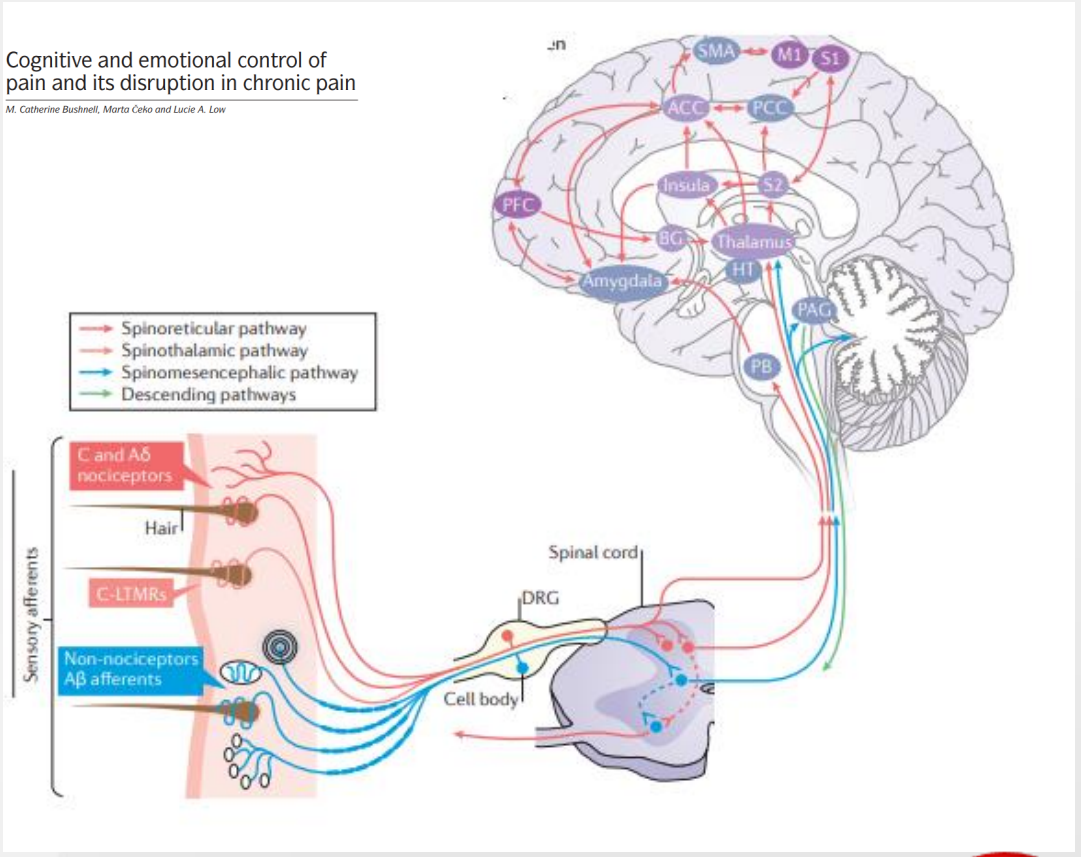
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

CERTAIN ANSWER!

Cyclic processing and synthesis of nerve impulses impose a standard feature of stimuli or "neural signature" (neurotags). Three principles relate to the intensity of these signatures and brain plasticity. They are: (1) neuronal mass, relating to the number of brain cells that are part of a given neurotag and their synaptic efficacy among them; (2) neural precision, that is, how much non-member brain cells can be inhibited; and (3) neuroplasticity, is the property of the central nervous system to undergo structural and functional changes in responses to activities.

Structural plasticity and reorganisation in chronic pain

Rohini Kuner[1,3] and Herta Flor[2,3]

NATURE REVIEWS | NEUROSCIENCE VOLUME 18 | JANUARY 2017 | 21

EXPERT REVIEW

The dynamics of the stress neuromatrix

N Sousa[1,2,3]

Molecular Psychiatry (2016) 21, 302–312

© 2016 Macmillan Publishers Limited All rights reserved 1359-4184/16

www.nature.com/mp

International Journal of Health Sciences
September 2014, Vol. 2, No. 3, pp. 33-45

Therapeutic Neuroscience Education, Pain, Physiotherapy and the Pain Neuromatrix

Adriaan Louw[1] & Emilio J Puentedura[2]

Cognitive and emotional control of pain and its disruption in chronic pain

M. Catherine Bushnell, Marta Čeko and Lucie A. Low

return

Menu

25 - What is the neuromatrix of pain?

A. A specific area of the brain that is activated during nociceptive stimulation;

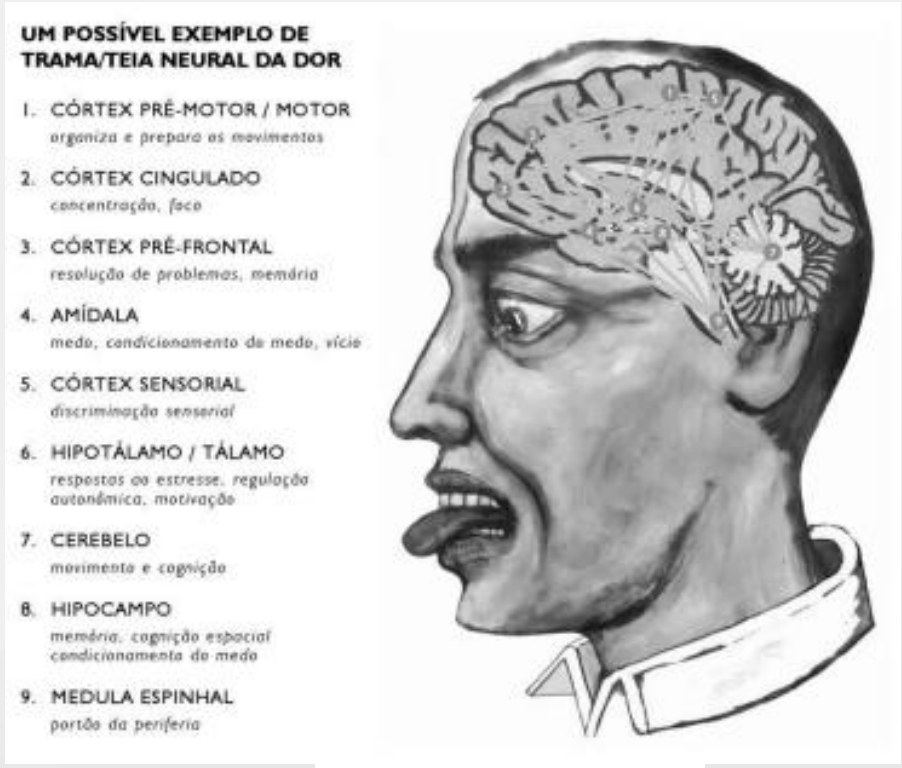
B. The somatosensory cortex region 1 and 2 responsible for pain processing;

C. The associated regions of the neocortex responsible for pain processing;

D. The set of different regions of the brain that are activated during nociceptive stimulation.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Explicando a Dor
David S. Butler e G. Lorimer Moseley
Tradução: Tanja Samira Jorgic
Noigroup Publications, Adelaide, Australia, 2009
19 North Street, Adelaide City West, South Australia 5000

return

Menu

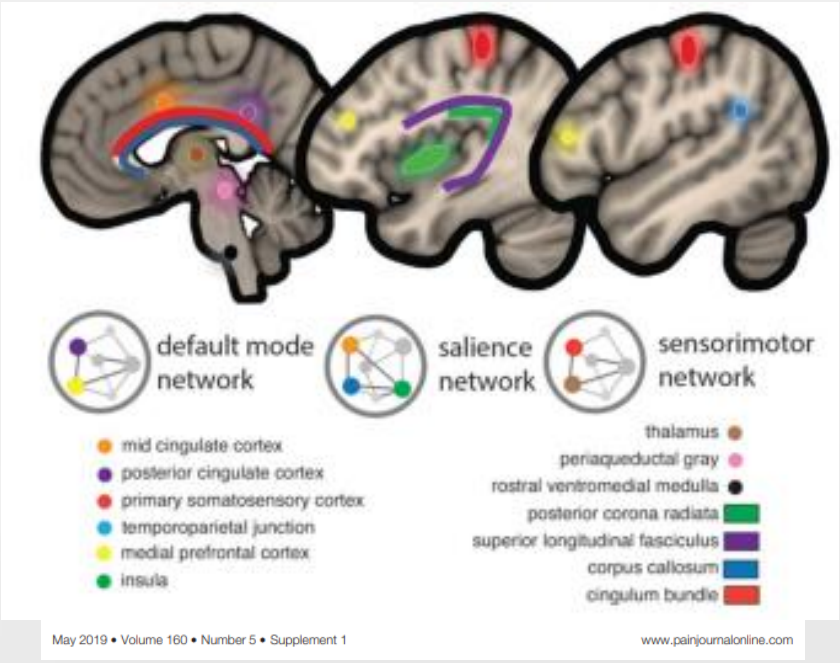
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Explicando a Dor
David S. Butler e G. Lorimer Moseley
Tradução: Tanja Samira Jorgic
Noigroup Publications, Adelaide, Australia, 2009
19 North Street, Adelaide City West, South Australia 5000

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

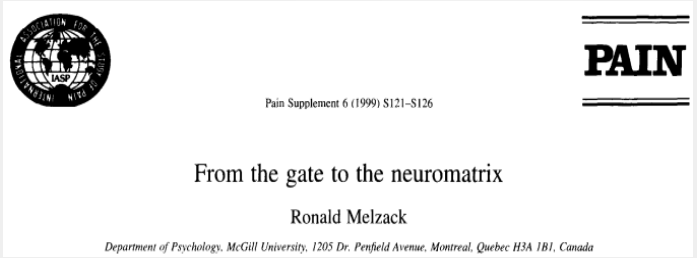


return

Menu

CERTAIN ANSWER!

The neuromatrix of pain is the set of different regions of the brain that are activated during nociceptive stimulation. The descriptions of neuromatrix studies relate to nine cortical and subcortical areas. These different areas interact with your demands and your different tasks. The stimuli diverge to allow parallel processes in different components of the neuromatrix and converge to allow interactions between output processing products. Cyclic processing and synthesis of nerve impulses impose a standard feature of stimuli or "neural signature" (neurotags).


PAIN

Pain Supplement 6 (1999) S121–S126

From the gate to the neuromatrix

Ronald Melzack

Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, Montreal, Quebec H3A 1B1, Canada


The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

An fMRI-Based Neurologic Signature of Physical Pain

Tor D. Wager, Ph.D., Lauren Y. Atlas, Ph.D., Martin A. Lindquist, Ph.D., Mathieu Roy, Ph.D., Choong-Wan Woo, M.A., and Ethan Kross, Ph.D.


Explicando a Dor

David S. Butler e G. Lorimer Moseley

Tradução: Tanja Samira Jorgic

Noigroup Publications, Adelaide, Australia, 2009

19 North Street, Adelaide City West, South Australia 5000

return

Menu

26 - A pain neurotag is described as: "a functional network of neurons in different areas of the brain involved in pain processing." They share information from:

A. nociceptive, motor, emotional, sympathetic and parasympathetic autonomic nervous system stimuli;

B. sensory stimuli of threat and protection from pain;

C. motor stimuli of pain protection and surveillance;

D. central nociceptive stimuli and peripheral nervous system stimuli.

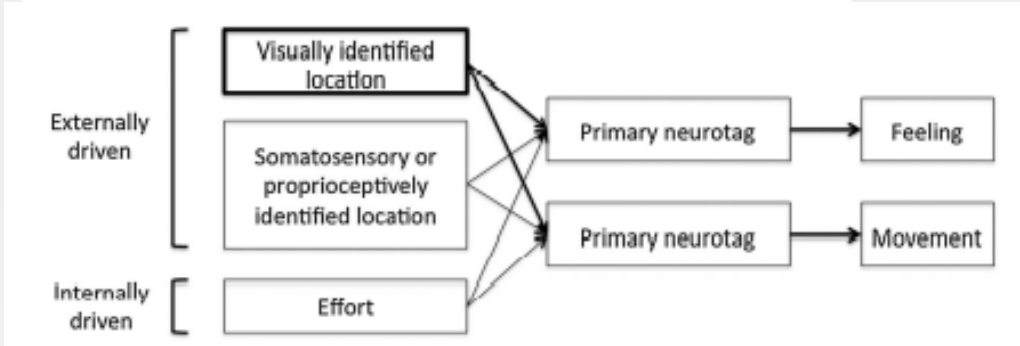
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Neural representations and the cortical body matrix: implications for sports medicine and future directions

Sarah B Wallwork,[1] Valeria Bellan,[1] Mark J Catley,[1] G Lorimer Moseley[1,2]

Wallwork SB, *et al. Br J Sports Med* 2016;**50**:990–996. doi:10.1136/bjsports-2015-095356



return

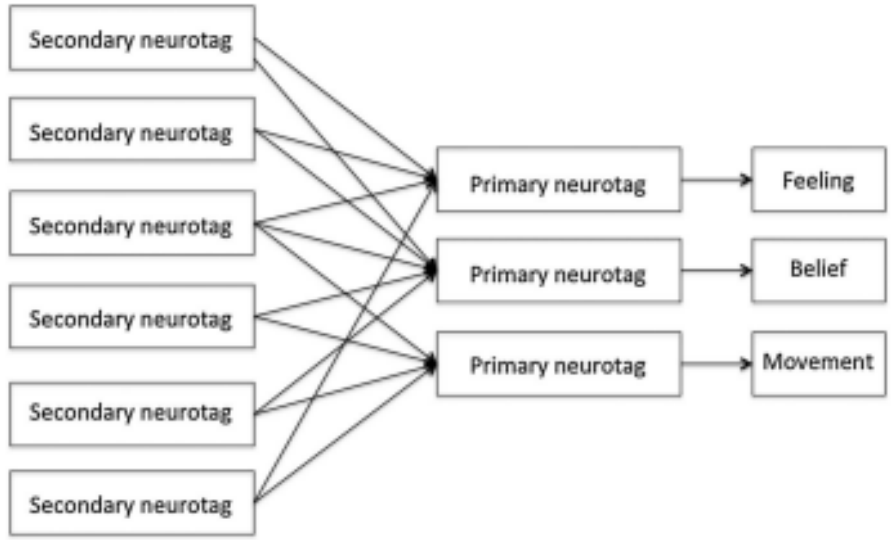
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Neural representations and the cortical body matrix: implications for sports medicine and future directions

Sarah B Wallwork,[1] Valeria Bellan,[1] Mark J Catley,[1] G Lorimer Moseley[1,2]

Wallwork SB, *et al. Br J Sports Med* 2016;**50**:990–996. doi:10.1136/bjsports-2015-095356



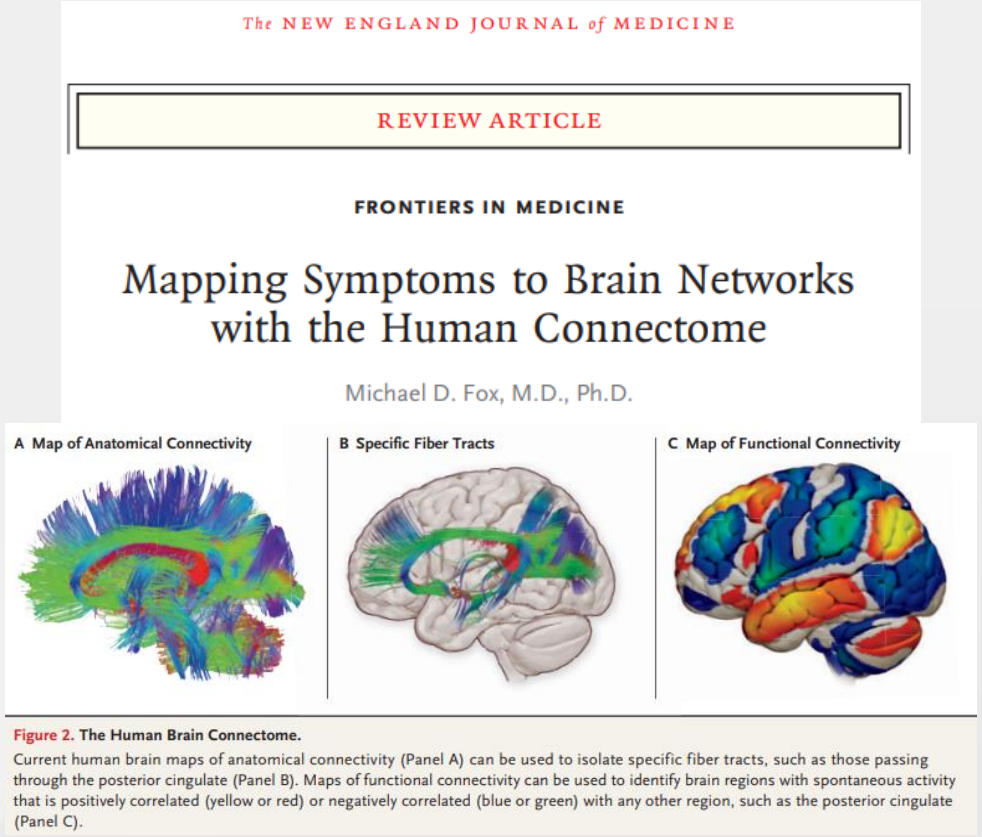
return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

The NeuroTag of Pain, are regions of the brain that at the same time can work in a joint or isolated way. Therefore, pain is the result of several entries or inputs on the nervous system.

QUESTIONADOR PODCAST



return

Menu

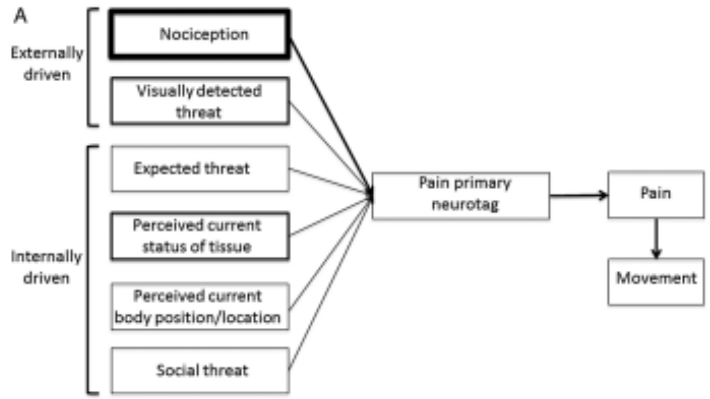
CERTAIN ANSWER!

The intensity of the pain can be dissociated from the magnitude of the response in the pain matrix. Nociceptive stimuli can elicit cortical responses with a spatial configuration similar to that of the neuromatrix. Thus, a pain neurotag is described as: "a functional network of neurons in different areas of the brain involved in pain processing." They are regions of the brain that at the same time share information of nociceptive, motor, emotional stimuli, of the sympathetic and parasympathetic autonomic nervous system, being able to work together or in isolation. That is, the pain is a result of the activation of several inputs on the CNS.

Neural representations and the cortical body matrix: implications for sports medicine and future directions

Sarah B Wallwork,[1] Valeria Bellan,[1] Mark J Catley,[1] G Lorimer Moseley[1,2]

Wallwork SB, *et al. Br J Sports Med* 2016;**50**:990–996. doi:10.1136/bjsports-2015-095356



return

Menu

27 - They are phenomena of a plasticity that facilitates information transmitted from the periphery to the higher centers:

A — Altered sensitization and blurring;

B — Hyperalgesia and allodynia;

C — Hyperalgesia and altered sensitization;

D — Diffuse sensitization and hypersensitivity.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

New concepts of pain

Anne-Priscille Trouvin [a, b], Serge Perrot [a, b, *]

[a] Unité INSERM U987, Hôpital Ambroise Paré, Paris Descartes University, 9 avenue Charl
Boulogne Billancourt, France
[b] Centre d'Evaluation et Traitement de la Douleur, Hôpital Cochin, Paris Descartes Univ
Faubourg Saint Jacques, 75014, Paris, France

Pain Ther (2020) 9:S1–S15
https://doi.org/10.1007/s40122-020-00217-w

PRACTICAL APPROACH

Not All Pain is Created Equal: Basic Definitions and Diagnostic Work-Up

Cesare Bonezzi · Diego Fornasari · Claudio Cricelli · Alberto Magni · Giuseppe Ventriglia

The Discriminative Validity of "Nociceptive," "Peripheral Neuropathic," and "Central Sensitization" as Mechanisms-based Classifications of Musculoskeletal Pain

Keith M. Smart, PhD, Catherine Blake, PhD,† Anthony Staines, PhD,‡*

Quantitative Sensory Testing in Chronic Musculoskeletal Pain

Zakir Uddin, BScPT, MSc, PhD*,† and Joy C. MacDermid, BSc, BScPT, MSc, PhD*,‡

Pain Medicine 2016
doi: 10.1093/pm/p

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Quantitative Sensory Testing in Chronic Musculoskeletal Pain

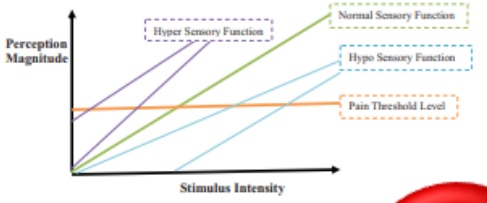
Zakir Uddin, BScPT, MSc, PhD*,† and
Joy C. MacDermid, BSc, BScPT, MSc, PhD*,‡

Pain Medicine 2016; 0: 1–10
doi: 10.1093/pm/pnv105

QST Modality	QST Parameter	Method
Current	Current perception threshold	Method of limits
Vibration	Vibration threshold	Method of limits
Pointing touch	Touch threshold	Method of limits
Light touch	Touch threshold	Method of levels
Blunt pressure	Pressure pain threshold and tolerance	Method of levels

Basal Pain Sensitivity	Hypersensitivity	Hyperesthesia (Hyperalgesia, Allodynia)
	Normal sensitivity	
	Hyposensitivity	Hypoesthesia (Numbness, Paresthesia)

Figure 2 Basal pain sensitivity and abnormal pain response detection.



return

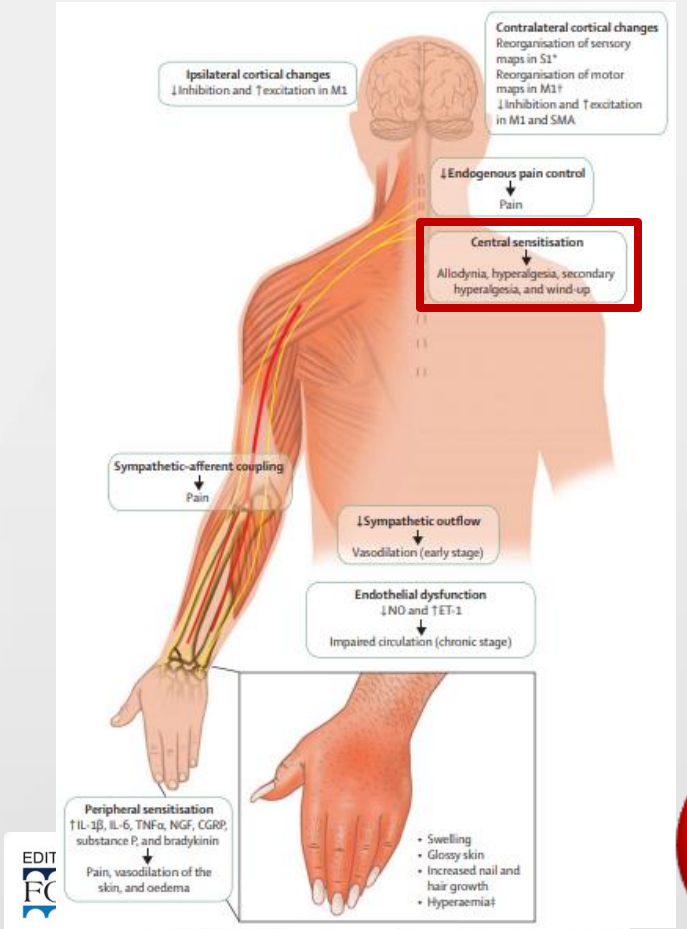
Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Clinical features and pathophysiology of complex regional pain syndrome

www.thelancet.com/neurology Vol 10 July 2011

Johan Marinus, G Lorimer Moseley, Frank Birklein, Ralf Baron, Christian Maihöfner, Wade S Kingery, Jacobus J van Hilten

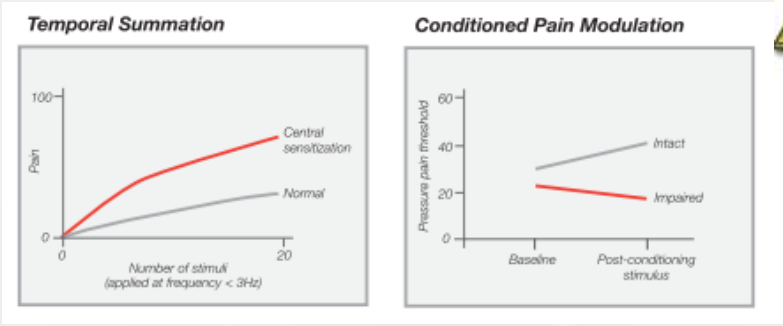


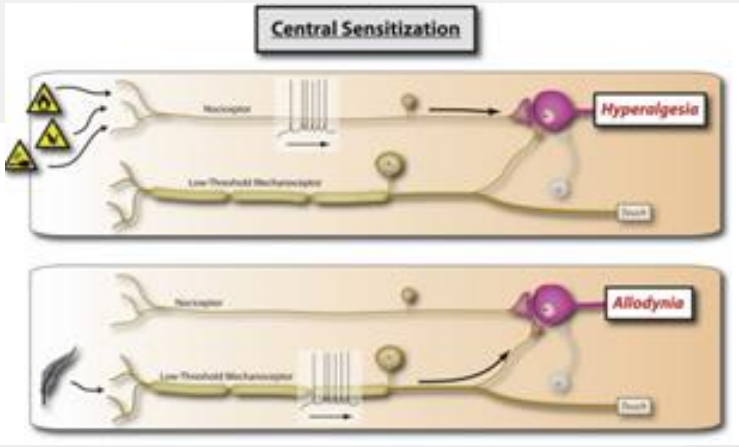
return

Menu

CERTAIN ANSWER!

The facilitation of the signal so that the information retransmitted to higher centers is not coupled to the intensity or duration of the peripheral stimulus, which may result in neurophysiological phenomena of hyperalgesia (given that the stimulus is painful, it is perceived as more painful) and allodynia (perceiving a symptom as painful, when in fact it is not). These two phenomena will be presented in any type of sensitization: which is an amplification of a neuronal response, of a sensory pathway (in this case the nociceptive pathway), once it comes into contact with the stimulus.





IASP
PAIN® 152 (2011) S2–S15
PAIN®
www.elsevier.com/locate/pain

Review

Central sensitization: Implications for the diagnosis and treatment of pain

Clifford J. Woolf

Program in Neurobiology and FM Kirby Neurobiology Center, Children's Hospital Boston, Department of Neurobiology, Harvard Medical School, Boston, MA, USA

New concepts of pain

Anne-Priscille Trouvin [a, b], Serge Perrot [a, b, *]

[a] *Unité INSERM U987, Hôpital Ambroise Paré, Paris Descartes University, 9 avenue Charles de Gaulle, 92100, Boulogne Billancourt, France*
[b] *Centre d'Evaluation et Traitement de la Douleur, Hôpital Cochin, Paris Descartes University, 27 rue du Faubourg Saint Jacques, 75014, Paris, France*

JOURNAL OF MANUAL & MANIPULATIVE THERAPY, 2017
VOL. 25, NO. 3, 118–127
https://doi.org/10.1080/10669817.2017.1300397
Taylor & Francis
Taylor & Francis Group
Check for updates

Mechanisms of chronic pain – key considerations for appropriate physical therapy management

Carol A. Courtney[a], César Fernández-de-las-Peñas[b,c] and Samantha Bond[a]

Quantitative Sensory Testing in Chronic Musculoskeletal Pain

Zakir Uddin, BScPT, MSc, PhD*,† and
Joy C. MacDermid, BSc, BScPT, MSc, PhD*,‡

Pain Medicine 2016; 0: 1–10
doi: 10.1093/pm/pnv105

return

Menu

28 - We can evaluate signs of deficiency of the somatosensory system from low-cost materials, through simple stimuli such as:

A. Light touch (cotton ball or gauze), chopped (paper clip or pin) and temperature (water tube at 40 degrees C);

B. Light touch (cotton ball or gauze), tendon reflex (buck's neurological hammer) and stethoscope;

C. Tendon reflex (Taylor neurological hammer), tuning fork and maddox lens;

D. Otoscope, clinical flashlight and sphygmomanometer.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Journal of Psychosomatic Research 117 (2019) 32–40

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Review article

Central sensitization in chronic pain and medically unexplained symptom research: A systematic review of definitions, operationalizations and measurement instruments

Carine den Boer[a,*], Linne Dries[a], Berend Terluin[a], Johannes C. van der Wouden[a], Annette H. Blankenstein[a], C. Paul van Wilgen[b,d], Peter Lucassen[c], Henriëtte E. van der Horst[a]

Table 6
Categories of measurement instruments and examples of specific tests.

Measurement instrument	What is measured?	Examples
Quantitative sensory testing (QST)	Hyperalgesia, allodynia, temporal summation	Thermal stimuli: thresholds for cold pain, heat pain, cold detection and heat detection; e.g., putting the hand in an iced water bath [41] Tactile stimuli: pressure pain thresholds (PPTs) [109,116] Vibratory or vibrotactile stimuli: detection thresholds for vibration or combination of tactile and vibratory stimuli, e.g., electric toothbrush [120] Electrical stimuli: reaction to electrical pulses with electrodes Distention: distending the rectum or oesophagus with an inflatable balloon [66] Ischemic stimuli: ischemic compression of the arm with a cuff [110] Reaction on specific pain mediators, e.g. reaction on injection with hypertonic saline [67]
Two different quantitative sensory tests together	Conditioned pain modulation (CPM)	Tonic phasic stimulation: phasic heat test with counterirritation of cold [60,130] Ischemic stimulation: inflating an occlusion cuff, comparing pressure pain prior to and during cuff inflation [110] The nociception withdrawal reflex e.g. H(offman) reflex: stimulation of median nerve with an EMG device, measurement of H wave (a compound muscle action potential) [44] Measurement of the cutaneous silent period (CSP): a brief pause in muscle action potentials following strong stimulation of a cutaneous nerve during a sustained voluntary contraction [45]
MRI, fMRI, PET, somatosensory evoked potentials (SEP)	Structural and functional brain changes	Measurement of changes in brain morphology (global and regional gray matter volumes), changes in density and changes in signaling [98]
Measurement of cytokine levels	Laboratory evaluation	Measurement of serum levels of pro-inflammatory interleukines (Il-1, IL-6, IL-8) and anti-inflammatory interleukines (IL-4, IL-10); serum levels of TNF-alpha, a pro-inflammatory cytokine [87,140]
Measurement of neurotrophine levels	Laboratory evaluation	Measurement of serum levels of nerve growth factor (NGF) and brain derived neurotrophic factor (BDNF) [26]
Questionnaires	Symptoms, history of functional syndromes	Central sensitization Inventory (CSI) [27,49–54]
	Sensory aspects of hypersensitivity	Sensory Hypersensitivity Scale (SHS) [80]

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

[VIEWPOINT]

HUBERT VAN GRIENSVEN, MSc (Pain), PhD, MCSP, DipAc[1] • ANNINA SCHMID, M Manip Ther, PhD, MMACP, MCSP[2]
TEODORA TRENDAFILOVA, BSc (Hons)[2] • MATTHEW LOW, BSc (Hons), MSc, MMACP, MCSP[3,4]

Central Sensitization in Musculoskeletal Pain: Lost in Translation?

Clinical Sign	Semi-objective Test (QST)	Clincial Test	Interpretation	Measurement Properties of Clinical Tests Compared to Semi-objective Tests
Cold/heat hyperalgesia	Thermal tester	Cold/hot test tubes	Presence of secondary or tertiary hyperalgesia raises suspicion of contribution of central mechanisms	Agreement rates (Zhu et al): Cold tube: 45.0%-69.7% Hot tube: 45.0%-69.2% Cold tube: high test-retest reliability (Cathcart and Pritchard, Tilley and Bisset)
Mechanical hyperalgesia	Weighted pinprick stimulators, blunt pressure algometer	Toothpick/neurotip, blunt pressure (thumb, eraser)	Presence of secondary or tertiary hyperalgesia raises suspicion of contribution of central mechanisms	Agreement rates (Zhu et al): Toothpick: 52.6% -84.6% Thumb pressure: 57.5%-86.8% Eraser pressure: 60.0%-84.2% Nerve palpation: high test-retest reliability (Pedersini et al, Fingleton et al)
Dynamic mechanical allodynia	Cotton wool tip, soft brush	Cotton wool tip, soft brush	Pain elicited on light touch raises suspicion of contribution of central mechanisms	Good to high intertester reliability of allodynia tests (Geber et al)
Temporal summation	Repeated nociceptive stimulation (eg, pinprick, thermal, electrical)	Repeated pinprick stimulation with toothpick/neurotip	Exacerbation of pain ratings for a train of stimuli compared to a single stimulus raises suspicion of contribution of central mechanisms. Also observe for painful aftersensations (prolonged pain after repeated stimuli have stopped)	Agreement rates (Zhu et al): Toothpick: 47.5%-76.5% Wind-up with pinprick moderate test-retest reliability (Geber et al)
Spatial summation	Different sizes of thermodes, different numbers of pressure probes		Presence raises suspicion of contribution of central mechanisms	
Descending inhibition	Conditioned pain modulation (conditioning and test stimulus over different areas, for example, one foot immersed in ice water and pressure pain threshold over the contralateral foot)		Presence raises suspicion of contribution of central mechanisms	Test-retest reli… lent (Kenn…

FOA UniFOA Centro Universitário de Volta Redonda
Mestrado Profissional em Ensino em Ciências da Saúde e do Meio Ambiente UniFOA

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

PAIN

NeuP SIG

Imaging vs quantitative sensory testing to predict chronic pain treatment outcomes

Karen D. Davis[a,b]

ELSEVIER

Available online at www.sciencedirect.com

SCIENCE DIRECT®

European Journal of Pain 10 (2006) 77–88

www.EuropeanJournalPain.com

Quantitative sensory testing: a comprehensive protocol for clinical trials

R. Rolke [a,b], W. Magerl [a], K. Andrews Campbell [c], C. Schalber [a], S. Caspari [a], F. Birklein [b], R.-D. Treede [a,*]

Quantitative sensory testing (QST)

Schmerz
DOI 10.1007/s00482-015-0093-2

M. Mücke[1,2] · H. Cuhls[2] · L. Radbruch[2] · R. Baron[3] · C. Maier[4] · T. Tölle[5] · R.-D. Treede[6] · R. Rolke[7]

return

EDITORA FOA

UniFOA Centro Universitário de Volta Redonda

Mestrado Profissional em Ensino em Ciências da Saúde e do Meio Ambiente UniFOA

Menu

CERTAIN ANSWER!

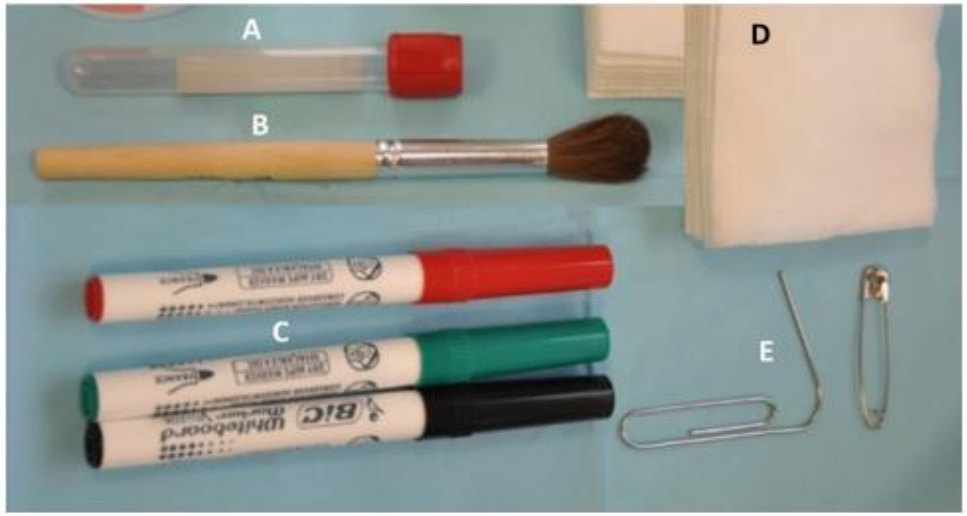
Three stimuli are used to test the sensitivity to light touch discrimination with a cotton ball or gauze, chopped with the use of a paper clip and/or pin and temperature with a water tube at 40 ° C. From this evaluation, we can determine whether or not the patient has sensory deficits, making it possible to evaluate the response of the nervous system to harmful and harmless somatic exogenous stimuli, and eventually pain. Physical therapists should be mindful of central sensitization and consider top-down and bottow-up potentials in the context of a person-centered biopsychosocial approach.

Pain Ther (2020) 9:S1–S15
https://doi.org/10.1007/s40122-020-00217-w

PRACTICAL APPROACH

Not All Pain is Created Equal: Basic Definitions and Diagnostic Work-Up

Cesare Bonezzi · Diego Fornasari · Claudio Cricelli · Alberto Magni · Giuseppe Ventriglia



[VIEWPOINT]

HUBERT VAN GRIENSVEN, MSc (Pain), PhD, MCSP, DipAc[1] • ANNINA SCHMID, M Manip Ther, PhD, MMACP, MCSP[2]
TEODORA TRENDAFILOVA, BSc (Hons)[2] • MATTHEW LOW, BSc (Hons), MSc, MMACP, MCSP[3,4]

Central Sensitization in Musculoskeletal Pain: Lost in Translation?

return

Menu

29 - We can quantitatively assess the pain threshold that may be associated with central and/or peripheral sensitization processes in the clinical setting through which device?

A. Sphygmomanometer;

B. Pressure algometer;

C. Baropodometer;

D. Dynamometer.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

[RESEARCH REPORT]

DAVID WALTON, PT, PhD[1] • JOY MACDERMID, PT, PhD[2] • WARREN NIELSON, PhD[3]
ROBERT TEASELL, MD[4] • MARCO CHIASSON[5] • LAUREN BROWN[5]

Reliability, Standard Error, and Minimum Detectable Change of Clinical Pressure Pain Threshold Testing in People With and Without Acute Neck Pain

FIGURE 1. Locations for testing of the upper fibers of the trapezius (A) and tibialis anterior (B) s

return

Menu

WRONG ANSWER! CLICK ON THE PODCAST, INTERACT AND TRY AGAIN!

Quantitative Sensory Testing (QST) indices of pain hypersensitivity can help develop targeted interventions aimed at improving outcomes in a variety of musculoskeletal conditions offered by the physiotherapist.

QUESTIONADOR PODCAST

Quantitative sensory testing (QST)

M. Mücke[1,2] · H. Cuhls[2] · L. Radbruch[2] · R. Baron[3] · C. Maier[4] · T. Tölle[5] · R.-D. Treede[6] · R. Rolke[7]

Schmerz
DOI 10.1007/s00482-015-0093-2

Table 1 Clinical signs, quantitative sensory testing, and possible underlying neurobiological mechanisms

Clinical signs	Definition	Quantitative sensory testing	Possible underlying neurobiological mechanisms		
		Testing for presence of plus or minus signs (tested peripheral fiber types)	Deafferentation	Peripheral sensitization	Central sensitization
Plus signs			Sensitivity to test stimuli		
Hyperalgesia	Increased pain sensitivity[a] of				
To heat	… the skin	Heat stimulation by means of thermotesting (C, Aδ)	↓	↑↑	→?
To cold	… the skin	Cold stimulation by means of thermotesting (C, Aδ)	↓	→	↑?
For pinprick stimuli	… the skin	Calibrated needle stimuli (pinprick) (C, Aδ)	↓	↑?	↑↑
For blunt pressure	… deeper tissues	Pressure algometer (C, Aδ)	↓	↑?	→?
Allodynia[b]	Pain in response to non-nociceptive stimuli[a]	Brush, cotton swab, Q-tip (Aβ) to skin brushing	→	→	↑
Minus signs					
Hypoesthesia (thermal/ mechanical/other)	Decreased sensitivity for nonpainful stimuli	Light cold stimulation by means of thermotesting (Aδ), light heat stimulation by means of thermotesting (C), von Frey filaments (Aβ), calibrated tuning fork (64 Hz, Rydel–Seiffer) (Aβ)	↓	→	→, ↓[c]
Hypoalgesia (thermal/ mechanical/other)	Decreased sensitivity for painful stimuli	To cold/heat stimulus by means of thermotesting (C, Aδ)Calibrated needle stimuli (pinprick) (C, Aδ)Pressure algometer (C, Aδ)	↓	→	→

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Systematic Review and Meta-Analysis

PAIN

Quantitative sensory testing and predicting outcomes for musculoskeletal pain, disability, and negative affect: a systematic review and meta-analysis

Vasileios Georgopoulos[a,b,*], Kehinde Akin-Akinyosoye[a,b], Weiya Zhang[a,b,c], Daniel F. McWilliams[a,b,c], Paul Hendrick[b,c,d], David A. Walsh[a,b,c]

Contents lists available at ScienceDirect

European Journal of Pain

journal homepage: www.EuropeanJournalPain.com

ELSEVIER

Reference values of mechanical and thermal pain tests in a pain-free population

Alban Y. Neziri[a,*], Pasquale Scaramozzino[b], Ole K. Andersen[c], Anthony H. Dickenson[d], Lars Arendt-Nielsen[c], Michele Curatolo[a]

Instrumental validity and intra/inter-rater reliability of a novel low-cost digital pressure algometer

Daniel Jerez-Mayorga[1], Carolina Fernanda dos Anjos[2], Maria de Cássia Macedo[2], Ilha Gonçalves Fernandes[2], Esteban Aedo-Muñoz[3], Leonardo Intelangelo[4] and Alexandre Carvalho Barbosa[2]

PeerJ

return

Menu

CERTAIN ANSWER!

We can evaluate the pressure pain threshold with the use of a pressure algometer that provides us with information in Kg/cm2. Pressure algometry can be used in several body segments (muscle belly, tendon and bone surfaces). The device validates and enables quantitative measurements of pain thresholds in the clinical routine, benefiting the evaluation mainly in primary care. The current proof is that a low-cost pressure algometer is valid and reliable enough to be considered standard equipment for assessing the pressure pain threshold. Quantitative sensory testing indices of pain hypersensitivity can help develop targeted interventions aimed at improving outcomes in a variety of musculoskeletal conditions.

Figure 1 **Adapted pressure algometer—PA.** (1) Display; (2) On-Off button; (3) Tare button; (4) Unit selection button; (5) Adapted terminal. Full-size DOI: 10.7717/peerj.10162/fig-1

Systematic Review and Meta-Analysis

PAIN

Quantitative sensory testing and predicting outcomes for musculoskeletal pain, disability, and negative affect: a systematic review and meta-analysis

Vasileios Georgopoulos[a,b,*], Kehinde Akin-Akinyosoye[a,b], Weiya Zhang[a,b,c], Daniel F. McWilliams[a,b,c], Paul Hendrick[b,c,d], David A. Walsh[a,b,c]

ELSEVIER

Contents lists available at ScienceDirect

European Journal of Pain

journal homepage: www.EuropeanJournalPain.com

Reference values of mechanical and thermal pain tests in a pain-free population

Alban Y. Neziri[a,*], Pasquale Scaramozzino[b], Ole K. Andersen[c], Anthony H. Dickenson[d], Lars Arendt-Nielsen[c], Michele Curatolo[a]

Instrumental validity and intra/inter-rater reliability of a novel low-cost digital pressure algometer

Daniel Jerez-Mayorga[1], Carolina Fernanda dos Anjos[2], Maria de Cássia Macedo[2], Ilha Gonçalves Fernandes[2], Esteban Aedo-Muñoz[3], Leonardo Intelangelo[4] and Alexandre Carvalho Barbosa[2]

PeerJ

retornar

Menu

30 - Endogenous analgesia and the functioning of the anti-nociceptive system can be investigated in a clinical setting through the testing of:

A. Dysdiadochokinesis test;

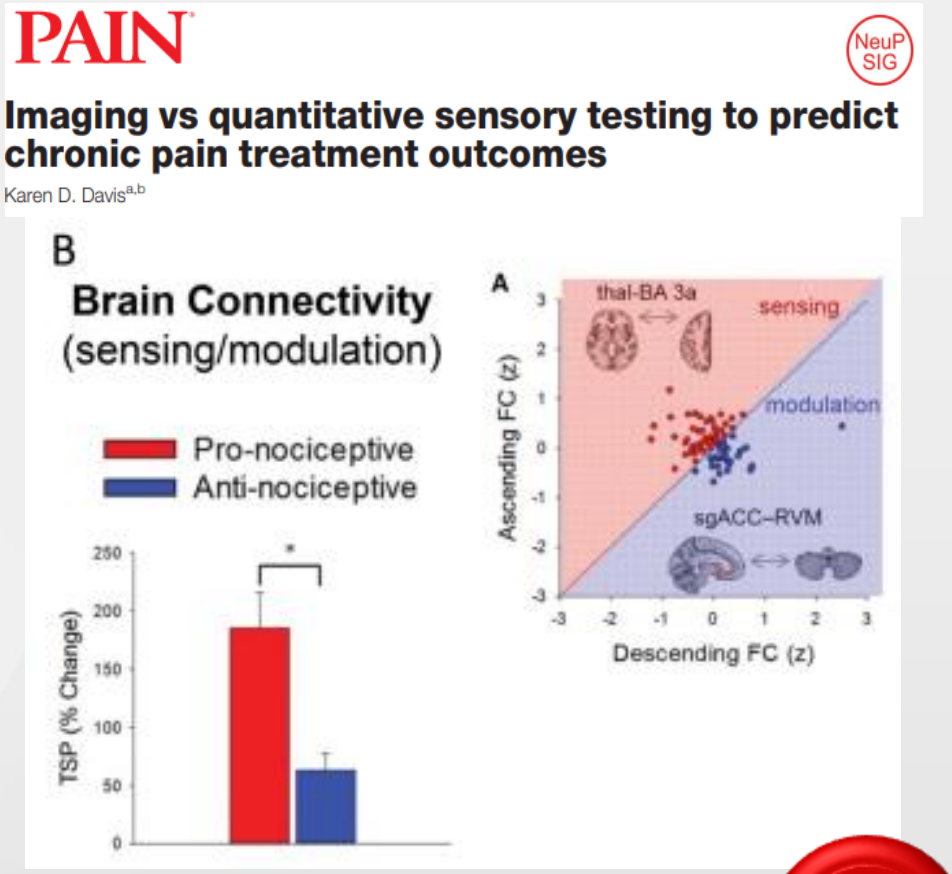
B. Epicritical pain sensitivity test;

C. CPM (conditioned pain modulation) test;

D. Test for palesthesia or vibratory sensitivity.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Personalized Pain Medicine: The Clinical Value of Psychophysical Assessment of Pain Modulation Profile

Rambam Maimonides Medical Journal 4 October 2013 • Volume 4 • Issue 4 • e0024

Yelena Granovsky, Ph.D.* and David Yarnitsky, M.D.

Special Issue on Pain

Guest Editors: Elon Eisenberg and Simon Vulfsons

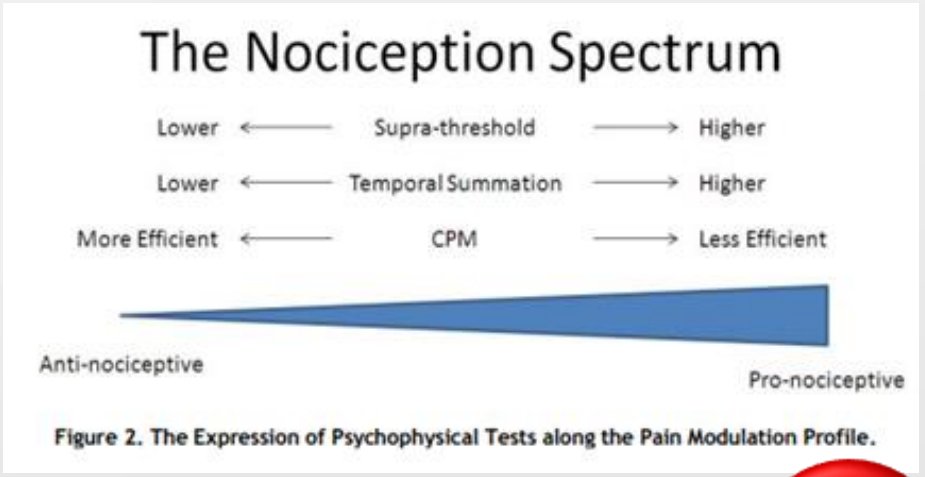


Figure 2. The Expression of Psychophysical Tests along the Pain Modulation Profile.

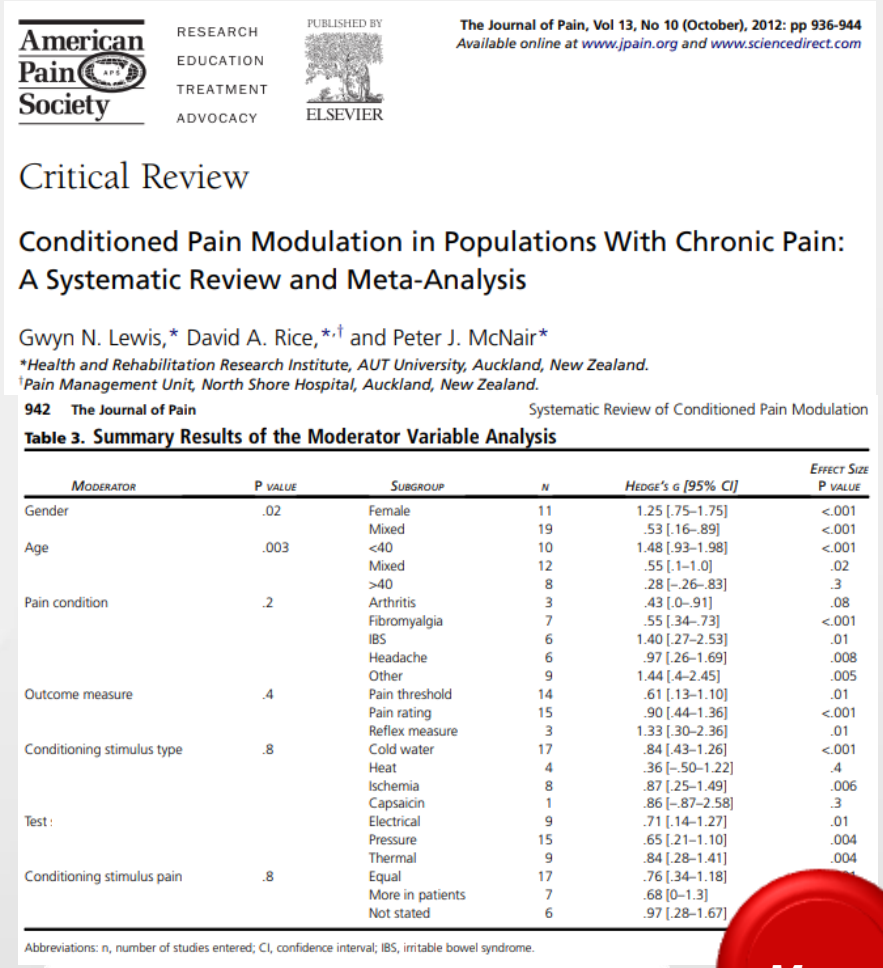
return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Through the CPM test (Conditioned Pain Modulation) we evaluated the functioning of the Anti-Nociceptive system. Therefore, the amount of pain experienced with the primary test stimulus may be reduced during the presentation of a second conditioning event. It is presumed, then, that the extent of pain inhibition during testing behavior primarily reflects the effectiveness of the Diffuse Noxious Inhibitory System. However, supraspinal mechanisms may also be involved in this control.

QUESTIONADOR PODCAST



American Pain Society — RESEARCH EDUCATION TREATMENT ADVOCACY — PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 13, No 10 (October), 2012: pp 936-944
Available online at www.jpain.org and www.sciencedirect.com

Critical Review

Conditioned Pain Modulation in Populations With Chronic Pain: A Systematic Review and Meta-Analysis

Gwyn N. Lewis,* David A. Rice,*,† and Peter J. McNair*
*Health and Rehabilitation Research Institute, AUT University, Auckland, New Zealand.
†Pain Management Unit, North Shore Hospital, Auckland, New Zealand.

Table 3. Summary Results of the Moderator Variable Analysis

Moderator	P value	Subgroup	n	Hedge's g [95% CI]	Effect Size P value
Gender	.02	Female	11	1.25 [.75–1.75]	<.001
		Mixed	19	.53 [.16–.89]	<.001
Age	.003	<40	10	1.48 [.93–1.98]	<.001
		Mixed	12	.55 [.1–1.0]	.02
		>40	8	.28 [–.26–.83]	.3
Pain condition	.2	Arthritis	3	.43 [.0–.91]	.08
		Fibromyalgia	7	.55 [.34–.73]	<.001
		IBS	6	1.40 [.27–2.53]	.01
		Headache	6	.97 [.26–1.69]	.008
		Other	9	1.44 [.4–2.45]	.005
Outcome measure	.4	Pain threshold	14	.61 [.13–1.10]	.01
		Pain rating	15	.90 [.44–1.36]	<.001
		Reflex measure	3	1.33 [.30–2.36]	.01
Conditioning stimulus type	.8	Cold water	17	.84 [.43–1.26]	<.001
		Heat	4	.36 [–.50–1.22]	.4
		Ischemia	8	.87 [.25–1.49]	.006
		Capsaicin	1	.86 [–.87–2.58]	.3
Test :		Electrical	9	.71 [.14–1.27]	.01
		Pressure	15	.65 [.21–1.10]	.004
		Thermal	9	.84 [.28–1.41]	.004
Conditioning stimulus pain	.8	Equal	17	.76 [.34–1.18]	
		More in patients	7	.68 [0–1.3]	
		Not stated	6	.97 [.28–1.67]	

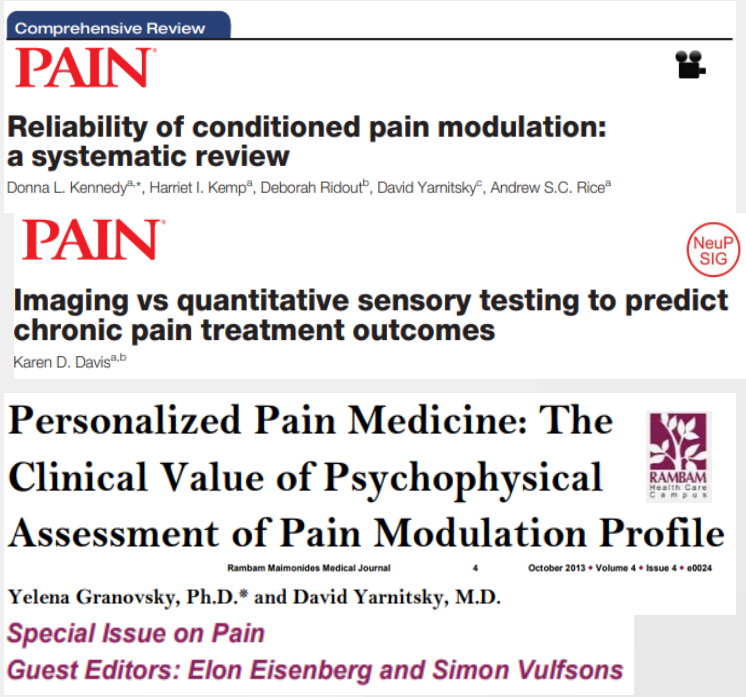
Abbreviations: n, number of studies entered; CI, confidence interval; IBS, irritable bowel syndrome.

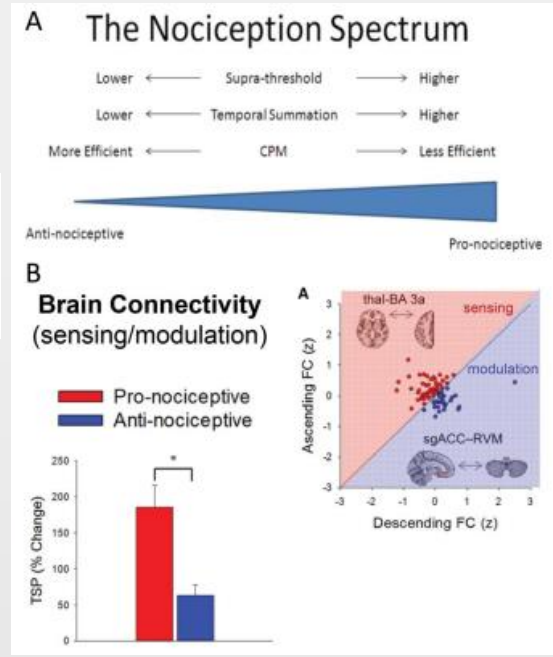
return

Menu

CERTAIN ANSWER!

O teste Conditioned Pain Modulation (CPM) pode ser utilizado na prática clínica paira avaliar o funcionamento anti-nociceptivo. A Modulação Condicionada da Dor (CPM) pode ser usada para predizer a saúde, integridade e a força dos sistemas endógenos da dor (dois paradigmas psicofísicos – espectro pró ou anti-nociceptivo), através de controle inibitório nocivo difuso. Os circuitos endógenos de modulação da dor possuem a capacidade de aumentar ou reduzir a magnitude percebida dos estímulos aferentes, ou seja, uma dor pode inibir a outra (controle inibitório nocivo difuso);





return

Menu

31 - The search to inhibit pain by conditioning stimulus by the passive MPC test, which can be done in what way?

A. Electroneuromyography and dynamometry;

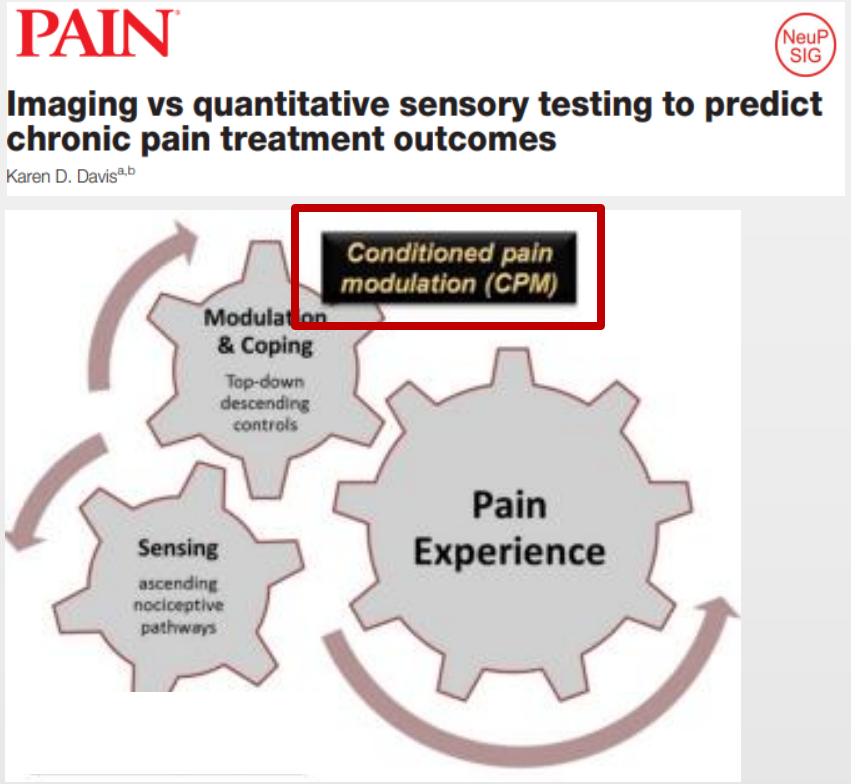
B. Ice bucket and sphygmomanometer;

C. Electromyography and aesthesiometry;

D. Baropodometry and stabilometry.

Menu

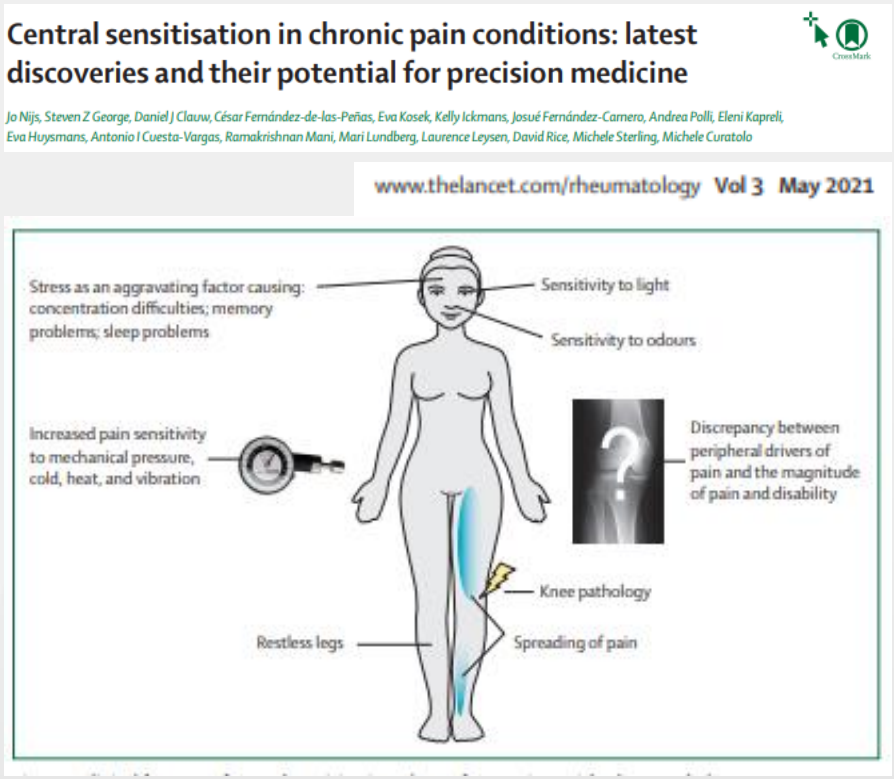
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Central sensitisation in chronic pain conditions: latest discoveries and their potential for precision medicine

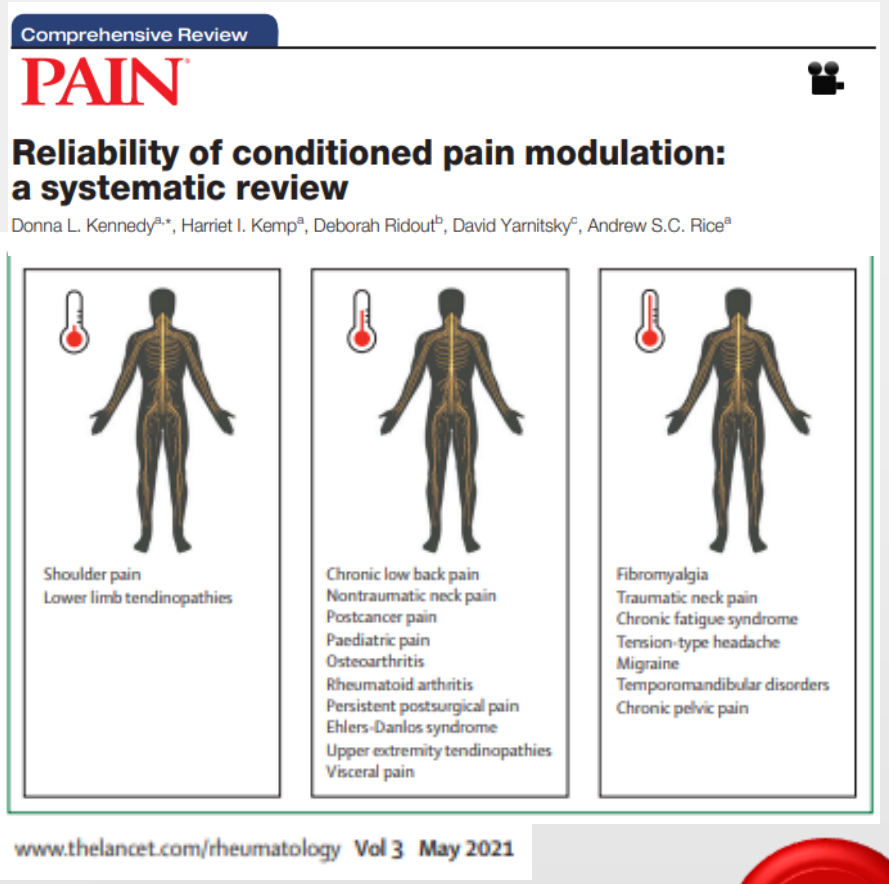
Jo Nijs, Steven Z George, Daniel J Clauw, César Fernández-de-las-Peñas, Eva Kosek, Kelly Ickmans, Josué Fernández-Carnero, Andrea Polli, Eleni Kapreli, Eva Huysmans, Antonio I Cuesta-Vargas, Ramakrishnan Mani, Mari Lundberg, Laurence Leysen, David Rice, Michele Sterling, Michele Curatolo

www.thelancet.com/rheumatology Vol 3 May 2021

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

CERTAIN ANSWER!

Comprehensive Review

PAIN

Reliability of conditioned pain modulation: a systematic review

Donna L. Kennedy[a,*], Harriet I. Kemp[a], Deborah Ridout[b], David Yarnitsky[c], Andrew S.C. Rice[a]

Personalized Pain Medicine: The Clinical Value of Psychophysical Assessment of Pain Modulation Profile

Yelena Granovsky, Ph.D.* and David Yarnitsky, M.D.

Special Issue on Pain
Guest Editors: Elon Eisenberg and Simon Vulfsons

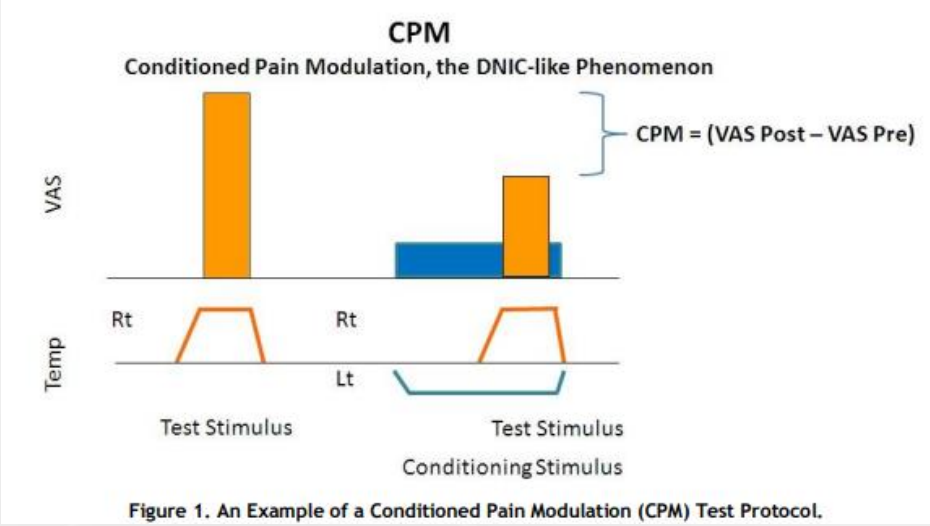


Figure 1. An Example of a Conditioned Pain Modulation (CPM) Test Protocol.

The passive MPS test consists of 3 (three) phases: pre-stimulus: evaluate the pain threshold at pressure 3 different anatomical sites with the pressure algometer. The conditioning stimulus that can be applied by immersing one of the limbs in a bucket with ice or through a sphygmomanometer. The description of cuff load is 20 in 20mmHg until pain perception occurs, and after this first threshold the patient should report a mean VAS of 5 points. From this conditioning stimulus we waited 2 minutes in test. Post-stimulus: we reassessed the pressure pain threshold at the 3 anatomical points still under the effect of the stimulus through the sphygmomanometer. Positive scores with: (post threshold – pre threshold) = are indicative of a preserved anti-nociceptive function. That is, the nociceptor system assists with the endogenous mechanism.

return

Menu

32 - We can quantify in a quantified way a state of increased pronociception in clinical practice, through which phenomenon?

A. Wind-Up (time summation));

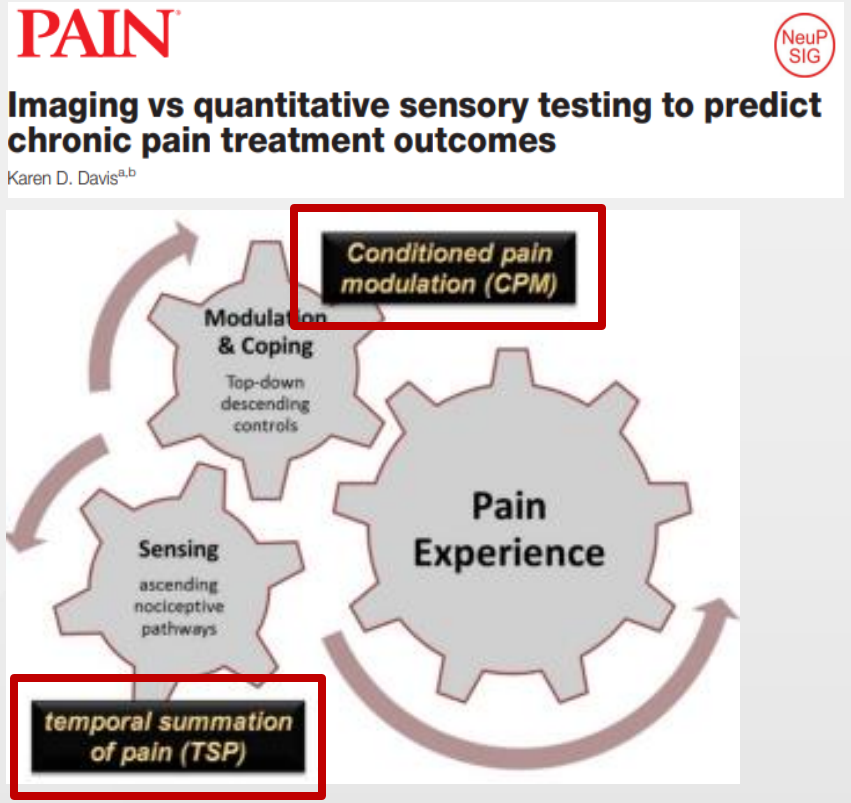
B. Paresthesia;

C. Paresis;

D. Hypoesthesia.

Menu

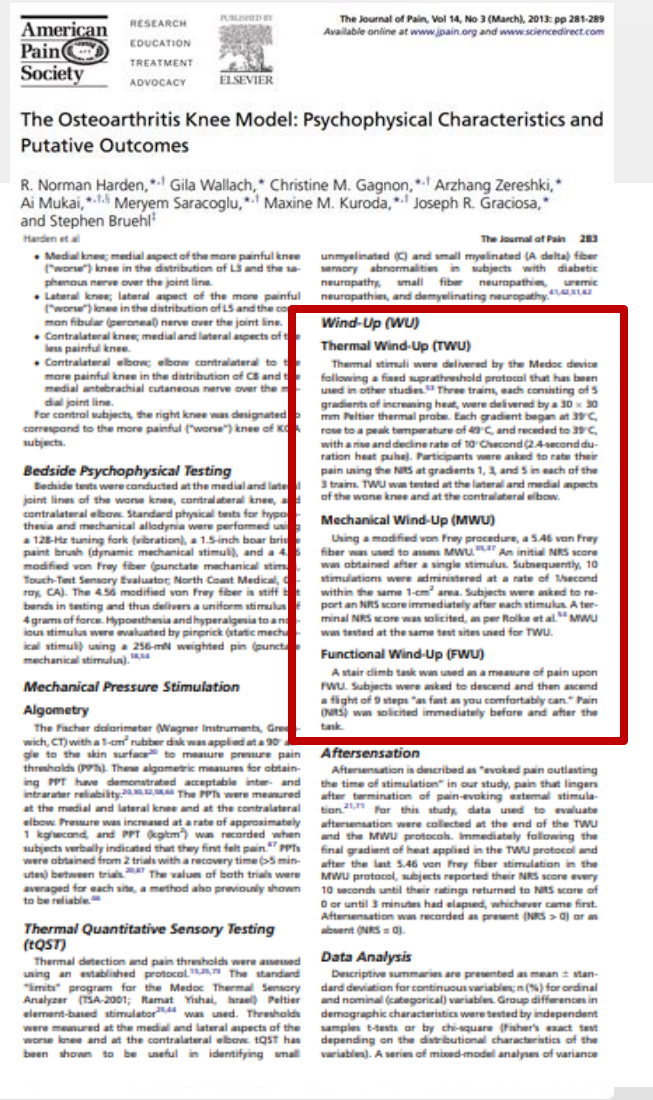
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



American Pain Society — RESEARCH EDUCATION TREATMENT ADVOCACY — PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 14, No 3 (March), 2013: pp 281-289
Available online at www.jpain.org and www.sciencedirect.com

The Osteoarthritis Knee Model: Psychophysical Characteristics and Putative Outcomes

R. Norman Harden,*,† Gila Wallach,* Christine M. Gagnon,*,† Arzhang Zereshki,* Ai Mukai,*,†,‡ Meryem Saracoglu,*,† Maxine M. Kuroda,*,† Joseph R. Graciosa,* and Stephen Bruehl‡

Harden et al — The Journal of Pain 283

- Medial knee; medial aspect of the more painful knee ("worse") knee in the distribution of L3 and the saphenous nerve over the joint line.
- Lateral knee; lateral aspect of the more painful ("worse") knee in the distribution of L5 and the common fibular (peroneal) nerve over the joint line.
- Contralateral knee; medial and lateral aspects of the less painful knee.
- Contralateral elbow; elbow contralateral to the more painful knee in the distribution of C8 and the medial antebrachial cutaneous nerve over the medial joint line.

For control subjects, the right knee was designated to correspond to the more painful ("worse") knee of KOA subjects.

Bedside Psychophysical Testing

Bedside tests were conducted at the medial and lateral joint lines of the worse knee, contralateral knee, and contralateral elbow. Standard physical tests for hypoesthesia and mechanical allodynia were performed using a 128-Hz tuning fork (vibration), a 1.5-inch boar bristle paint brush (dynamic mechanical stimuli), and a 4.56 modified von Frey fiber (punctate mechanical stimuli; Touch-Test Sensory Evaluator; North Coast Medical, Morgan Hill, CA). The 4.56 modified von Frey fiber is stiff but bends in testing and thus delivers a uniform stimulus of 4 grams of force. Hypoesthesia and hyperalgesia to a noxious stimulus were evaluated by pinprick (static mechanical stimuli) using a 256-mN weighted pin (punctate mechanical stimulus).[18,54]

Mechanical Pressure Stimulation

Algometry

The Fischer dolorimeter (Wagner Instruments, Greenwich, CT) with a 1-cm² rubber disk was applied at a 90° angle to the skin surface[20] to measure pressure pain thresholds (PPTs). These algometric measures for obtaining PPT have demonstrated acceptable inter- and intrarater reliability.[20,30,32,58,64] The PPTs were measured at the medial and lateral knee and at the contralateral elbow. Pressure was increased at a rate of approximately 1 kg/second, and PPT (kg/cm²) was recorded when subjects verbally indicated that they first felt pain.[67] PPTs were obtained from 2 trials with a recovery time (>5 minutes) between trials.[20,67] The values of both trials were averaged for each site, a method also previously shown to be reliable.[66]

Thermal Quantitative Sensory Testing (tQST)

Thermal detection and pain thresholds were assessed using an established protocol.[19,20,73] The standard "limits" program for the Medoc Thermal Sensory Analyzer (TSA-2001; Ramat Yishai, Israel) Peltier element-based stimulator[20,44] was used. Thresholds were measured at the medial and lateral aspects of the worse knee and at the contralateral elbow. tQST has been shown to be useful in identifying small unmyelinated (C) and small myelinated (A delta) fiber sensory abnormalities in subjects with diabetic neuropathy, small fiber neuropathies, uremic neuropathies, and demyelinating neuropathy.[41,42,51,62]

Wind-Up (WU)

Thermal Wind-Up (TWU)

Thermal stimuli were delivered by the Medoc device following a fixed suprathreshold protocol that has been used in other studies.[63] Three trains, each consisting of 5 gradients of increasing heat, were delivered by a 30 × 30 mm Peltier thermal probe. Each gradient began at 39°C, rose to a peak temperature of 49°C, and receded to 39°C, with a rise and decline rate of 10°C/second (2.4-second duration heat pulse). Participants were asked to rate their pain using the NRS at gradients 1, 3, and 5 in each of the 3 trains. TWU was tested at the lateral and medial aspects of the worse knee and at the contralateral elbow.

Mechanical Wind-Up (MWU)

Using a modified von Frey procedure, a 5.46 von Frey fiber was used to assess MWU.[65,67] An initial NRS score was obtained after a single stimulus. Subsequently, 10 stimulations were administered at a rate of 1/second within the same 1-cm² area. Subjects were asked to report an NRS score immediately after each stimulus. A terminal NRS score was solicited, as per Rolke et al.[54] MWU was tested at the same test sites used for TWU.

Functional Wind-Up (FWU)

A stair climb task was used as a measure of pain upon FWU. Subjects were asked to descend and then ascend a flight of 9 steps "as fast as you comfortably can." Pain (NRS) was solicited immediately before and after the task.

Aftersensation

Aftersensation is described as "evoked pain outlasting the time of stimulation" in our study, pain that lingers after termination of pain-evoking external stimulation.[21,71] For this study, data used to evaluate aftersensation were collected at the end of the TWU and the MWU protocols. Immediately following the final gradient of heat applied in the TWU protocol and after the last 5.46 von Frey fiber stimulation in the MWU protocol, subjects reported their NRS score every 10 seconds until their ratings returned to NRS score of 0 or until 3 minutes had elapsed, whichever came first. Aftersensation was recorded as present (NRS > 0) or as absent (NRS = 0).

Data Analysis

Descriptive summaries are presented as mean ± standard deviation for continuous variables; n (%) for ordinal and nominal (categorical) variables. Group differences in demographic characteristics were tested by independent samples t-tests or by chi-square (Fisher's exact test depending on the distributional characteristics of the variables). A series of mixed-model analyses of variance

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

European Journal of Pain (2000) **4**: 5–17
doi:10.1053/eujp.1999.0154, available online at http://www.idealibrary.com on IDEAL®

Review Article

Wind-up and the NMDA receptor complex from a clinical perspective

Per K. Eide

Department of Neurosurgery, The National Hospital, University of Oslo, Oslo, Norway

PAIN

NeuP SIG

Imaging vs quantitative sensory testing to predict chronic pain treatment outcomes

Karen D. Davis[a,b]

return

Menu

CERTAIN ANSWER!

The presence of temporal summation (Wind-Up) is one of the symptoms that may indicate a state of increased pronociception. With the use of a low-cost pressure algometer, we can quantify the evaluation. The first step is to identify the patient's pressure pain threshold using the algometer. The Wind-Up protocol is described with the performance of 10 (ten) repetitions of the pressure stimulus in the patient, returning to the zero (or negative of the device). During the fifth (5) and tenth (10) repetitions, we asked the patient to point out his VAS again. As we have already performed 5 pressures until this moment of the test, the tendency is for the patient to perceive a little more the threshold already evaluated, with a little more intensity. Thus, we will have 3 (three) values: at the beginning of the protocol when we evaluate the patient's analog pain threshold. During the 5th (fifth) moment and the 10th (tenth) moment of the test. The result of the temporal summation protocol is the difference between the visual analog school in the 10th (tenth) stimulus and in the initial stimulus. Test interpretation: (positive score = increased pronociception).

European Journal of Pain (2000) **4**: 5–17
doi:10.1053/eujp.1999.0154, available online at http://www.idealibrary.com on IDEAL®

Review Article

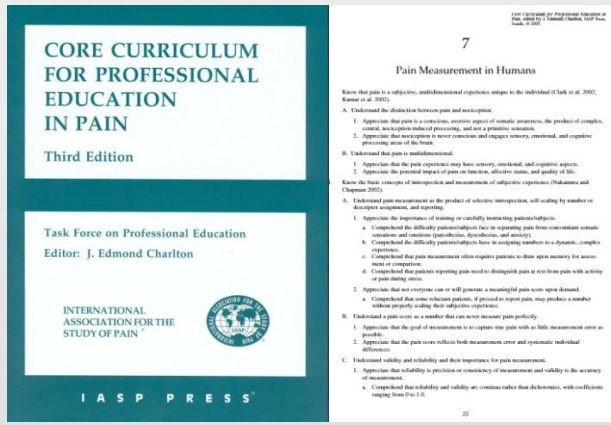
Wind-up and the NMDA receptor complex from a clinical perspective

Per K. Eide

Department of Neurosurgery, The National Hospital, University of Oslo, Oslo, Norway

Comments: Peripheral abnormal mechanisms - temporal summation

"The results of clinical trials with patients with chronic pain suggest that the NMDA receptor may present a novel target for abnormal pain summation modulation."



return

Menu

33 - Female patient, arrives at the consultation presenting low back pain for about 6 (six) years, of slow and progressive origin, without traumatic events described, leading to a low probability of tissue lesions, and greater possibilities of events of increased sensitivity in the tissues. Sedentary and history of depression. Quantitative sensory tests (QST) showed hyperalgesia and allodynia. Thus, a possible hypothesis with the descriptions of the anamnesis leads us to the mechanism:

A — Neuropathic;

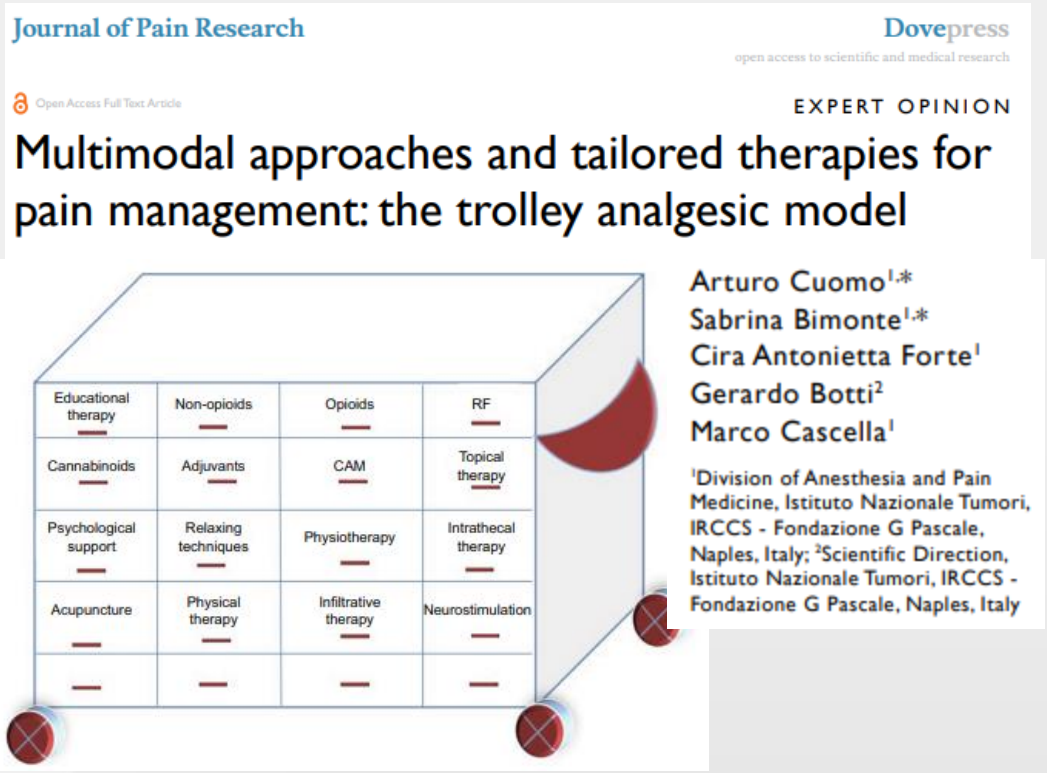
B — Nociceptive;

C — Nociplastic;

D — Neuropathic with nociceptive overlap.

Menu

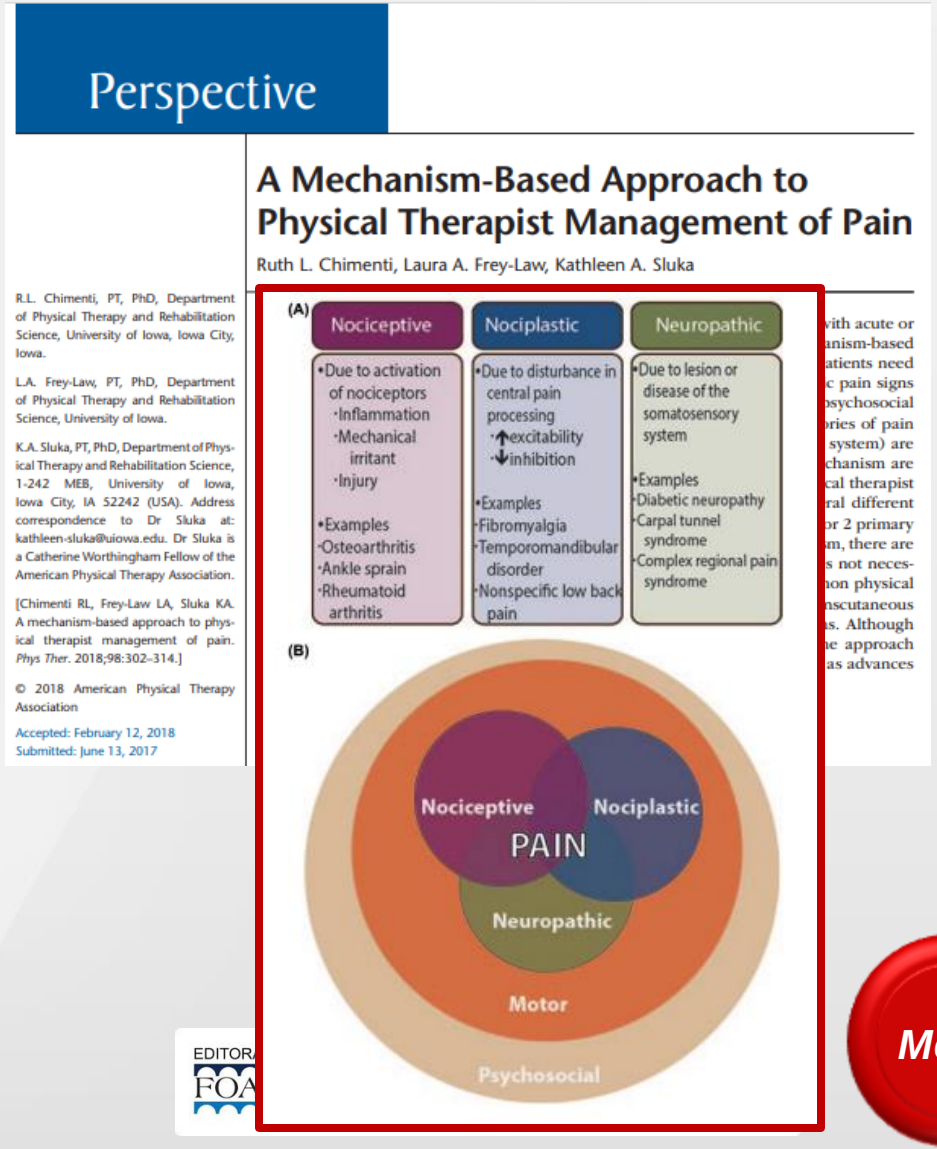
WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Perspective

A Mechanism-Based Approach to Physical Therapist Management of Pain

Ruth L. Chimenti, Laura A. Frey-Law, Kathleen A. Sluka

R.L. Chimenti, PT, PhD, Department of Physical Therapy and Rehabilitation Science, University of Iowa, Iowa City, Iowa.

L.A. Frey-Law, PT, PhD, Department of Physical Therapy and Rehabilitation Science, University of Iowa.

K.A. Sluka, PT, PhD, Department of Physical Therapy and Rehabilitation Science, 1-242 MEB, University of Iowa, Iowa City, IA 52242 (USA). Address correspondence to Dr Sluka at: kathleen-sluka@uiowa.edu. Dr Sluka is a Catherine Worthingham Fellow of the American Physical Therapy Association.

[Chimenti RL, Frey-Law LA, Sluka KA. A mechanism-based approach to physical therapist management of pain. *Phys Ther*. 2018;98:302–314.]

Accepted: February 12, 2018
Submitted: June 13, 2017

(A)

Nociceptive	Nociplastic	Neuropathic
•Due to activation of nociceptors ·Inflammation ·Mechanical irritant ·Injury •Examples ·Osteoarthritis ·Ankle sprain ·Rheumatoid arthritis	•Due to disturbance in central pain processing ·↑excitability ·↓inhibition •Examples ·Fibromyalgia ·Temporomandibular disorder ·Nonspecific low back pain	•Due to lesion or disease of the somatosensory system •Examples ·Diabetic neuropathy ·Carpal tunnel syndrome ·Complex regional pain syndrome

(B)

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Perspective

A Mechanism-Based Approach to Physical Therapist Management of Pain

Ruth L. Chimenti, Laura A. Frey-Law, Kathleen A. Sluka

R.L. Chimenti, PT, PhD, Department of Physical Therapy and Rehabilitation Science, University of Iowa, Iowa City, Iowa.

L.A. Frey-Law, PT, PhD, Department of Physical Therapy and Rehabilitation Science, University of Iowa.

Pain reduction is a primary goal of physical therapy for patients who present with acute or persistent pain conditions. The purpose of this review is to describe a mechanism-based approach to physical therapy pain management. It is increasingly clear that patients need to be evaluated for changes in peripheral tissues and nociceptors, neuropathic pain signs and symptoms, reduced central inhibition and enhanced central excitability, psychosocial factors, and alterations of the movement system. In this Perspective, 5 categories of pain

(A)

Nociceptive	Nociplastic	Neuropathic	Psychosocial	Motor
•Exercise •Massage •TENS	•Education •Exercise •Massage •Manipulation •TENS	•Exercise	•Education •Exercise •Massage	•Education •Exercise •Manipulation

(B)

Nociceptive	Nociplastic	Neuropathic	Psychosocial	Motor
•Topical analgesic •Nonsteroidal Anti-inflammatory •Opioid •Channel blocker	•Serotonin-noradrenaline reuptake inhibitor •Tricyclic antidepressant	•Gabapentinoid	•Serotonin-noradrenaline reuptake inhibitor •Tricyclic antidepressant	•Muscle relaxant

return

Menu

CERTAIN ANSWER!

Pain of slow and progressive origin, without traumatic events described, leading to a low probability of tissue lesions, and greater possibilities of events of increased sensitivity in the tissues (sensitization). Thus, a possible hypothesis with the descriptions of the anamnesis leads us to the nociplastic neurophysiological mechanism. Associated with quantitative sensory tests, with the use of the pressure algometer, evaluating mechanical hyperalgesia/allodynia, following a more local or diffuse pattern (as reported by the patient) without a specific neuroanatomical area.

We used a load of 1kgf for 1 second, within the painful reported area. We request the patient's report of the moment at which the pressure begins to bother. Then we evaluated the threshold on the contralateral side of the painful complaint. Looking at the sensitivity compared to pressure, the painful side has a lower threshold. When we progressed with the tests for thoracic area, we observed a more predominant pain threshold in a body diminid. That is, without a defined neuroanatomical pattern. When we advanced with the evaluation of quantitative sensory tests (QST) with needle prick stimuli, and compared the middle finger of the right hand with the lumbar spine on the right, we observed increased sensory signals and possible hyperalgesia. When we continue the exploration of the test, we observe signs of hyperalgesia and allodynia spread throughout the right diminium and suggest a possible increased pro-nociception in higher regions of the nociceptive pathways. Continuing the evaluation through the Wind-Up or Temporal Summation test, we evaluate the repetition of the stimulus several times and the tendency is for the pain to worsen for the same stimulus, leading to a positive result in the test. When we reached the fifth repetition of the test, he presented more pain reported by the VAS (from 5 went to 7) and in the tenth repetition again more pain (VAS from 7 went to 8), which indicates positive Wind-Up.

The positive test refers to signs of pro-nociception, so an increase in the nociceptive pathway is present. We followed the clinical examination through the MPS evaluating the functioning of the endogenous analgesic pathway, inducing pain in the patient. We standardized the region of the contralateral trapezius to the painful side, inflating the sphygmomanometer until the painful sensation of medium VAS. We waited for 2 minutes, repeated the algometry on the same area initially tested, still on the effect of the conditioning stimulus (sphygmomanometer) presenting an increase in the threshold at the end of the test, which shows a functioning analgesic pathway. The approaches that can contribute to the aid of progression of the painful picture are in exercises that will activate the endogenous opioid system assisting in the neuromodulation of pain through a cascade of analgesic and anti-inflammatory effects.

return

Menu

34 - From the data collected in the anamnesis and clinical examination, the patient presented the tests of Wind-Up (Temporal Summation) positive and CPM (Conditioned Modulation of Pain) functioning. Thus, we opted for physiotherapeutic conduct through manual therapy and kinesiotherapy. With positive Wind-Up, we would have some possibilities for intervention through manual therapy. . When we observe by CPM (Conditioned Pain Modulation) a preserved anti-nociception system, the choice for therapeutic exercises becomes a good strategy. The choice for this conduct can benefit then:

A) grade 2 joint mobilization associated with localized exercises;

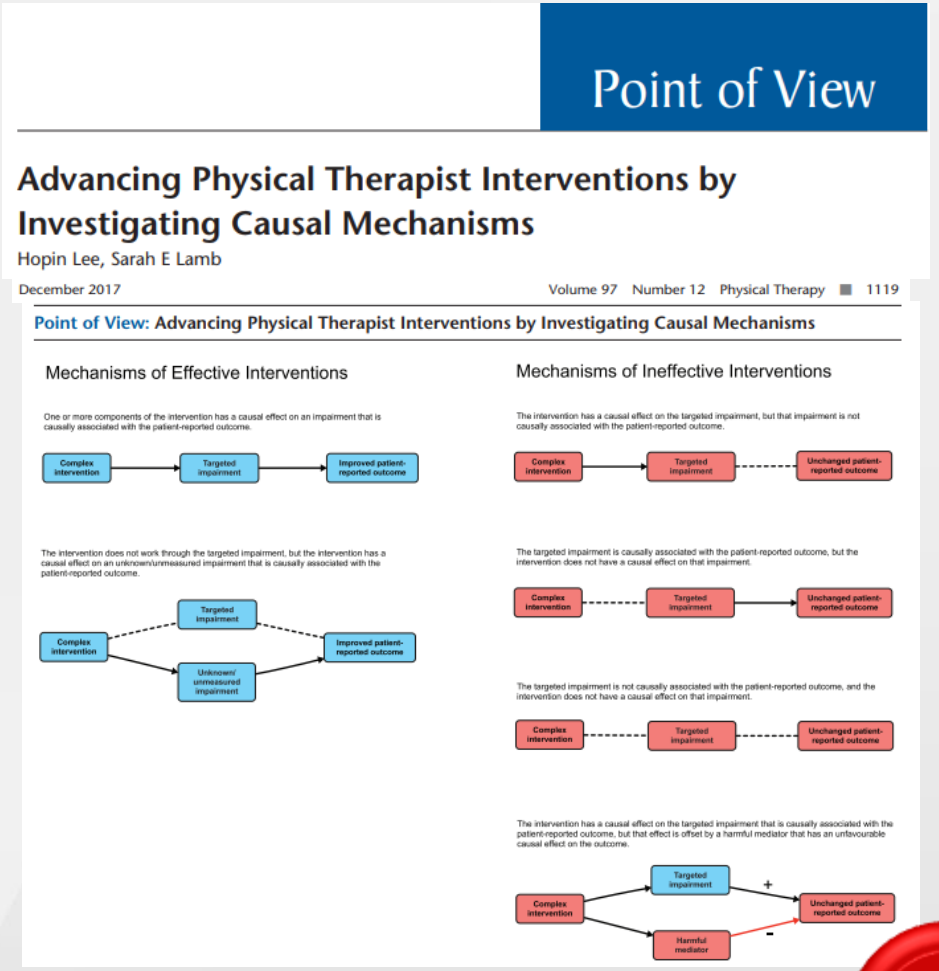
B) joint manipulation (thrust) associated with systemic exercises;

C) joint manipulation (thrust) associated with localized exercises;

D) grade 4 joint mobilization associated with systemic exercises.

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!



Point of View

Advancing Physical Therapist Interventions by Investigating Causal Mechanisms

Hopin Lee, Sarah E Lamb

December 2017 Volume 97 Number 12 Physical Therapy ■ 1119

Point of View: Advancing Physical Therapist Interventions by Investigating Causal Mechanisms

Mechanisms of Effective Interventions

One or more components of the intervention has a causal effect on an impairment that is causally associated with the patient-reported outcome.

Complex intervention → Targeted impairment → Improved patient-reported outcome

The intervention does not work through the targeted impairment, but the intervention has a causal effect on an unknown/unmeasured impairment that is causally associated with the patient-reported outcome.

Complex intervention → Targeted impairment; Unknown/unmeasured impairment → Improved patient-reported outcome

Mechanisms of Ineffective Interventions

The intervention has a causal effect on the targeted impairment, but that impairment is not causally associated with the patient-reported outcome.

Complex intervention → Targeted impairment → Unchanged patient-reported outcome

The targeted impairment is causally associated with the patient-reported outcome, but the intervention does not have a causal effect on that impairment.

Complex intervention → Targeted impairment → Unchanged patient-reported outcome

The targeted impairment is not causally associated with the patient-reported outcome, and the intervention does not have a causal effect on that impairment.

Complex intervention → Targeted impairment → Unchanged patient-reported outcome

The intervention has a causal effect on the targeted impairment that is causally associated with the patient-reported outcome, but that effect is offset by a harmful mediator that has an unfavourable causal effect on the outcome.

Complex intervention → Targeted impairment (+) → Unchanged patient-reported outcome; Complex intervention → Harmful mediator (-)

return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Contents lists available at ScienceDirect

Musculoskeletal Science and Practice

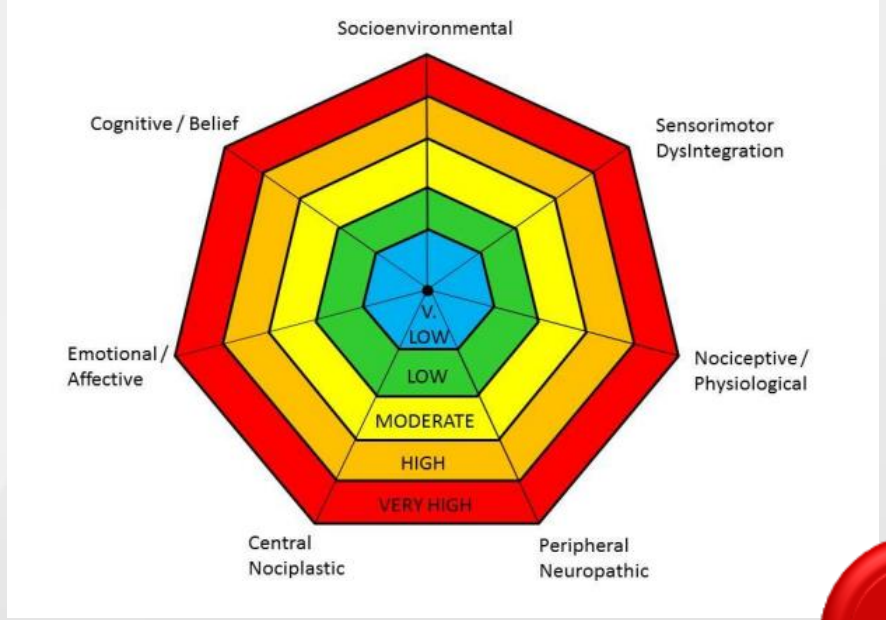
journal homepage: www.elsevier.com/locate/msksp

Original article

A new clinical model for facilitating the development of pattern recognition skills in clinical pain assessment☆

David M. Walton[a,*], James M. Elliott[b]

[a] Faculty of Health Science, Western University Canada, Canada
[b] Faculty of Health Sciences, The University of Sydney, and the Kolling Institute, Royal North Shore Hospital, NSW, Australia



return

Menu

WRONG ANSWER! CLICK ON THE VIDEO, INTERACT AND TRY AGAIN!

Pain Physician 2014; 17:447-457 • ISSN 1533-3159

Epidemiology

Applying Modern Pain Neuroscience in Clinical Practice: Criteria for the Classification of Central Sensitization Pain

Jo Nijs, PhD[1,2], Rafael Torres-Cueco, MSc[3], C. Paul van Wilgen, PhD[4], Enrique Lluch Girbés, MSc[3], Filip Struyf, PhD[1,5], Nathalie Roussel, PhD1,5, Jessica Van Oosterwijck, PhD[1,6], Liesbeth Daenen, PhD[1,7], Kevin Kuppens, MSc[1,5,7], Luc Vanderweeën, MSc[1,8], Linda Hermans, MSc[6], David Beckwée, MSc[1], Lennard Voogt, PhD[1,9], Jacqui Clark, MSc[10], Niamh Moloney, PhD[1,11], and Mira Meeus, PhD[6,7]

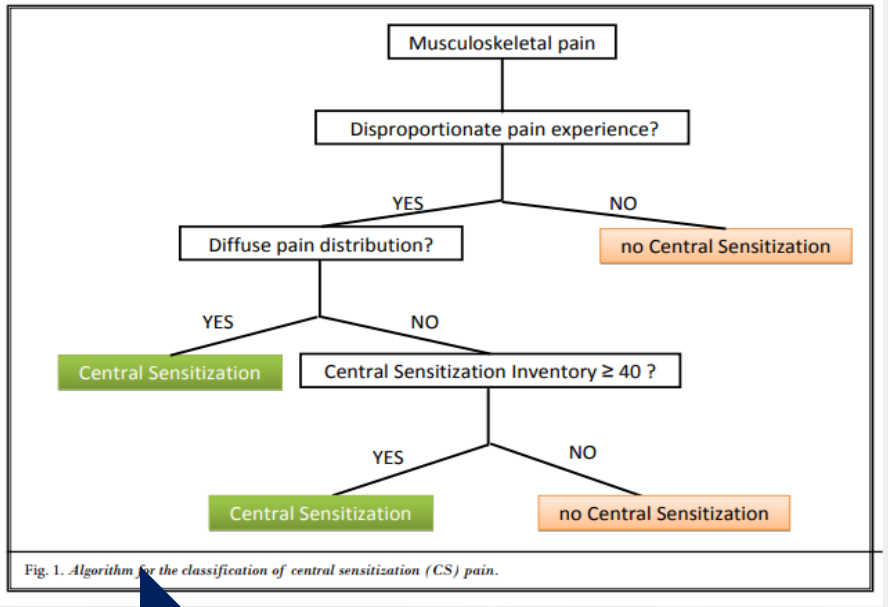


Fig. 1. *Algorithm for the classification of central sensitization (CS) pain.*

American Pain Society
RESEARCH
EDUCATION
TREATMENT
ADVOCACY
PUBLISHED BY ELSEVIER
The Journal of Pain, Vol 20, No 11 (November), 2019: pp 1249–1266
Available online at www.jpain.org and www.sciencedirect.com

Focus Article

Exercise-Induced Hypoalgesia in Pain-Free and Chronic Pain Populations: State of the Art and Future Directions

David Rice,[*,†] Jo Nijs,[‡,§,¶] Eva Kosek,[‖,**] Timothy Wideman,[††] Monika I Hasenbring,[‡‡] Kelli Koltyn,[§§] Thomas Graven-Nielsen,[¶¶] and Andrea Polli[‡,§,¶,‖‖]

return
Menu

CERTAIN ANSWER!

With positive Wind-Up, we would have some possibilities for intervention through manual therapy. Joint manipulation (thrust) or joint mobilization, generating the same therapeutic effect.

The choice for manipulation is based on the fact that a single stimulus will not reproduce the sum of stimuli that the joint mobilization repeatedly can generate on the patient's nervous system, which may contribute to increased sensitivity, not being able to generate analgesic effects using the test as a reference for the choice of the type of maneuver used. The prescription of exercises is also based on this hypothesis.

When we observe by CPM (Conditioned Pain Modulation) a preserved anti-nociception system, the choice for therapeutic exercises becomes a good strategy. However, as the patient has positive Wind-Up, she may not respond well to exercises with a local focus on the painful area, due to the summation of stimuli. Therefore, the choice of exercises that are not analytical but systemic, such as low-moderate intensity aerobic exercise, can help more by stimulating the descending endogenous system of pain neuromodulation.

return

Menu

When we think of a dialogue that contemplates and assists all the content we have learned so far, it should be based on exercises directed to cognition. Through a communication that transfers the theory of Pain Neuroscience Education to clinical practice.

We have reached the end of the Modern Pain quiz, and in this last question I present a practical dialogue between physiotherapist and patient, transcribed and narrated as in section Box 1, 3 and 4, of the article "Applying contemporary neuroscience in exercise interventions for chronic pain: treatment protocol", published in 2017 in BjPT.

Dialogue

Brazilian Journal of Physical Therapy 2017;21(5):378–387

Brazilian Journal of Physical Therapy

https://www.journals.elsevier.com/brazilian-journal-of-physical-therapy

CLINICAL TRIAL PROTOCOL

Applying contemporary neuroscience in exercise interventions for chronic spinal pain: treatment protocol☆

Anneleen Malfliet[a,b,f,*], Jeroen Kregel[b,c], Mira Meeus[b,c,d], Barbara Cagnie[c], Nathalie Roussel[b,d], Mieke Dolphens[c], Lieven Danneels[c], Jo Nijs[a,b,e]

Table 1 Example of an 'activity form' completed by a chronic low back pain patient.

Write down movements/activities of which you think they will worsen your complaints or disorder, and/or that are limited due to your pain	Level of conviction 0----------10 Unconvinced – Extremely convinced
Vacuuming, mopping the floor, bending forward	9
Bending forward and lifting something heavy	10
Carrying groceries on one side	8
Rotational movements of the back	6
Prolonged sitting or standing	6

Exercise program at home

Several exercises, movements and activities used in the exercise program were also practiced at home. However, some general principles were applied:

- The content, frequency and amount of home exercises should always be decided in consultation with the patient.
- Home-exercises should also be applied using a time-contingent approach.
- Home exercises should be implemented in a functional way (e.g. bending forward while unloading the dish-washer).
- Exercises should only be performed at home when the patient is confident and secure to perform the exercise alone.

Box 1: Communication regarding the transfer from theory (PNE) to practice (cognition-targeted exercises).

lot regarding the underlying mechanisms of your pain problem. I know that you have got a lot of new information. How are you processing this? Do you feel like this new knowledge is applicable to your situation?''
Patient (P): *''Well, as you indicate, it has been a great deal of new information, a lot of things I have never heard of, but it is all very recognizable and it gives me a little relieve. I am still a bit reticent, but I feel confident.''*
T: *''Do you now understand how pain, behavior, thoughts and emotions are related and how they all influence and maintain each other? Is it clear that avoiding certain painful or fearful movements will maintain the pain problem?''*
P: *''Yes, that is clear, but I do not see how we will change this?''*
T: *''Well, that is something we will do together during the next step of this therapy, in which we will initiate certain movements and activities. During these movements/activities we will no longer pay attention to the pain, this pain will no longer be of any value to you. Do you understand why?''*
P: *''Because the pain signal is not a reliable signal and not an accurate representation of what is effectively going on in my neck/back?*
T: *''Indeed! This means that when pain occurs while performing a certain exercise, you will not stop this exercise. You will complete the exact amount of repetitions we agreed on before starting the exercise. Do you feel confident about this approach?''*
P: *''I understand why I have to do this, but I am still a bit nervous about actually doing it.''*
T: *''That makes sense, that is why we will start with easily accessible exercises. I will guide you, perform the exercises together with you and all exercises will be applied in mutual agreement of both of us.*

Box 3: Communication regarding cognition-targeted motor control training.

contraction (e.g. 10 s) of some specific muscles in the painful region. Do you have any idea why we are activating these specific muscles?''
Patient (P): *''Maybe to strengthen these muscles so they will hurt less?''*
T: *''Remember all the things we discussed before. We then agreed on the fact that you have become more sensitive to signals coming from the neck/back than people without chronic neck/back pain. Remember that I have examined your neck/back before you started this program and that I did not find any important abnormalities in muscle strength or endurance. What does that tell you about this exercise now?''*
P: *''That it is not aiming at strengthening my muscles, but that it is targeting my pain system? But how does that work?''*
T: *''Indeed! When activating muscles, this will send signals to the brain. Normally these signals should be interpreted correctly, leading to the information that your muscles are working. Do you know why this leads to pain in your case?''*
P: *''Yes, because of the education I now understand that certain signals coming from my neck/back are interpreted as pain or danger, while they are just messages of movement.''*
T: *''Correctly, so when you are performing this exercise, muscles are being activated and sending signals to the brain. It is important that when you experience pain during this exercise, you are aware of this information and that you know that the pain is not a reliable signal.''*

Box 4: Communication regarding regular movement.

Therapist (T): ''During prolonged sitting, your muscles are logically registering this and certain signals will be produced. We already discussed that this is giving you pain because of your hypersensitive pain system, while normally (in non-pain persons) this should give at most a certain inconvenience. Could you come up with an explanation why regular movement will help you in this situation?''
Patient (P): *''Because regular movement will prevent these certain signals to be produced?''*
T: *''Indeed, that is one of the explanations! Besides this, there is also another very important mechanism that becomes active during movement. Do you remember the example I gave about the cyclists who reached the finish during a race even a broken collarbone?''*
P: *''Yes, I remember the story. I think it had something to do with what you called the 'pharmacy' in our body, which contains very strong analgesics.''*
T: *''Correct! Do you remember what can activate this pharmacy?''*
P: *''Physical activity.''*
T: *''Indeed, so does it sound logical th… movement during prolonged sitting wi… relieve by activating the pharmacy?''*
P: *''Yes, I guess it is worth a try.''*

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 1!

Physical therapist

Patient

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 1!

Physical therapist

Patient

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 1!

Physical therapist

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 2!

Physical therapist

Patient

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 2!

Physical therapist

Patient

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 2!

Physical therapist

Patient

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 2!

Fisioterapeuta

Paciente

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 3!

Fisioterapeuta

Paciente

return

Menu

CLICK ON THE VIDEO AND INTERACT WITH THE COMMUNICATION THROUGH EXERCISES AIMED AT COGNITION – DIALOGUE 3!

Fisioterapeuta

Paciente

return

Menu

THANK YOU VERY MUCH!