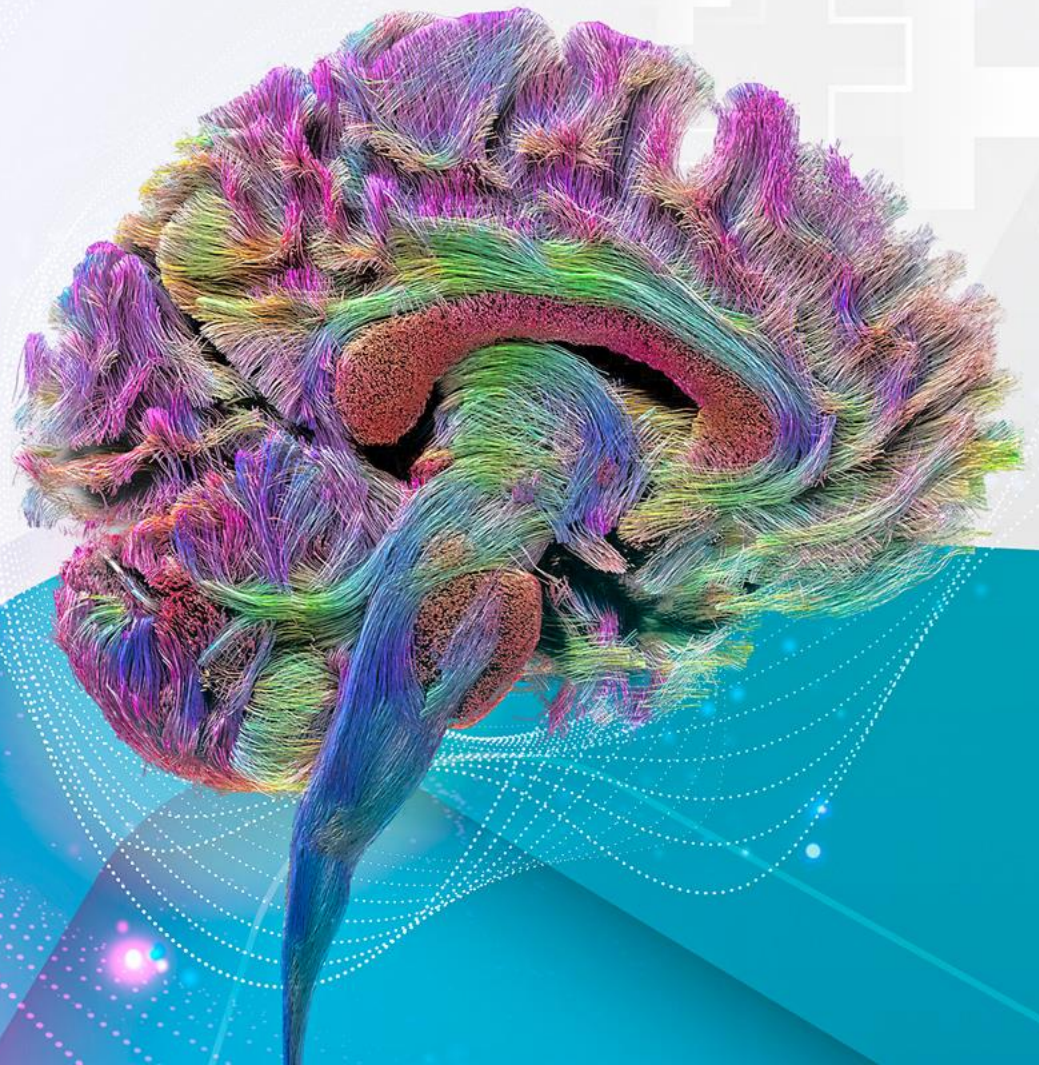

QUESTIONADOR

BASEADO NA MODERNA NEUROCIÊNCIA DA DOR

MESTRADO EM ENSINO EM CIÊNCIAS DA SAÚDE E MEIO AMBIENTE - MECSMA (UniFOA)

→INICIAR

CRÉDITOS | EDUCAÇÃO EM DOR | CURIOSIDADES

Autores:
Gustavo Ferraz Cardozo
Carlos Alberto Sanches Pereira

QUESTIONADOR COMO PRODUTO EDUCACIONAL

Olá, O Quiz QuestionaDor propõe um produto de ensino baseado na moderna neurofisiologia da dor. A proposta se baseia na teoria de aprendizagem significativa (TAS) através de conhecimento prévio do aluno, cognistivismo proposto pelo teórico David Ausubel, utilizando subsunçores como ferramenta educacional no ensino da novas evidências em neurociência da dor. O Quiz conta com 35 perguntas de múltiplas escolhas, onde o aluno possui 4 alternativas, sendo apenas 1 correta. Ao assinalar a respostas correta, conta com uma explanação mais ampla sobre a pergunta, dentro do contexto biopsicossocial integrado a educação em dor (PNE – Pain Neuroscience Education). Ao assinalar a alterativa errada, contará com um recurso de ensino digital (podcast e/ou vídeo) que o auxilie até a escolha da alternativa correta, fundamentada em artigos científicos sobre novos conceitos em dor. O Quiz conta com perguntas sobre a neurofisiologia da dor aguda e crônica, educação em dor, epidemiologia da dor, mecanismos neurofisiológicos envolvidos com a dor, classificação e sub-classificação da dor crônica, avaliação moderna da dor e casos clínicos envolvendo dor. O Quiz foi desenvolvido como produto educacional do Mestrado em Ensino em Ciências da Saúde e Meio-Ambiente (MECSMA) da Fundação Oswaldo Aranha - Centro Universitário de Volta Redonda – UniFOA.

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QUESTIONADOR COMO PRODUTO EDUCACIONAL

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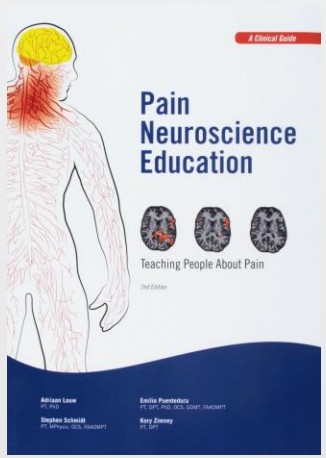
QUESTIONADOR COMO EDUCAÇÃO EM DOR

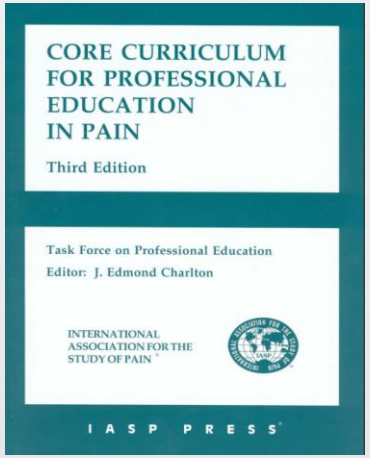
Narrative Review

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]





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QUESTIONADOR COMO EDUCAÇÃO EM DOR

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]

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DESENVOLVEDORES

GUSTAVO FERRAZ CARDOZO – AUTOR

Graduado em Fisioterapia, Especialista em Fisioterapia Esportiva pelo Conselho Federal de Fisioterapia e Sociedade Nacional de Fisioterapia Esportiva (COFFITO/SONAFE), Osteopata CO – Escuela de Osteopatia de Madrid, Pós-Graduado em Fisioterapia Cardiorespiratória Funcional, Pós-Graduado em Acupuntura, atualmente cursa o Mestrado Profissional em Ensino em Ciências da Saúde e Meio Ambiente (MECSMA) do UniFOA, servidor concursado no município de Itatiaia/RJ, atuante no Centro Municipal de Reabilitação e Fisioterapia, Docente dos curso de Pós-Graduação em Fisioterapia Traumato-Ortopédica Funcional do Centro Universitário de Barra Mansa (UBM), Pós-Graduação em Reabilitação Musculoesquelética pelo Movimento da Fase Educacional (RPX) e Pós-Graduação em Fisiologia do Exercício Aplicada ao Treinamento do Centro Universitário Geraldo Di Biasi (UGB).

CARLOS ALBERTO SANCHES PEREIRA - ORIENTADOR

Graduado em Ciências Biológicas, Especialista em Bioquímica, Especialista em Hematologia pela UFRJ em 2000, Mestre em Ciência e Tecnologia de Alimentos pela UFRRJ (2001) na área de concentração em Microbiologia Aplicada; Doutor em Biotecnologia Industrial (2007) EEL-USP na área de concentração em Microbiologia Aplicada. Possui experiência em Biotecnologia de micro-organismos: estudos com Lactobacillus e seu papel na estimulação da imunidade; Microbiologia Clínica e Médica; Hematologia Clínica e Laboratorial. Docente/Orientador do Mestrado Profissional em Ensino de Ciências da Saúde e do Meio Ambiente do UniFOA, programa no qual desenvolve estudos relacionados ao uso de atividades lúdicas como ferramenta para o ensino em Ciências Biológicas e Saúde. Estuda também os aspectos epidemiológicos de bactérias isoladas de Otites em Cães, e sua relação com a conduta terapêutica e com o ensino médico.

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Menu

1 - Segundo as atualizações da IASP nos últimos anos, sobre o conceito de dor e nocicepção, podemos dizer que:

A) são sinônimos;

B) são trajetos da via nociceptiva;

C) são fenômenos distintos;

D) a dor é determinada pela atividade dos neurônios sensitivos.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Narrative Review

PAIN

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

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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."

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CERTA RESPOSTA!

Dor e nocicepção são fenômenos diferentes: a experiência da dor não pode ser reduzida à atividade nas vias sensoriais. Segundo a última atualização da IASP, dor é: *"uma experiência sensorial e emocional desagradável associada a dano real ou potencial ao tecido, ou descrito em termos de tais danos "é muito claro que requer subjetividade, que por sua vez requer consciência e capacidade de avaliar um estímulo / situação*."

Narrative Review

PAIN

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2 - O nociceptor é um receptor sensitivo de alto limiar do sistema nervoso somatossensorial periférico capaz de transduzir e codificar estímulos nocivos. são basicamente classificados pelo seu diâmetro e grau de mielinização, o que determina o seu grau de velocidade de condução. Existem 2 (duas) classes principais de nociceptores, são eles:

A

Os A-Delta são mielinizados e de diâmetro médio, e são responsáveis pela transmissão de dor rápida (5 a 30 m/s – unimodais, mecânicos e térmicos) e os Tipo C são amielínicos e de diâmetro pequeno, que transmitem a nocicepção de forma lenta (menor que 1 m/s – polimodais, mecânicos, térmicos e químicos);

B

Os A-Delta são amielinizados e de diâmetro pequeno, são responsáveis pela transmissão lenta da dor (menor que 1m/s) e os Tipo C são mielínicos e grande diâmetro e transmitem a nocicepção de forma rápida (5 a 30m/s);

C

Os A-Gama são amielinizados e de diâmetro pequeno, são responsáveis pela transmissão lenta da dor (menor que 1m/s) e Tipo A são mielínicos e grande diâmetro e transmitem a nocicepção de forma rápida (5 a 30m/s);

D

Os neoespino-talâmicos lentos e o paleoespino-talâmico rápidos.

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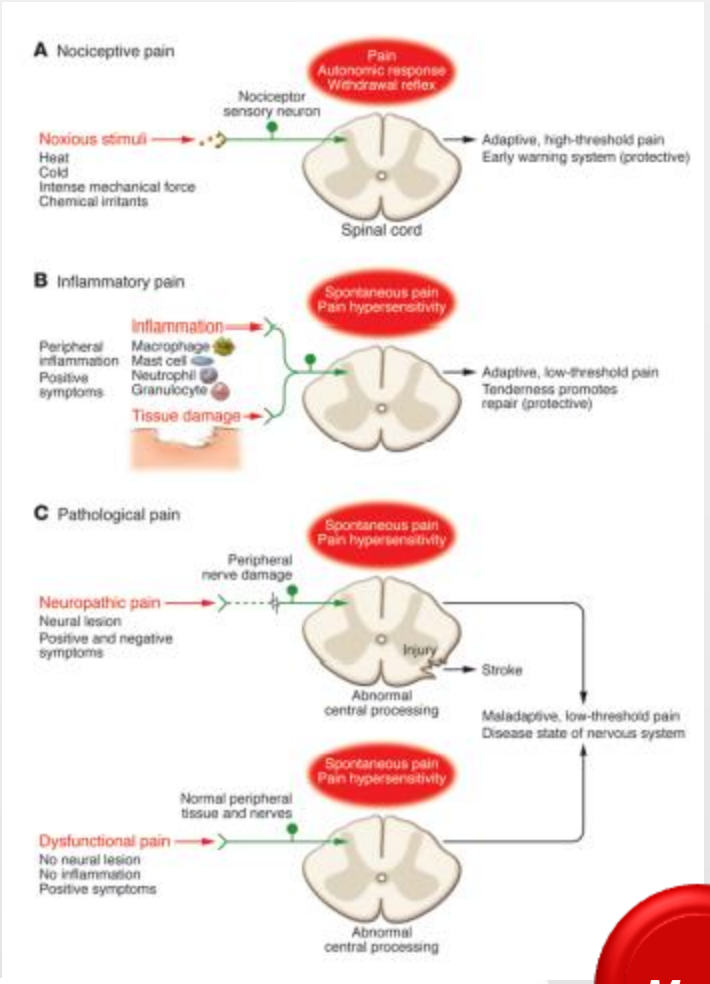
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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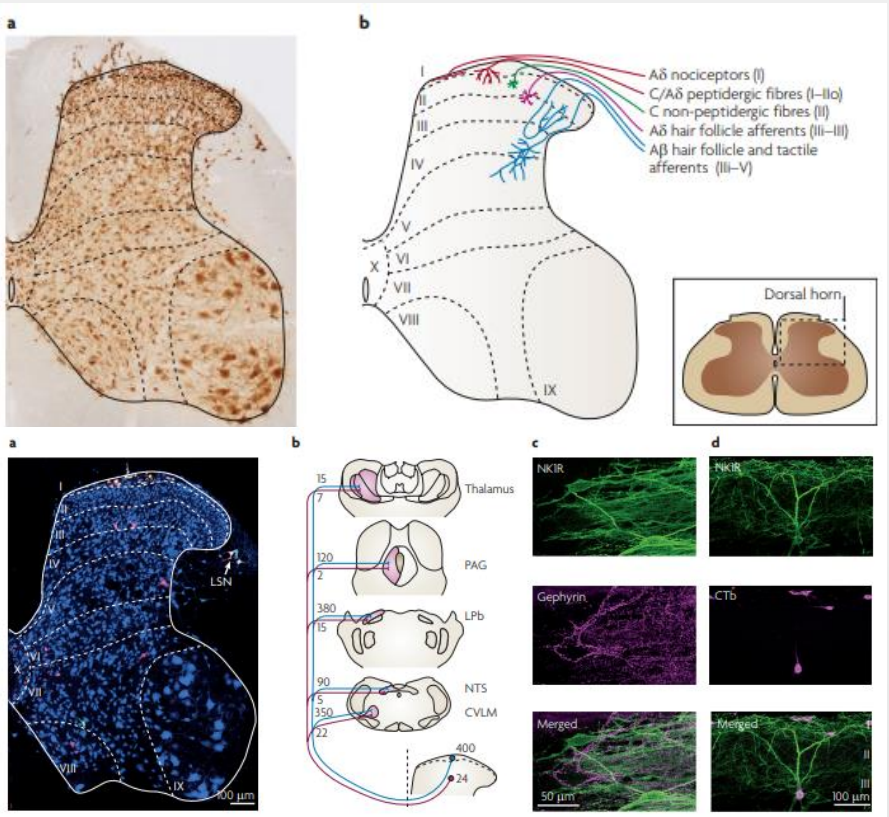

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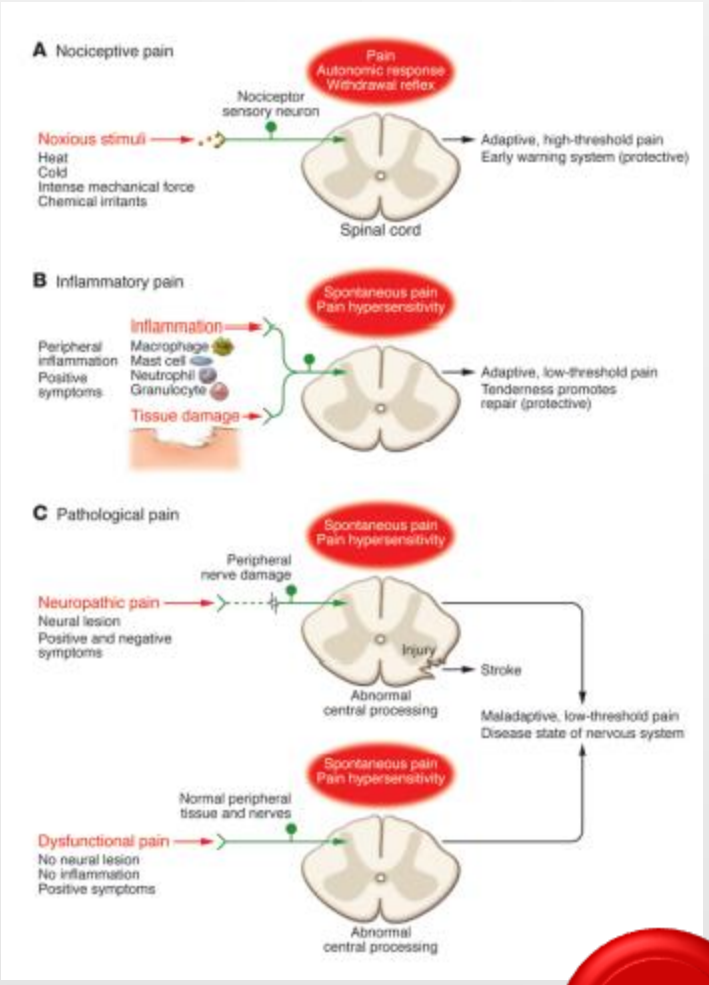

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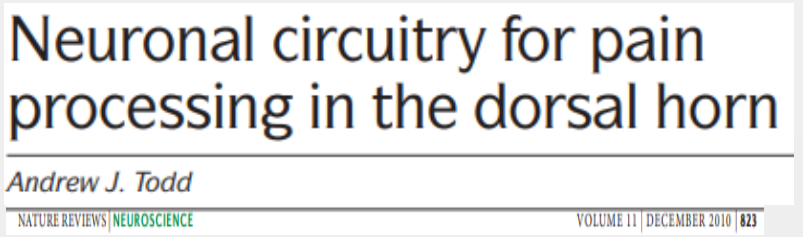
CERTA RESPOSTA!

Existem 2 (duas) classes principais de nociceptores. Os A-Delta são mielinizados e de diâmetro médio, e são responsáveis pela transmissão de dor rápida (5 a 30 m/s); são mecânicos e térmicos (unimodal;) havendo ainda uma divisão em dois grupos funcionais; o primeiro grupo A-Delta1respondem a estímulos mecânicos e químicos e têm alto limiar de calor, sensibilizando no contexto de lesão do tecido; é uma via que provavelmente respondem a primeira dor à estímulos mecânicos, as fibras do grupo A-Delta2 tem um limiar de calor mais baixo e alto limiar mecânico envolvidas em limiar rápidos de dor por exemplo ao calor; Os Tipo C são amielínicos e de diâmetro pequeno, que transmitem a nocicepção de forma lenta (menor que 1 m/s); são mecânicos, térmicos e químicos (polimodal); são classificados não de forma funcional, mas sim de condição molecular com base nos receptores e a neuroquímica subjacente a sua expressão; levando assim a uma ampla gama de marcadores estudados com objetivo de definir subpopulações neuronais e correlacioná-las com a propriedade de resposta desses receptores


What is this thing called pain?

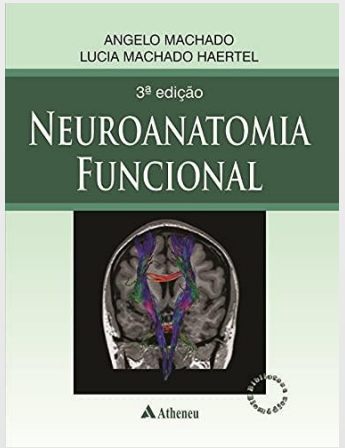
Clifford J. Woolf

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



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3 – Dor aguda e dor crônica se caracterizam por uma condição de carácter temporal, onde:

A dor aguda têm um papel protetor e dor crônica é uma doença;

B a dor crônica é uma dor aguda não curada;

C dor aguda e crônica tem um papel protetor;

D são resultados do subproduto da ativação das terminações nervosas livres.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Narrative Review

PAIN

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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

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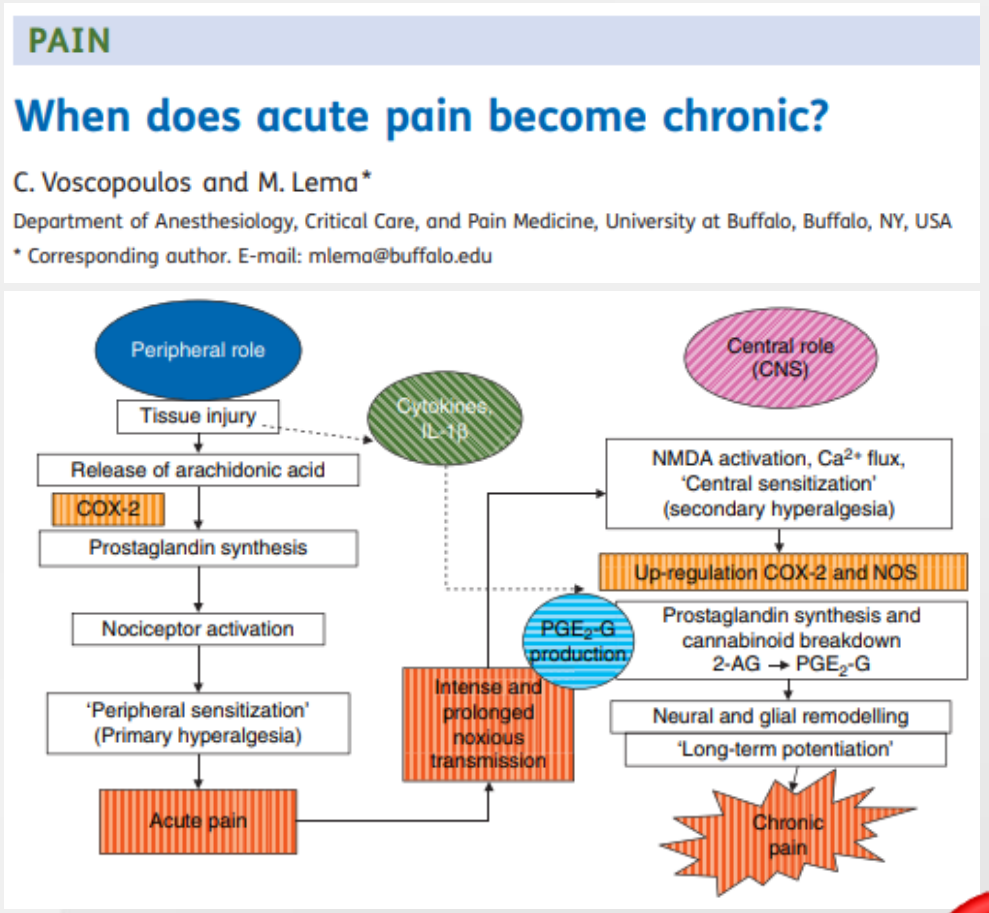
RESPOSTA ERRADA! CLIQUE NO AÚDIO, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



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CERTA RESPOSTA!

Segundo a força-tarefa da IASP, na publicação: *A revisão da Associação Internacional para o Estudo da Dor, definição de dor: conceitos, desafios, e compromissos***,** foi argumentado que a dor é mais do que um sintoma, que a dor crônica pode ser uma doença com curso clínico próprio, e daí a definição deve refletir essa perspectiva. A dor aguda tem um papel protetor de fundamental importância para nossa homeostase e sobrevivência, nos alertando e impulsionando a preservação da integridade do nosso corpo.


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?

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

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

continuar

Menu

4 - A dor é uma experiência complexa e única para cada indivíduo. Portanto:

A. A dor pode ou não ocorrer como resultado de um dano tecidual ou dano potencial do tecido inervado por nociceptores;

B. A dor ocorre como resultado de um dano do tecido inervado por nociceptores;

C. O impacto da dor não se estende além da sua percepção, e não afeta o estado emocional e social do indivíduo;

D. Não há impacto significativo nas atividades das pessoas com dor aguda e crônica, tanto recreacionais quanto de vida diária.

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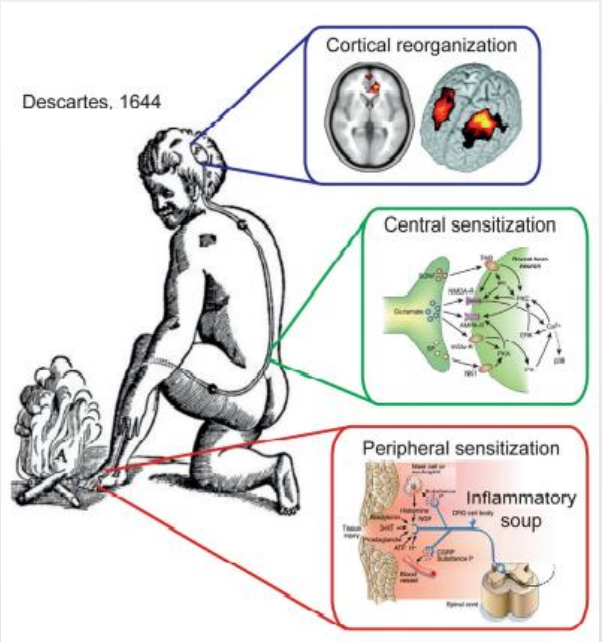
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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



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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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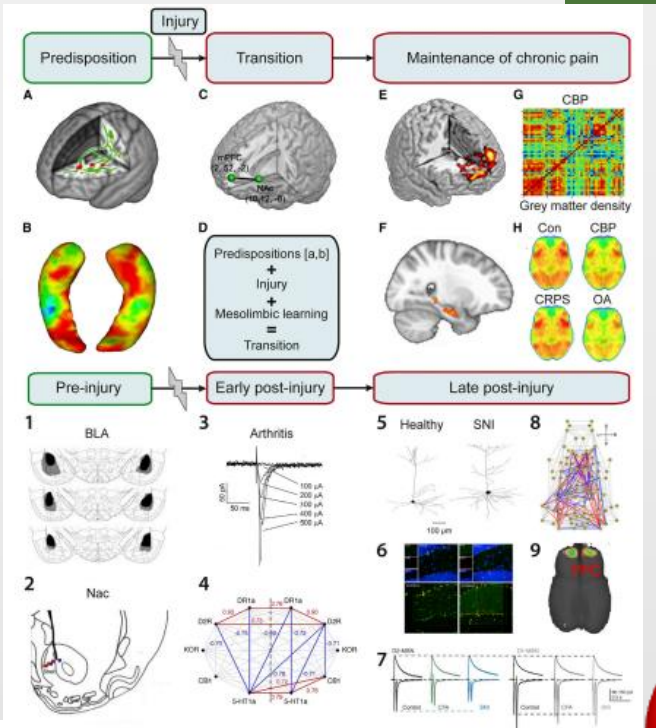

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Nociception, Pain, Negative Moods, and Behavior Selection

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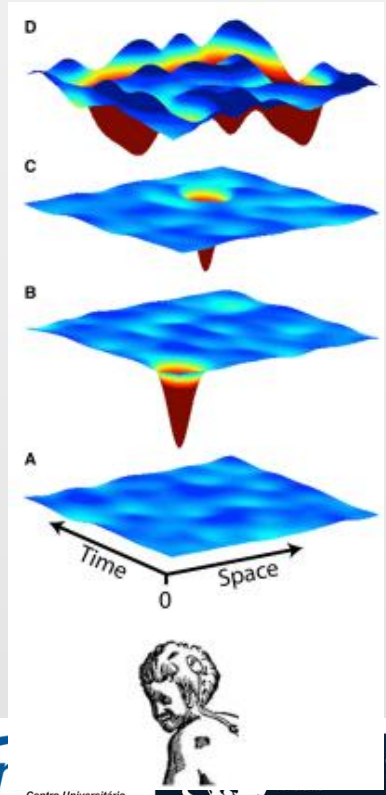

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Menu

CERTA RESPOSTA!

A dor é uma experiência complexa e única para cada indivíduo, sendo assim, se relaciona com ensino-aprendizagem ao longo de suas experiências durante sua vida. O impacto da dor se estende além da sua percepção, podendo afetar seu estado emocional e suas relações sociais. A dor é o principal motivo pelo qual uma pessoa procura atendimento médico. A dor crônica afeta 1/3 da população mundial e 20% desses indivíduos relatam dor de moderada a intensa.

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

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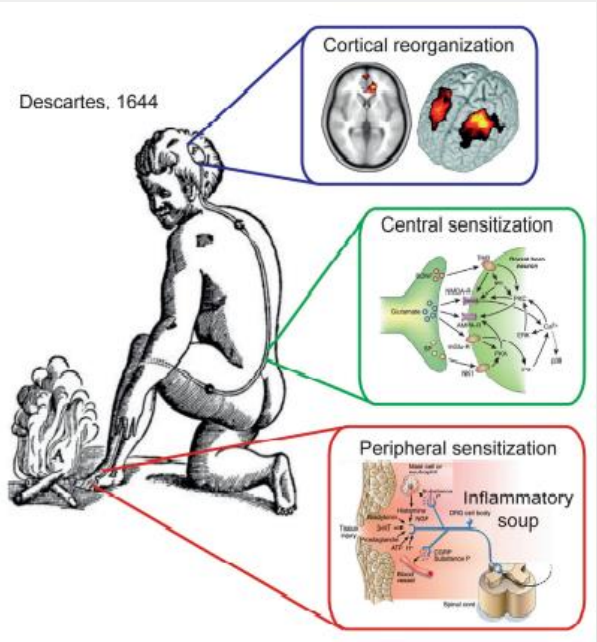

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5 - A educação em neurociência da dor (PNE - Pain Neuroscience Education) usa a ciência contemporânea da dor para educar os pacientes sobre a natureza biopsicossocial de sua experiência de dor. Os principais desfechos que são modificados com a PNE são:

A — A catastrofização, a ansiedade, a incapacidade e as restrições de movimento;

B — A força, a flexibilidade e tato epicrítico;

C — A hipervigilância, o equilíbrio, a coordenação motora e restrições de movimento;

D — O equilíbrio estativo e dinâmico, a marcha e tato sensório-discriminativo.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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*

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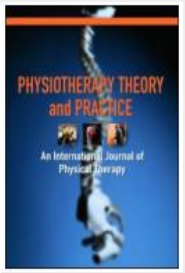
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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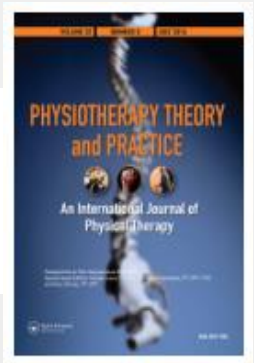
Menu

CERTA RESPOSTA!

Os objetivos da PNE são de diminuir as crenças, medos, vieses e mitos sobre dor. Diminuir as limitações das atividades e proporcionar conhecimento adequado sobre dor, facilitando estratégias de enfrentamento e engajamento. Ela é indicada como ferramenta que pode modificar o conhecimento dos pacientes sobre seu estado doloroso, alterar e propor novos conceitos sobre a neurociência da dor.

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]

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6 - No livro clássico “Explicando a Dor”, David Butler, na seção 5 intitulada como “lidando com a vida e com a dor”, o autor considera que “lutadores ativos” tratam a dor e muitas outras questões de saúde, melhor que os “lutadores passivos”. Considerando essa proposta do autor, são estratégias ativas no engajamento do tratamento:

A. Aprender sobre o problema, fazer planos, explorar maneiras de se movimentar e explorar e cutucar as “bordas” da dor;

B. Explorar maneiras de movimentar, acreditar que a outras pessoas tenham as respostas, esperar que as coisas aconteçam e não fazer nada;

C. Traçar planos, repouso, evitar movimentos que cutucam as “bordas da dor” e procurar um profissional do movimento para começar os exercícios;

D. Aprender sobre o problema, traçar metas, explorar maneiras de se movimentar e evitar explorar e cutucar as “bordas” da dor.

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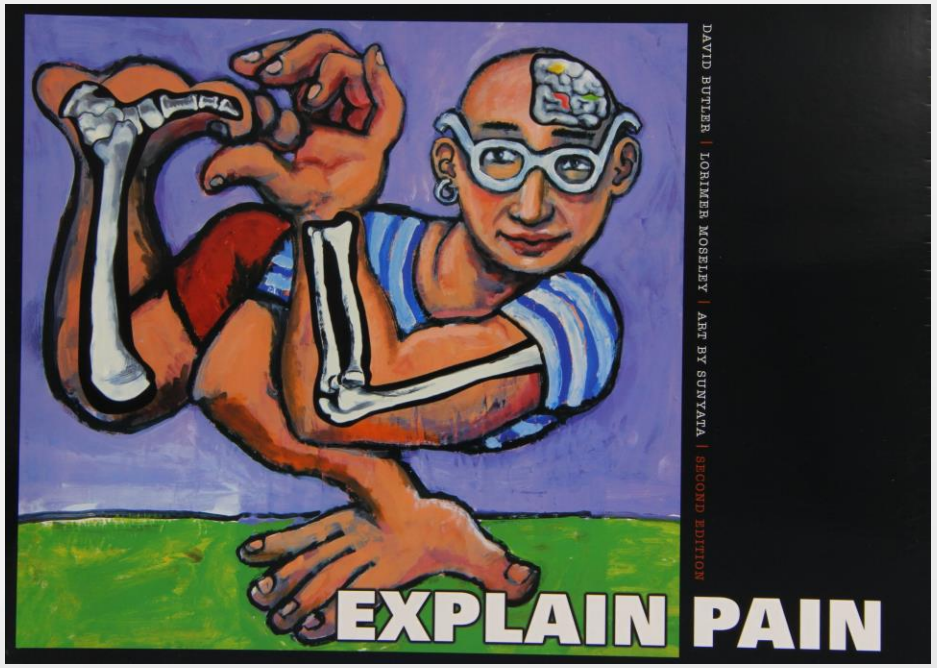
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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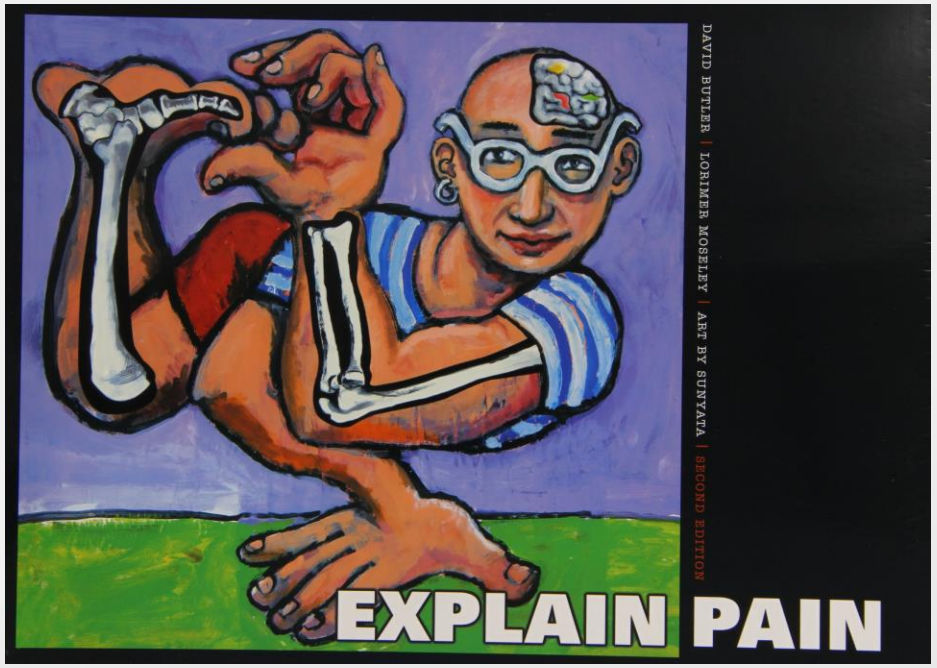

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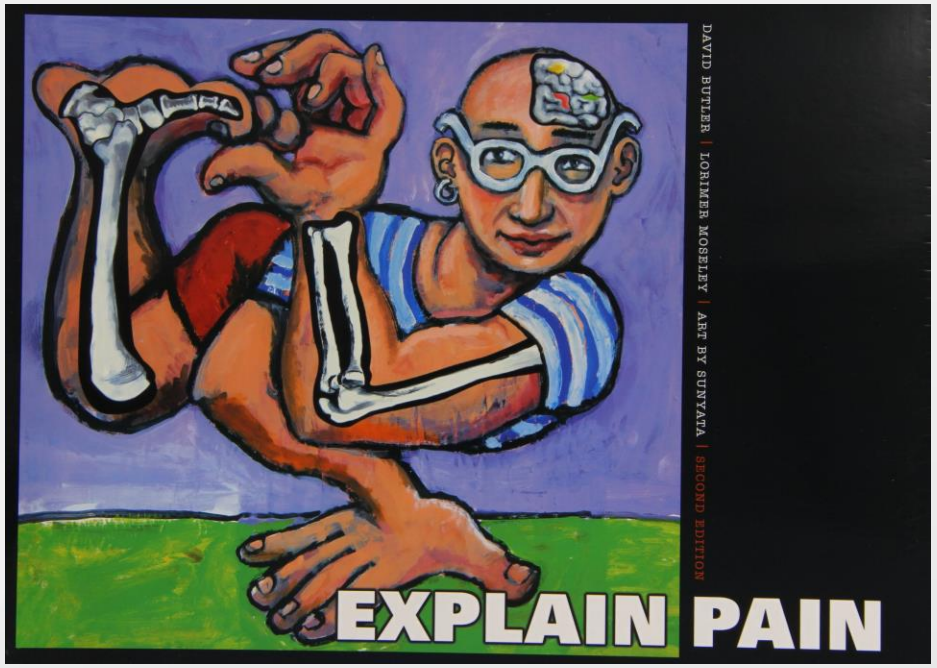

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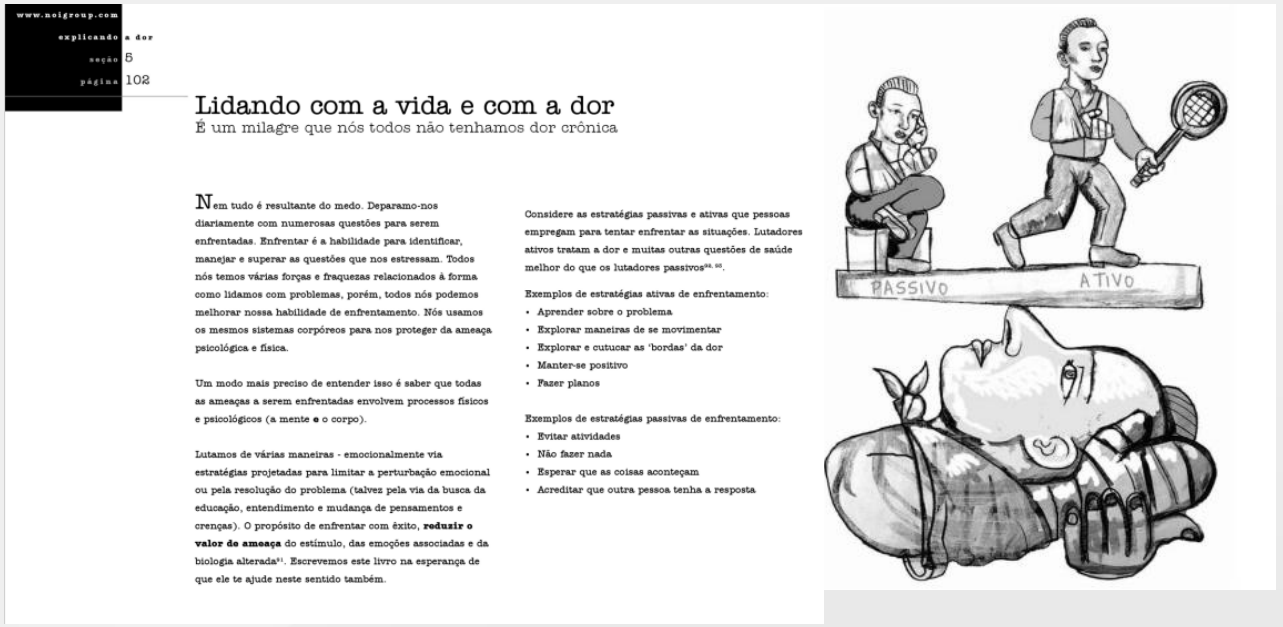
CERTA RESPOSTA!

“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). 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.”


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

retornar

Menu

7 - Ainda baseado no livro “Explicando a dor” e segundo a PNE (Pain Neuroscience Education) entender o quanto puder sobre o que está causando a sua dor, e não somente sobre o que poderia fazer em relação a ela são recomendações propostas pelos autores a IASP. Portanto:

A

Pessoas sem qualquer treinamento nas profissões da área da saúde ou em biologia podem compreender a fisiologia da dor e entender sobre a sua fisiologia dor reduz o valor de ameaça da dor;

B

Pessoas com formação na área da saúde ou biologia devem compreender a fisiologia da dor e entender sobre a fisiologia da dor pode levar a hipervigilância aumentando o valor de amaça da dor;

C

Médicos e fisioterapeutas devem compreender sobre a fisiologia da dor e somente esses profissionais devem explicar as condições para a população, evitando crenças, vieses, nocebos e hipervigilância sobre a ameaça de dor;

D

Fisioterapeutas devem compreender sobre a fisiologia da dor e somente esses profissionais devem explicar as condições para a população, evitando crenças, vieses, nocebos e hipervigilância sobre a ameaça de dor.

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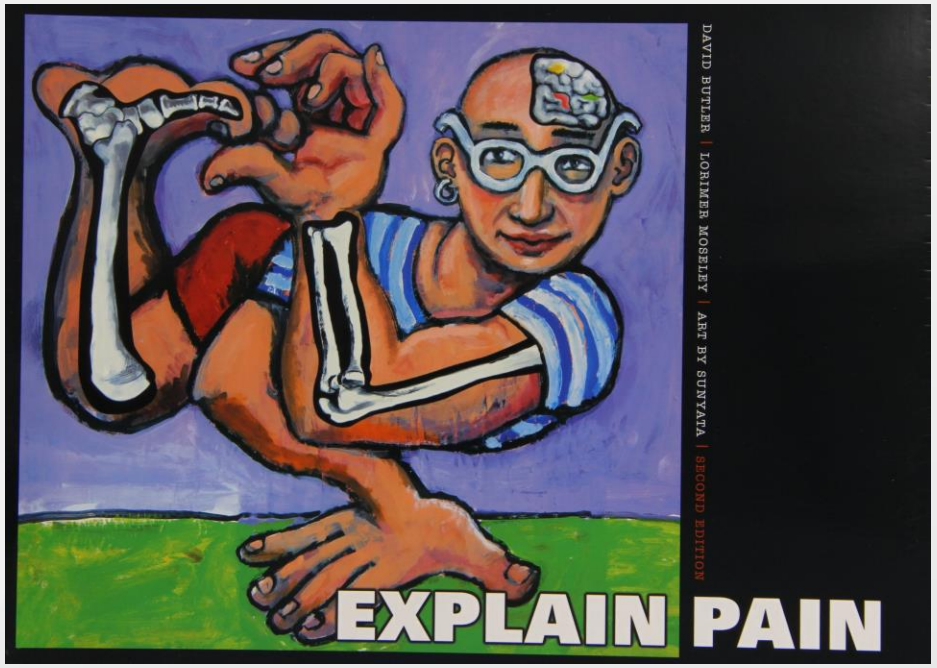
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



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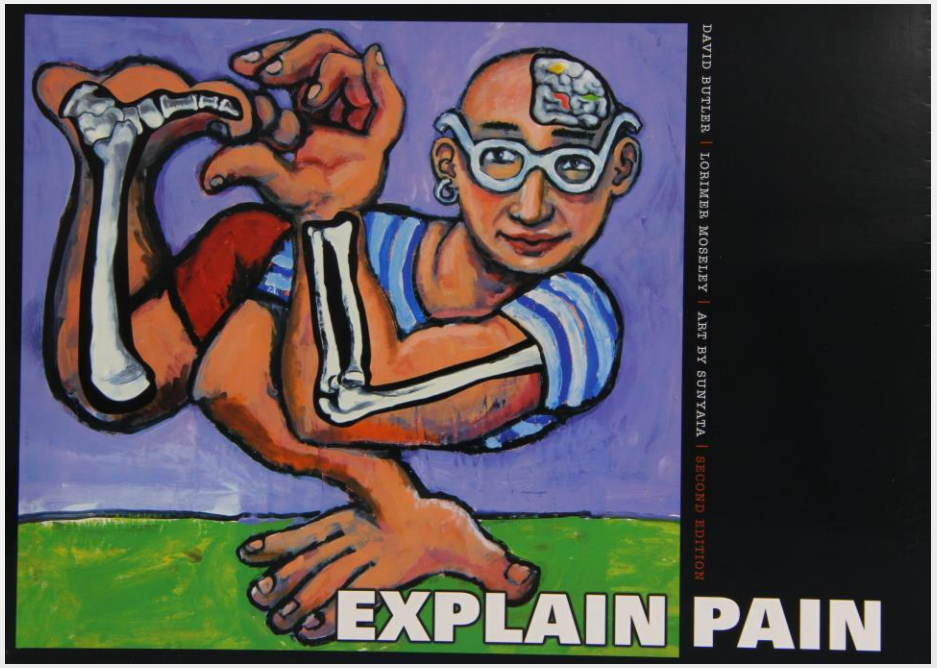
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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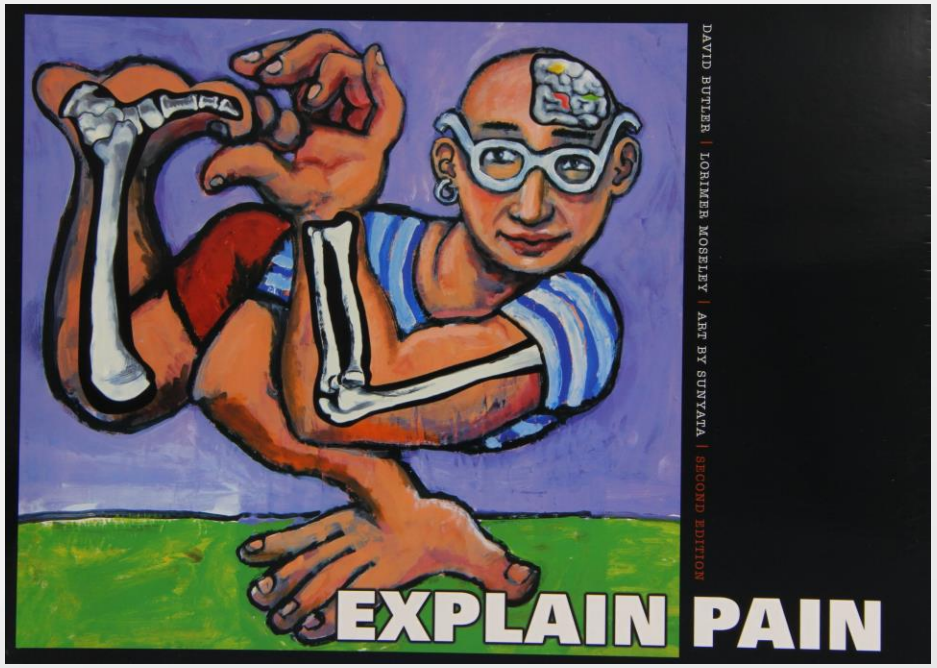
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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CERTA RESPOSTA!

Pessoas sem qualquer treinamento nas profissões de saúde ou em biologia podem compreender a fisiologia da dor. Um dos objetivos de se entender a fisiologia da dor é facilitar o processo chamado “aprendizagem profunda”, na qual a informação é retida, entendida e aplicada aos problemas disponíveis. Pode-se pensar que, simplesmente aprender sobre o que fazer, mas não aprender o porquê, é como uma aprendizagem “superficial”, que representa quando a informação é lembrada, mas não é entendida ou integrada nas atitudes e crenças.

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

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8 - Os estudos publicados por Barcellos e colaboradores em 2017 e Aguiar e colaboradores em 2021, evidenciaram que a epidemiologia da dor crônica no Brasil tem predominância de gênero, região demográfica e mecanismo neurofisiológico. Sendo estatisticamente apresentados:

A) 62,50% dos adultos do sexo feminino, sudeste e mecanismo neuropático;

B) 45,59% dos adultos do sexo feminino, centro-oeste e mecanismo nociceptivo;

C) 42% adultos sem predominância de sexo, centro-oeste e mecanismo nociplástico;

D) 40% dos adultos e 12% de crianças e adolescentes, nordeste e mecanismo neuropático;

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

BJPT Brazilian Journal of Physical Therapy

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

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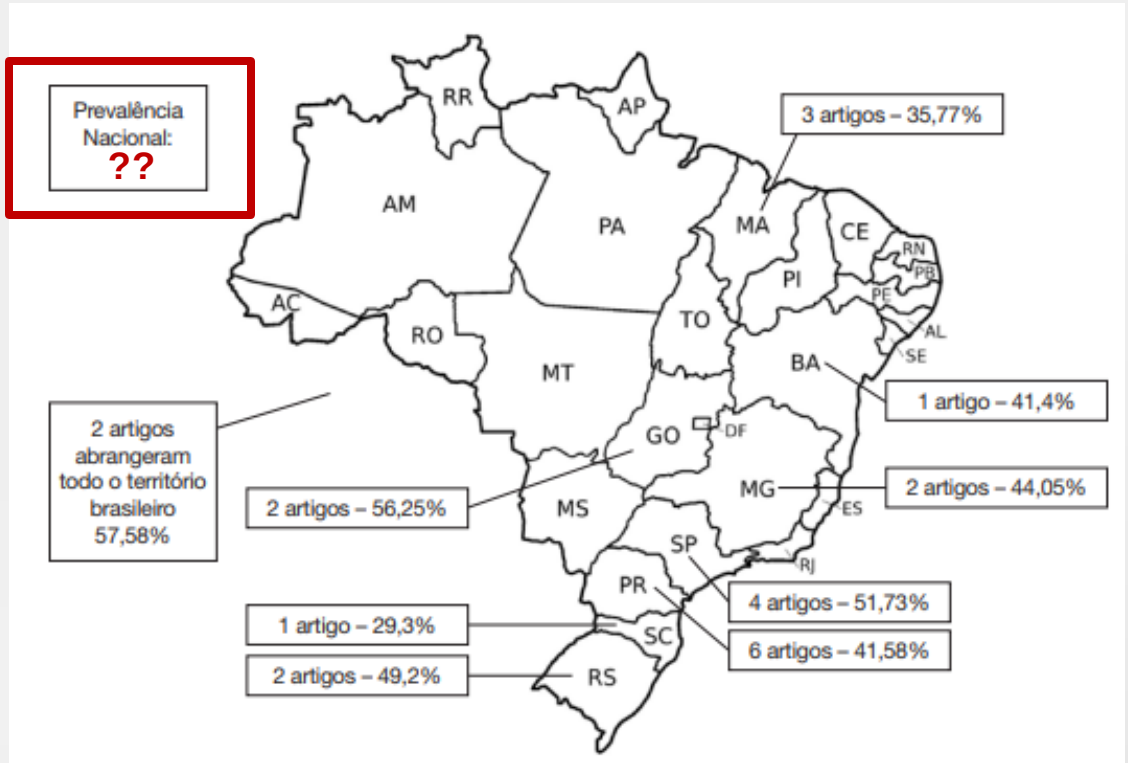
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

BJPT Brazilian Journal of Physical Therapy

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]

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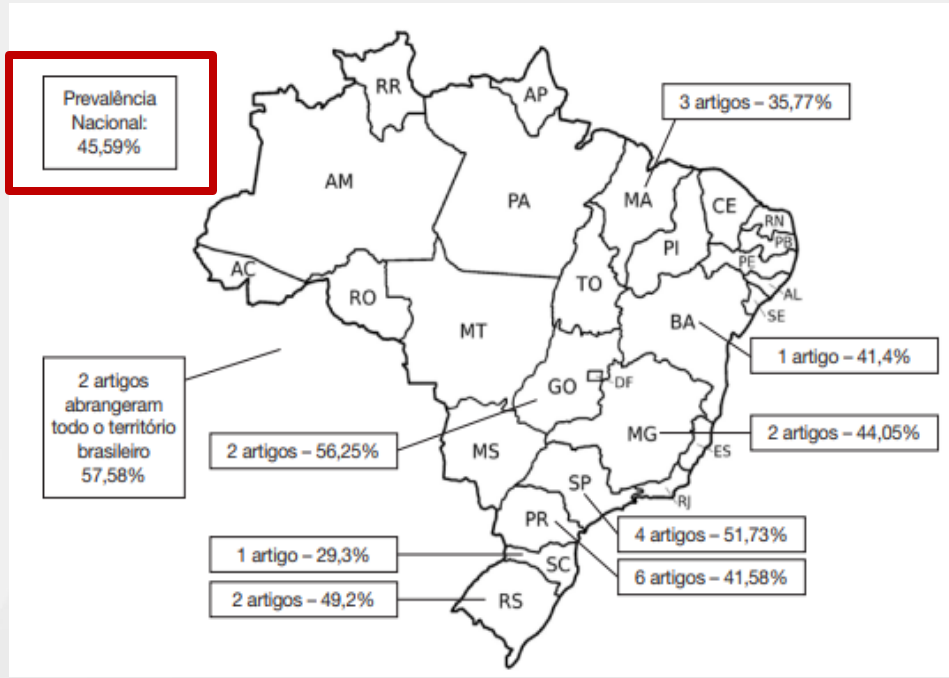
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Esses estudos tiveram como objetivo estimar a prevalência de dor crônica no Brasil, descrever e comparar as diferenças entre tipos e características da dor, identificando os tipos de terapias adotadas e o impacto da dor na vida diária dos brasileiros. A maioria das pessoas recorrem a especialistas nas áreas de ortopedia, reumatologia e neurologia. Apresentam também uma prescrição ampla de analgésicos e anti-inflamatórios para tratamento da dor. O estudo mostrou que a dor crônica no Brasil afeta 45,59% dos adultos, com predominância do sexo feminino, com predominância de possível mecanismo neurofisiológico nociceptivo e maior prevalência de acometimento em região de coluna lombar. A Sociedade Brasileira para o Estudo em Dor (SBED) propõe uma campanha nacional pelo tratamento e controle da dor aguda e crônica. O projeto brasil sem dor da SBED, visa um manejo educativo pela alta prevalência de acometimentos da doença em nosso território.



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9 – IASP define dor como uma experiência sensorial e emocional desagradável associada a dano real ou potencial em termos de tal dano. Melzack e Casey, em 1968, propuseram 3 (três) dimensões da dor, são elas:

A) Sensorial, discriminativa e interpretativa;

B) Avaliativa, discriminativa e perspectiva;

C) Afetiva-motivacional, cognitivo-avaliativa e sensório-discriminativa;

D) Sensório-discriminativa, avaliativa-interpretativa e afetivo-interpretativa.

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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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 features of pain, and ignore its motivational and affective properties, is to [illegible] part of the problem, and not even the most important part [illegible] and Livingston, 1963; Chapman, Dingman, and [illegible] theory that pain is a sensory modality is relati[illegible] theory of pain in the 19th century (Marsh[illegible] that it is an affective *quale* — the op[illegible] tion, and emphasized the unpl[illegible] organism into action. Tha[illegible] dimensions was clea[illegible] rarely, probably [illegible] is, without [illegible] amo[illegible] pain" (Sherrington, [illegible] ...sory physiology and psychophysics ...momentum to the concept of pain as a ...ved 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-

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Pain and the Neuromatrix in the Brain

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CERTA RESPOSTA!

Melzack e Casey, em 1968 relacionaram 3 dimensões ao universo da dor. A palavra “dor” é um rótulo, uma categoria, significando uma infinidade de experiências únicas e diferentes. A dor varia ao longo da vida, tanto pela dimensão sensitivo-discriminativa, quanto pela dimensões afetivo-motivacionais. A magnitude ou intensidade ao longo dessas dimensões é influenciada por atividades cognitivas. Se a lesão ou qualquer outa entrada nociva falha em evocar o impulso aversivo, a experiência não pode ser rotulada como dor. os autores, consideram que a dor é uma função de interação das 3 dimensões e não pode ser atribuída a nenhum deles isoladamente. Claramente, cada uma dessas áreas do sistema nervoso central envolvidas na experiência total da dor tem funções especializadas. Dentro desse modelo, essa “função” não reside em nenhuma área. Em vez disso, cada porção especializada do cérebro contribui para a experiência e a resposta como um todo.


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Pain and the Neuromatrix in the Brain

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10 - O componente sensório-discriminativo abrange:

A. Intensidade e qualidade da dor;

B. Intensidade e duração da dor;

C. Intensidade, qualidade, impacto e duração da dor;

D. Intensidade, localização, qualidade e duração da dor.

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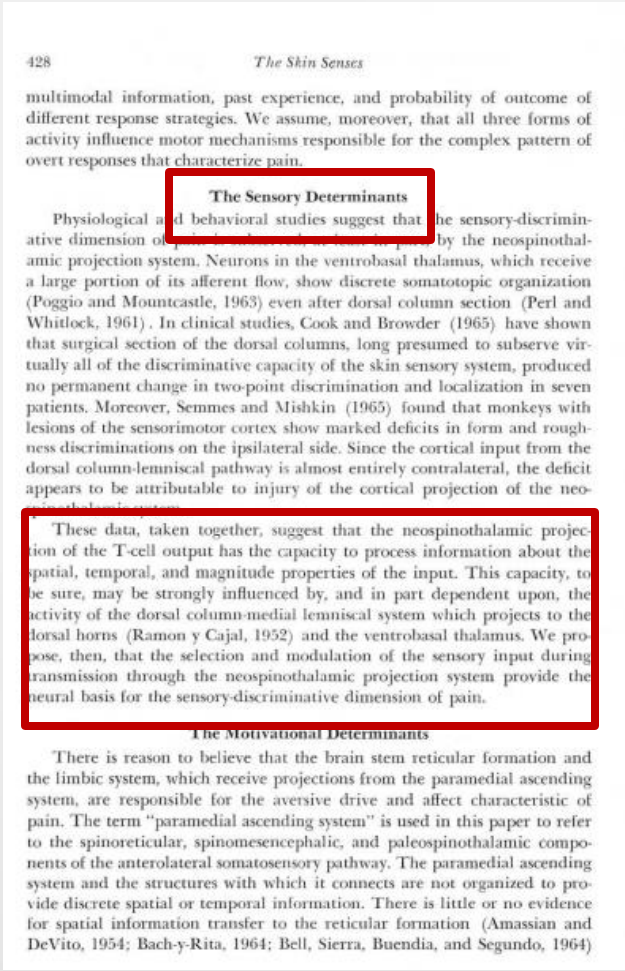
QUESTIONADOR PODCAST



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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)

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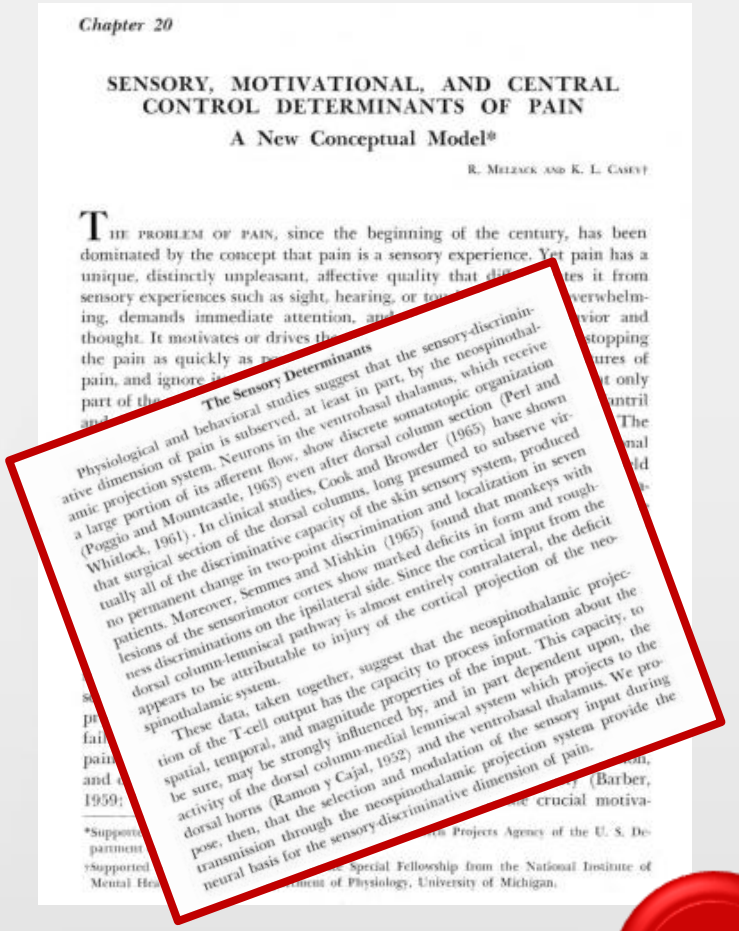
CERTA RESPOSTA!

Estudos fisiológicos e comportamentais sugerem quem a dimensão sensório-discriminativa da dor é atendida, pelo menos em parte pela projeção da via neoespinotalâmica. Neurônios no tálamo, em sua parte ventrobasal, recebem em grande parte seu fluxo aferente e apresentam discreta organização somatotópica. Os autores propõem então, que a seleção e modulação do input sensorial durante transmissão através do sistema de projeção neoespinotalâmico fornecem a base neural para a dimensão sensitivo-discriminativa da dor.



Chapter 20

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11 - O componente afetivo-motivacional abrange:

A — O sofrimento do indivíduo relativo a dor;

B — O impacto da experiência agradável, desagradável e criativa no indivíduo;

C — O impacto da relação emocional e psicológica da dor no indivíduo;

D — O impacto da relação complexa psicofísica na força de vontade no indivíduo.

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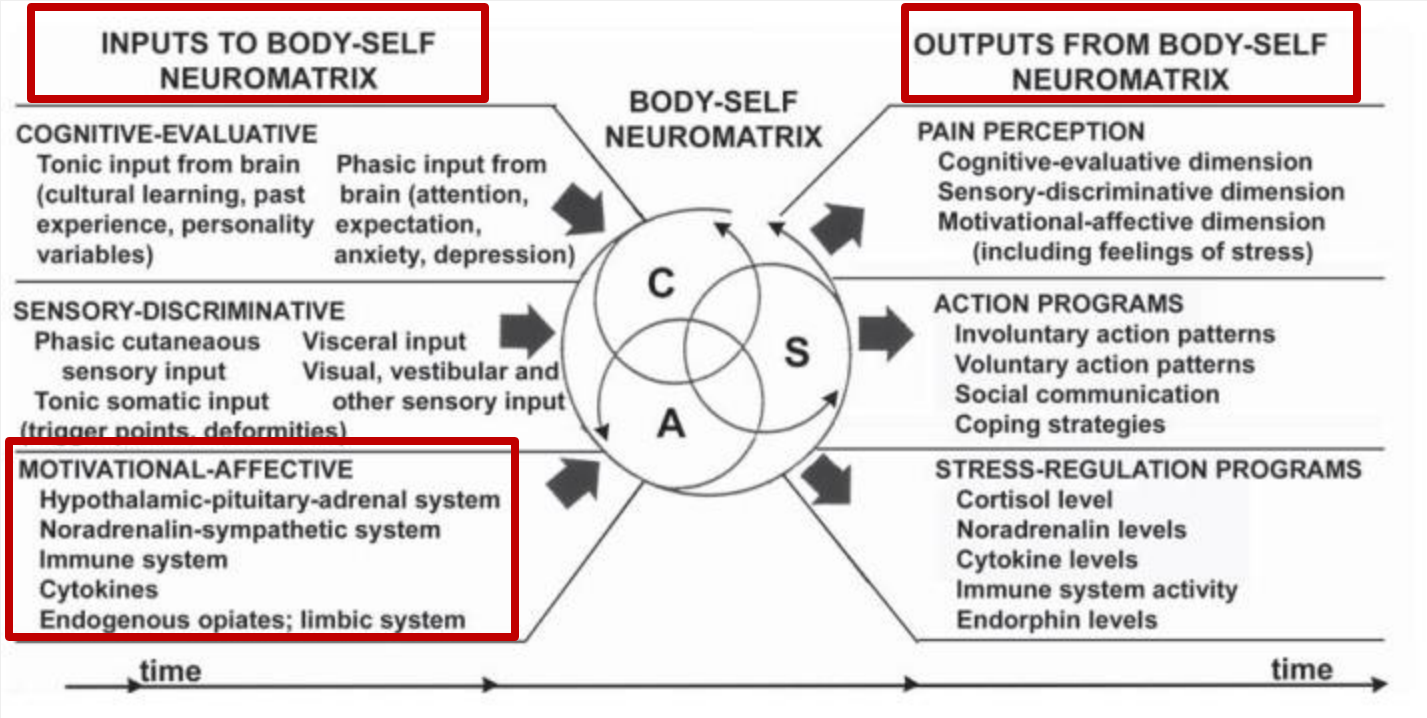
RESPOSTA ERRADA, INTERAJA COM O ESQUEMA E TENTE OUTRA VEZ!

Fatores que contribuem para padrões de atividades geradas pela neuromatriz, que compreende as áreas sensoriais, afetivos e cognitivos. Os padrões de saída da neuromatriz produzem múltiplas dimensões da experiência de dor, bem como respostas homesostáticas e comportamentais concomitantes.



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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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.

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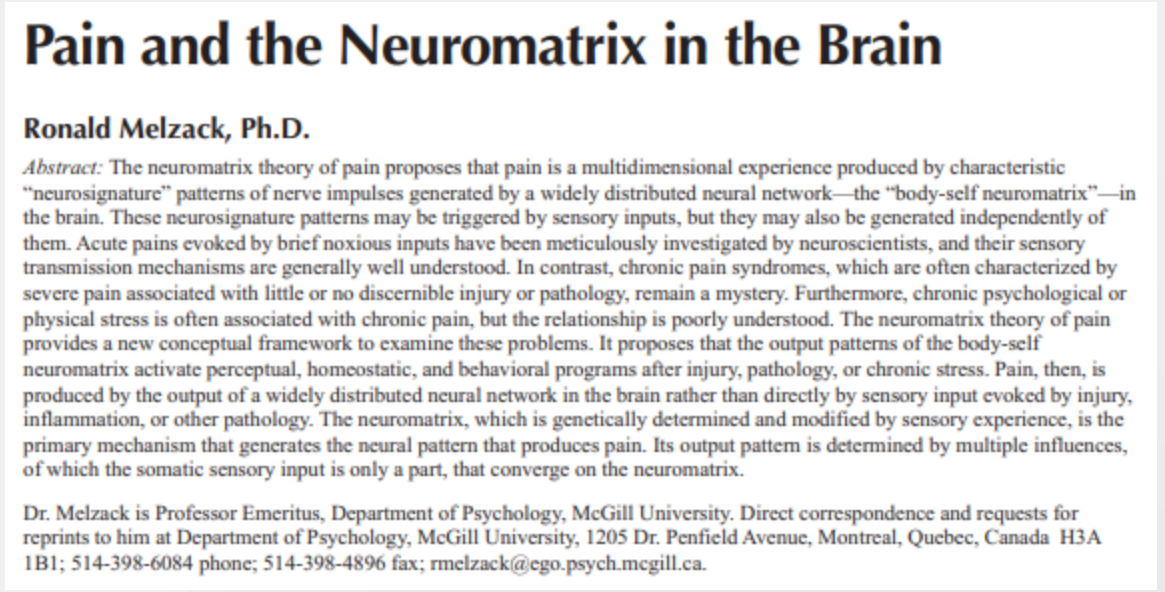
CERTA RESPOSTA!

O componente afetivo-motivacional está relacionado ao sofrimento relativo à dor. Interagem com as áreas do sistema límbico do sistema nervoso. Os múltiplos determinantes da dor incluem o sistema de regulação do estresse, com seu complexo e delicado equilíbrio, é parte integrante das múltiplas condições que origem a dor crônica. Melzack propõe ainda no estudo “Pain and the Neuromatrix in the Brain” que a expansão do campo da dor para incluir a parte neuro-hormonal, endocrinológica e imunológica, podem levar a novos caminhos no universo da avaliação e tratamento da dor.



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12 - O componente cognitivo-avaliativo abrange:

A — Contextualização da dor relacionada ao momento atual de ensino-aprendizagem da sensibilidade;

B — Contextualização da dor a partir das experiências atuais e passadas;

C — Avaliação da dor a partir de julgamento coerente do processo;

D — Avaliação da dor a partir de interpretação desproporcional ao processo.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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.

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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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QUESTIONADOR PODCAST

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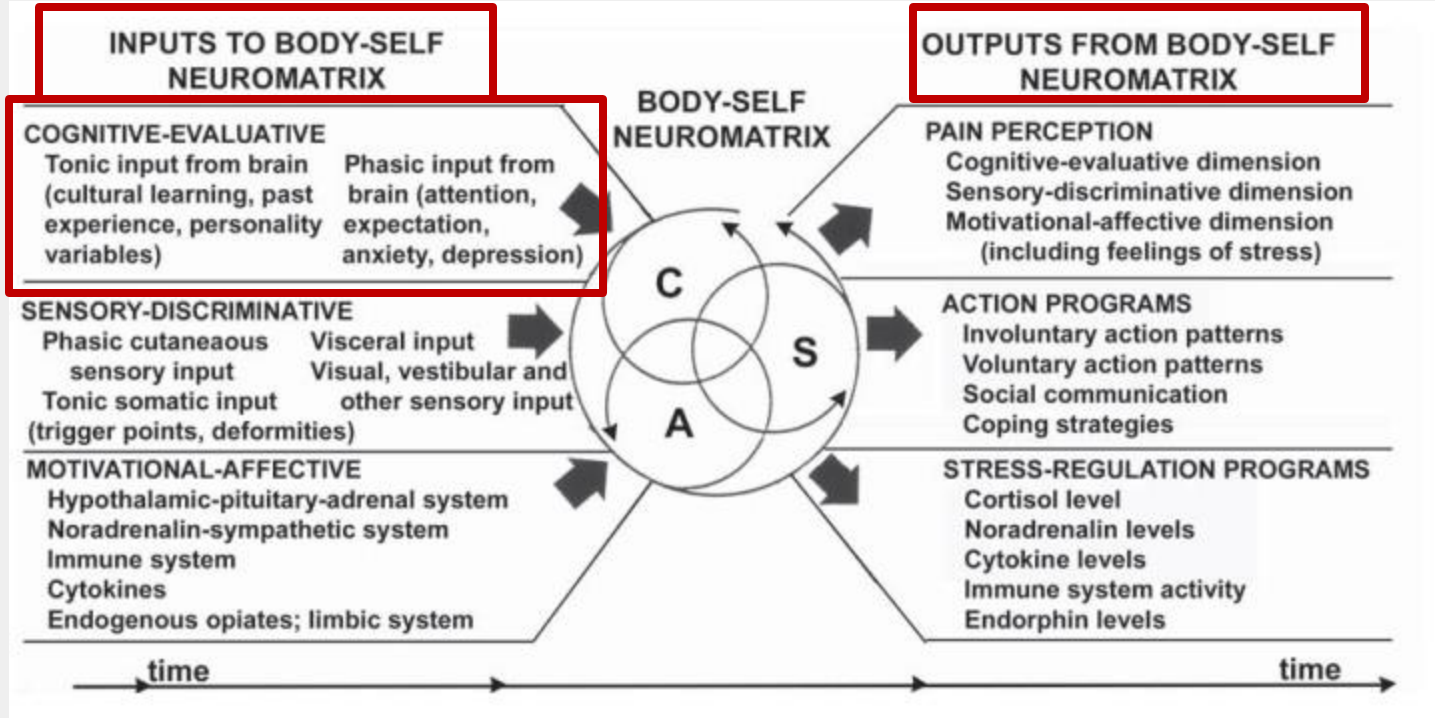
RESPOSTA ERRADA, INTERAJA COM O ESQUEMA E TENTE OUTRA VEZ!

Fatores que contribuem para padrões de atividades geradas pela neuromatriz, que compreende as áreas sensoriais, afetivos e cognitivos. Os padrões de saída da neuromatriz produzem múltiplas dimensões da experiência de dor, bem como respostas homesostáticas e comportamentais concomitantes.



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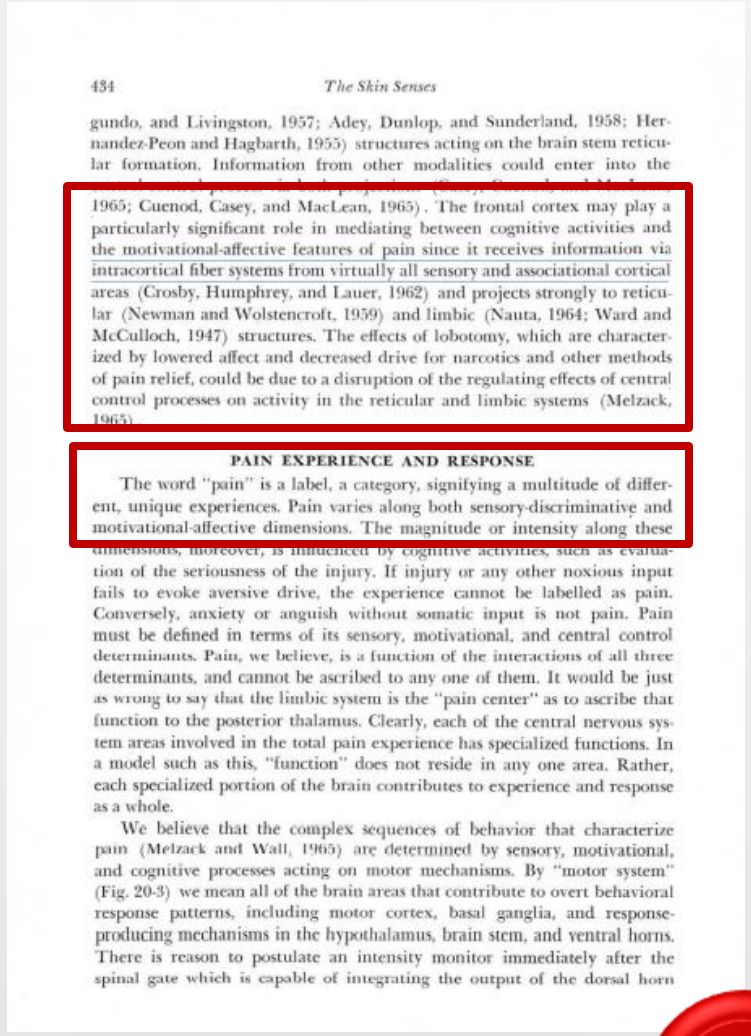
CERTA RESPOSTA!

O componente cognitivo-avaliativo contextualiza a dor a partir de experiência atuais e passadas. Portanto, relaciona-se com áreas do córtex frontal que desempenha um papel importantíssimo na mediação entre atividades cognitivas da dor, uma vez que recebe informações de fibras intracorticais de praticamente todos os sistemas sensoriais e associativos e se projeta para formação reticular e sistema límbico. Essas atividades que envolvidas no neocórtex e que atuam também na formação reticular, podem afetar tanto a experiência sensorial em suas propriedades físicas, avaliado em termos de experiência passada e presente e modificada antes de influenciar os sistemas sensoriais ou motivacionais.



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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] projections (Casey, Gernandt, and Magoun, 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

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13 - A partir da experiência de dor aguda, o indivíduo começa a aprender sobre dor. Essa experiência é aprendida por 3 processos:

A — Aprendizagem, cognição-emoções e comportamento;

B — Aprendizagem, intensidade da dor e localização da dor;

C — Aprendizagem, estado emocional e atenção focada;

D — Aprendizagem, nível intelectual indivíduo e intensidade da dor.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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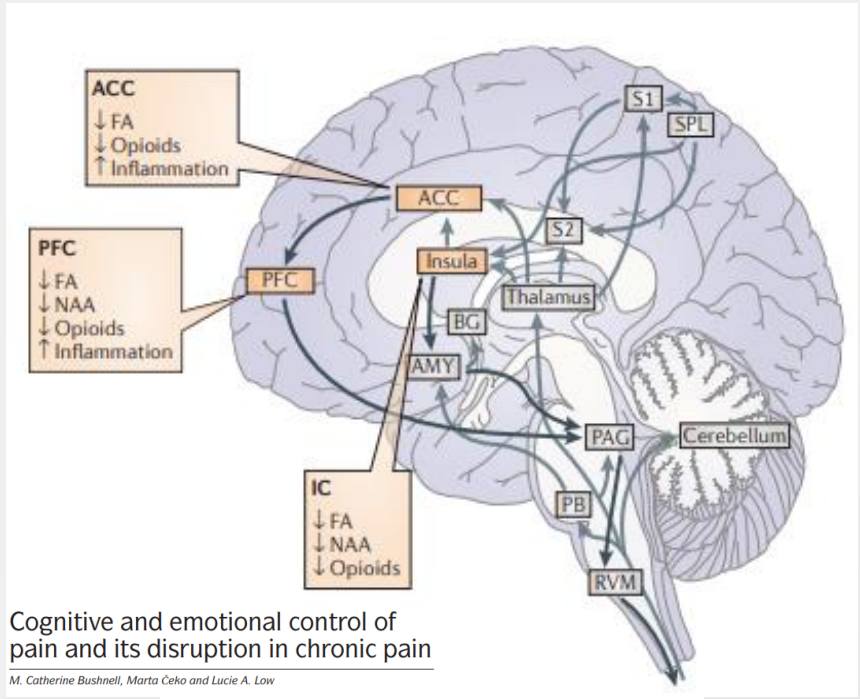

Cognitive and emotional control of pain and its disruption in chronic pain

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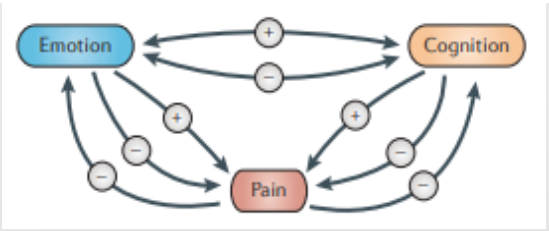

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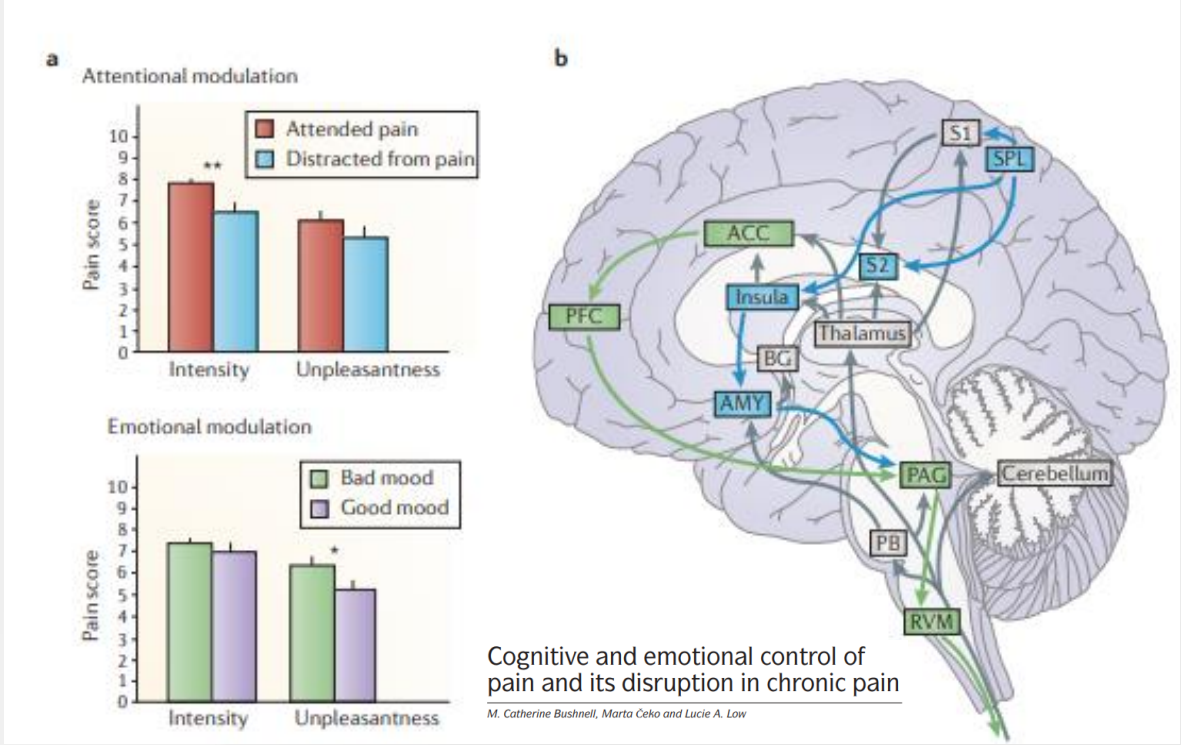
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



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CERTA RESPOSTA!

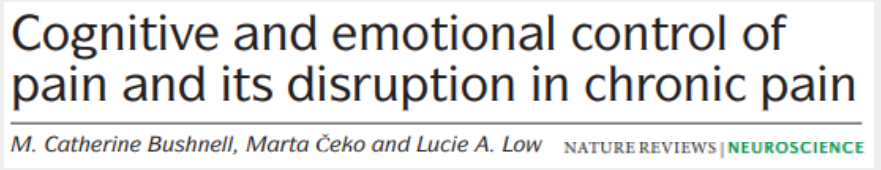
A modulação dessa experiência/sintoma de dor ocorre através de 3 (três) vias principais:

1 - aprendizagem: sensibilidade da via nociceptiva (o aprendizado não-associativo que envolve a modulação da sensibilidade da via nociceptiva, uma vez que o indivíduo entra em contato com o estímulo – processos de sensibilização e habituação; essa vida é inconsciente e implícita);

2 - cognição e emoções: medidas que tentarão ajudar o indivíduo a situações futuras potencialmente ameaçadoras (predizer eventos ou sensações ameaçadoras); e,

3 - comportamento: como a tentativa de modular sintomas pode influenciar no comportamento do indivíduo, levando a incapacidade (controlar o sintoma).

Estudos de imagem molecular mostram diminuição da ligação entre opioides endógenos e seus receptores em pacientes com dor crônica, em 3 regiões corticais que são o córtex cingulado anterior, córtex pré-frontal e a ínsula.


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

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14 - Segundo a última atualização da IASP juntamente a CID-11, a dor crônica é classificada como:

A. Uma dor aguda que não cicatrizou ao curso de 12 semanas (3 meses);

B. Uma doença com 7 classificações e subclassificações;

C. Uma dor persistente por mais de 12 semanas (3 meses);

D. Uma dor persistente por mais de 12 semanas (3 meses) sem sinais aos exames de imagem de cicatrização tecidual.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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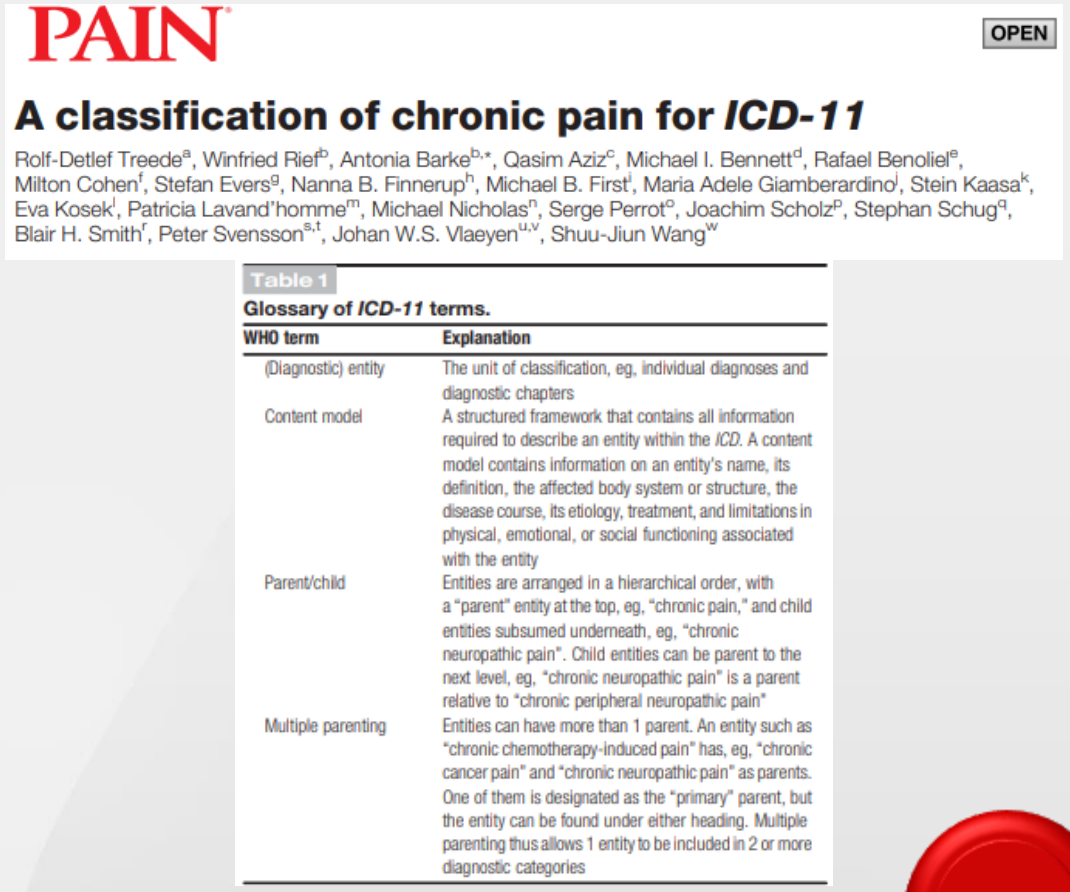
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



PAIN

OPEN

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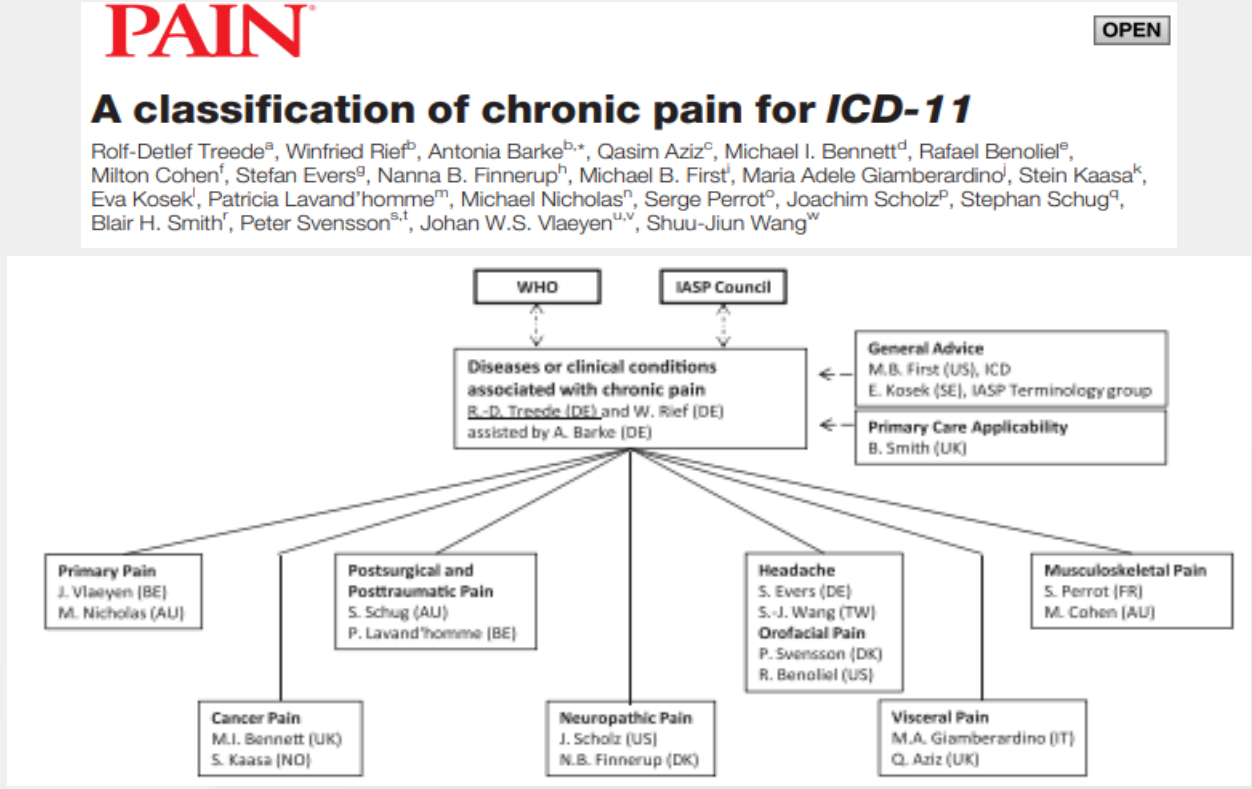
CERTA RESPOSTA!

Em 2013, a IASP formou uma força-tarefa para produzir e atualizar uma classificação de doenças dolorosas para uso internacional. Como resultado desse trabalho, a nova edição da Classificação Internacional de Doenças (CID-11) que a OMS adotou, em 2019, incluiu uma classificação de dor crônica pela primeira vez. Nos próximos anos, o CID-11 será adotado em vários países. Assim, uma definição revisada de dor é muito oportuno e alinha-se com este e outros esforços atuais para avançar estruturas ontológicas dentro das quais a dor reside. Esses esforços combinados da IASP são etapas importantes para reconhecer a dor como uma importante condição de saúde, transformando a pesquisa em dor e o cuidado de pessoas nessa condição no mundo todo.



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15 - A dor aguda acontece com como resultado direto de uma lesão potencial ou real de um tecido e é um sintoma. Seu início é bem definido e relaciona-se a uma condição conhecida. A dor crônica, por outro lado, não protege os tecidos e não possui uma função biológica clara. A dor pode ser considerada crônica se:

A) A debilidade é incompativelmente maior que os achados físicos, persiste além do tempo normal de recuperação e ocorre mesmo na ausência de lesão tecidual encontrada;

B) Persiste além do tempo normal de recuperação e o diagnóstico é conformado pelo exame de imagem;

C) A cicatrização do tecido não aconteceu cronologicamente dentro do tempo estabelecido pela IASP de dor aguda (3-6 meses);

D) Quando a lesão aguda não foi adequadamente tratada durante seu período temporal.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Narrative Review

PAIN

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]

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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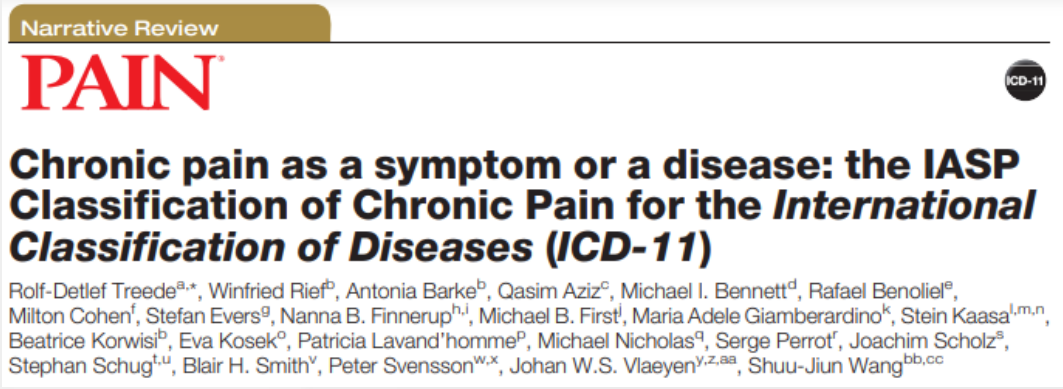

QUESTIONADOR PODCAST

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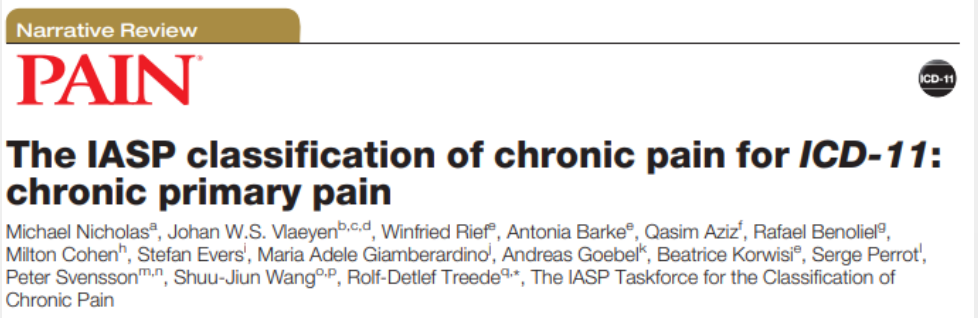
CERTA RESPOSTA!

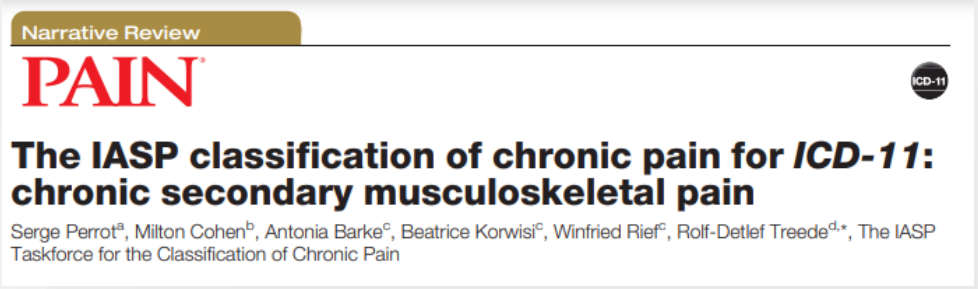
A dor crônica não possui uma função biológica clara, não é considerada um sintoma, mas a própria doença. Possui 3 subcategorias temporais e 2 associadas a origem. As temporais são: dor crônica contínua, dor crônica recorrente e dor crônica associada a flare-up. E as duas categorias são classificadas em dor crônica primária e secundária. O tratamento da dor crônica é complexo e as melhores respostas ocorrem quando a abordagem é interdisciplinar. . A implementação da dor crônica na CID-11 propõe levar melhor classificação e codificação diagnóstica, avançando assim o reconhecimento da dor crônica como uma condição de saúde prevalente.


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


Narrative Review

PAIN

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

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16 - O modelo biopsiciossocial proposto por Engle e amplamente adotado no mundo quando falamos em neurofisiologia da dor, descreve que a iniciação, manutenção e percepção da dor é influenciada por fatores biológicos, psicossociais e fatores do sistema de movimento. Quando falamos de abordagem atualizada sobre a dor, a ideia de se utilizar os mecanismos neurofisiológicos apresenta maiores probabilidade de auxílio ao paciente comparada a atuação pautada em sinais e sintomas, segundo Cliford Wolff e Mitchell Max. Quais são os mecanismos neurofisiológicos propostos atualmente pela IASP?

A Sensibilização central, sensibilização periférica e facilitação medular;'

B Nociceptivo, neuropático e sensibilização central;

C Nociplástico, neuropático e sensibilização periférica;

D Neuropático, nociceptivo e nociplástico.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

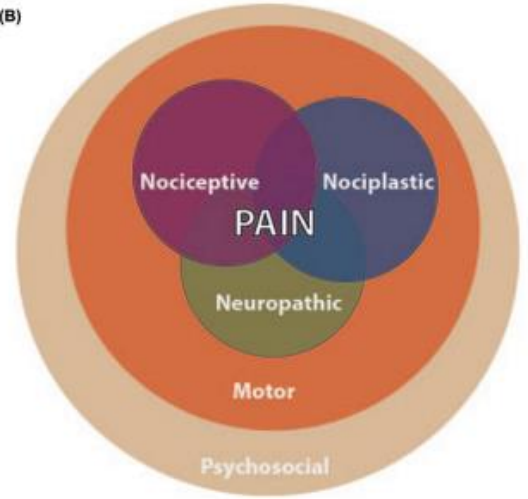
A Mechanism-Based Approach to Physical Therapist Management of Pain

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)



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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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***

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Contents lists available at ScienceDirect

Musculoskeletal Science and Practice

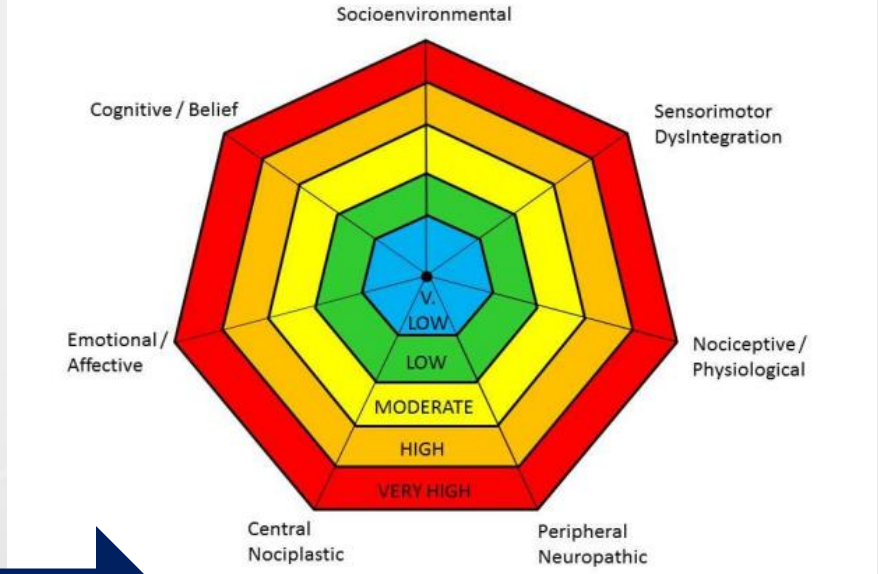
journal homepage: www.elsevier.com/locate/msksp

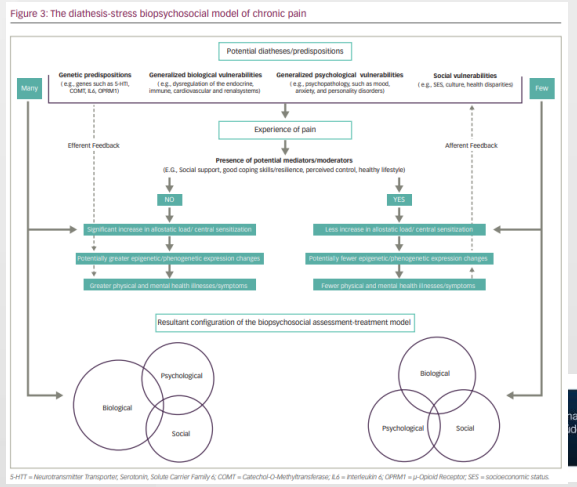
Original article

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





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CERTA RESPOSTA!

Os mecanismos neurofisiológicos apresentam uma probabilidade de maior auxílio ao paciente quando comparado ao modelo de sinais e sintomas apenas. Eles foram atualizados em 2016 pela IASP, sendo o último mecanismo a compor o quadro descrito foi o mecanismo nociplástico. O objeto dessa revisão em 2016, foi propor um debate através de um terceiro mecanismo destinado a caracterizar a dor crônica através de uma função nociceptiva alterada. Eles são categorizados em 3 classes. São eles: nociceptivo: dor devido a um dano real ou potencial ao tecido não neural proveniente da ativação de nociceptores; nociplástico: dor em decorrência de alterações na nocicepção apesar de ausência de evidências de dano tecidual real ou potencial, causando a ativação de nociceptores periféricos ou de evidências de doença ou lesão do sistema somatossensorial causando a dor; e neuropático: dor causada por uma lesão ou doença do sistema somatossensorial.

A Mechanism-Based Approach to Physical Therapist Management of Pain

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

Review Pain

The Biopsychosocial Model of the Assessment, Prevention, and Treatment of Chronic Pain

Kelley Bevers,[1] Lynette Watts,[1] Nancy D Kishino,[2] Robert J Gatchel[1]

1. The University of Texas at Arlington, Texas, US; 2. West Coast Spine Restoration Center, Riverside, California, US

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]

Pain Mechanisms: A New Theory

A gate control system modulates sensory input from the skin before it evokes pain perception and response.

Ronald Melzack and Patrick D. Wall

19 November 1965, Volume 150, Number 3699 SCIENCE

PAIN

Four decades later: what's new, what's not in our understanding of pain

Judith A. Turner[a], Lars Arendt-Nielsen[b]

September 2020 • Volume 161 • Number 9

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17 - O mecanismo nociceptivo é composto por 3 (três) subclassificações, são elas:

A. Química, química não inflamatória e manutenção tissular;

B. Química, térmica e mecânica;

C. Mecânica, química e motora;

D. Química, central e periférica.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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.

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CERTA RESPOSTA!

É uma dor devido a dano real ou potencial ao tecido não neural, proveniente da ativação de nociceptores; existindo uma série de drivers (fontes geradoras) da dor predominantemente nociceptiva. Possui 3 (três) subclassificações descritas pela IASP atualmente, são elas: química, química não inflamatória e manutenção tissular.

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####

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18 - O mecanismo neuropático é composto por 2 (duas) subclassificações, são elas:

A — Localizada e difusa;

B — Difusa e generalizada;

C — Central e periférica;

D — Periférica e somática.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

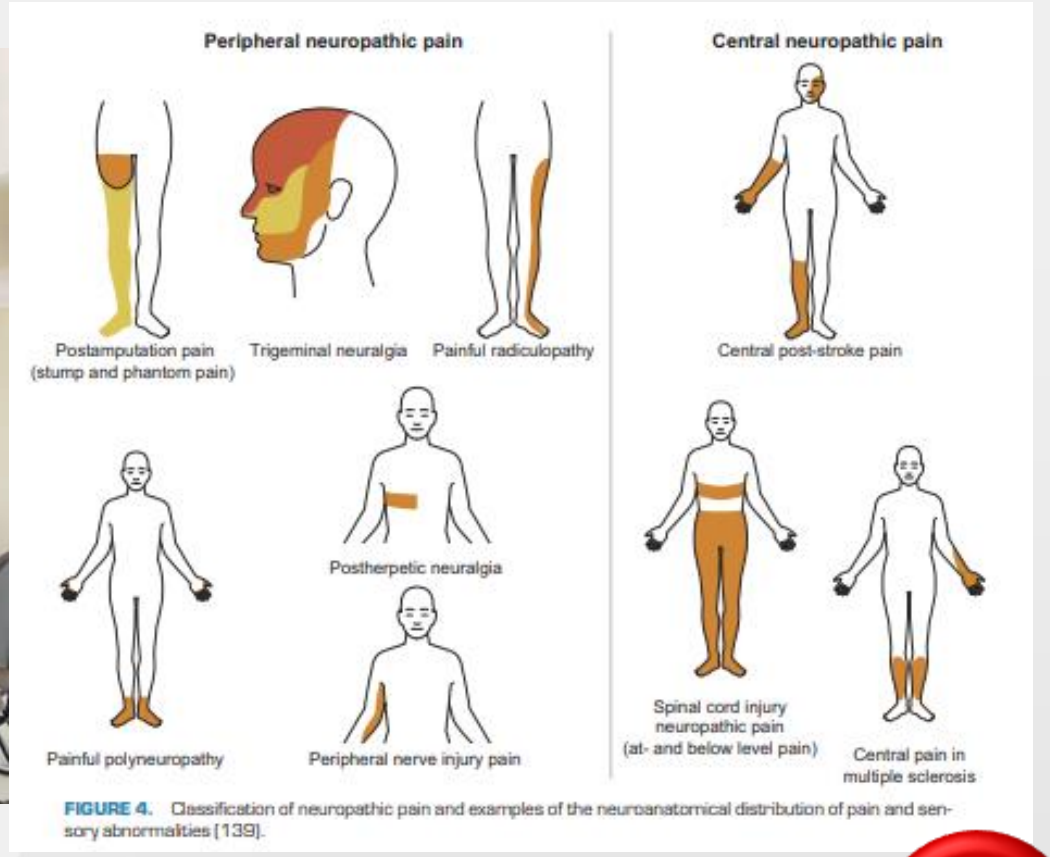

FIGURE 4. Classification of neuropathic pain and examples of the neuroanatomical distribution of pain and sensory abnormalities [139].

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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST

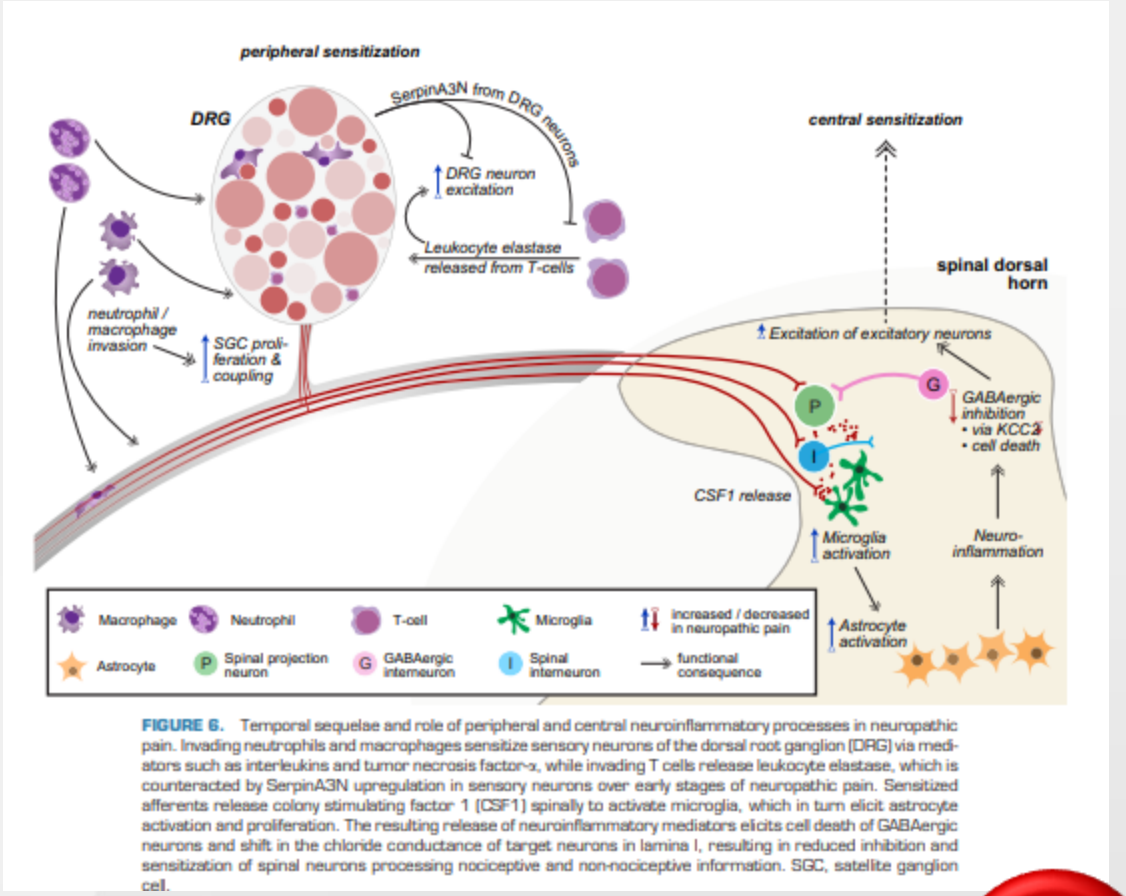

FIGURE 6. Temporal sequelae and role of peripheral and central neuroinflammatory processes in neuropathic pain. Invading neutrophils and macrophages sensitize sensory neurons of the dorsal root ganglion (DRG) via mediators such as interleukins and tumor necrosis factor-α, while invading T cells release 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.

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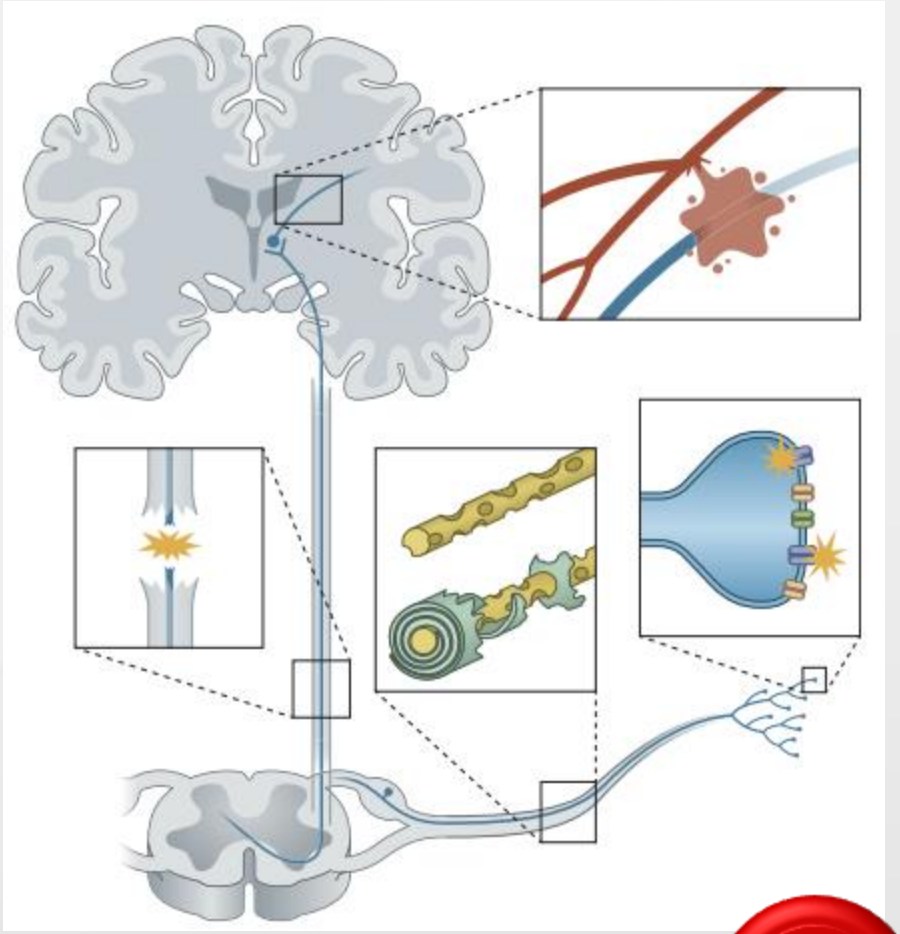
CERTA RESPOSTA!

Dor causada por uma lesão ou doença do sistema nervoso somatossensorial; mesmo que a condição de base tenha sido tratada ou curada. A dor neuropática pode ser espontânea ou evocada, como resposta aumentada a um estímulo doloroso. O diagnóstico requer um histórico de lesão ou doença do sistema nervoso e uma distribuição neuro anatomicamente plausível da dor; os sintomas e sinais negativos como diminuição ou perda da sensibilidade, e positivos como hiperalgesia e alodinia, indicando o envolvimento do sistema nervoso somatossensorial devem ser compatíveis com o território de inervação da estrutura nervosa afetada.



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19 - O mecanismo nociplástico é constituído por 2 (duas) subclassificações, são elas:

A. Verdadeira e aumentada;

B. Verdadeira e falsa;

C. Intensa e desproporcional;

D. Real e imaginária.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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***

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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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 "nocioceptive–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.

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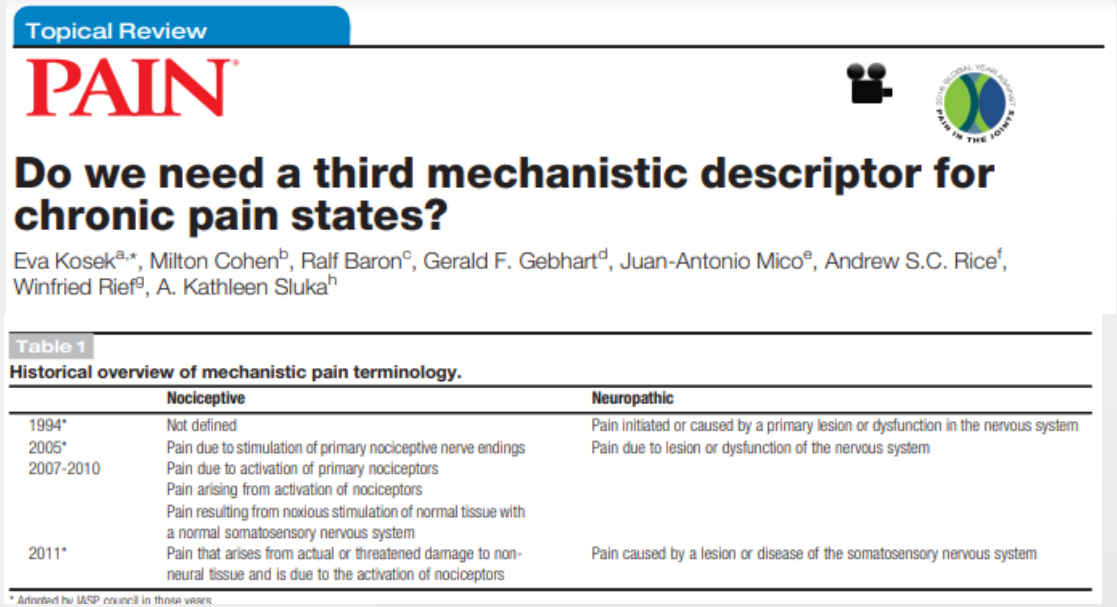
CERTA RESPOSTA!

Refere-se a uma categoria de base fisiológica, que é particularmente aplicável a condições de dor primária crônica, descrita na CID-11 em 2019 junto a OMS, ou seja, é a dor que surge do processamento anormal de dor sem qualquer evidência clara de dano ao tecido ou patológica discreta, envolvendo o sistema somatossensorial; os procedimentos intervencionistas estão relacionados a resultados piores em indivíduos com dor nociplástica quando comparados a indivíduos com dor nociceptiva.



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

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20 - A dor crônica pode ser dividida em primária e secundária. A dor crônica primária é definida como dor em uma ou mais regiões anatômicas que persiste ou se repete por mais de três meses e está associada a sofrimento emocional significativo ou incapacidade funcional (interferência nas atividades de vida diária e participação em papéis sociais) e que não pode ser mais bem explicada por outra condição de dor crônica. Elas são sub-classificadas em:

A) dor crônica generalizada; síndrome de dor complexa regional tipo 1; dor crônica primária orofacial ou cabeça; dor crônica primária visceral e dor crônica primária musculoesquelética;

B) dor crônica esporádica; dor visceral; dor orofacial ou cabeça e dor musculoesquelética;

C) dor crônica difusa; dor generalizada e dor somática;

D) dor crônica musculoesquelética; dor crônica visceral; dor crônica orofacial ou cabeça e dor psicossomática.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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]

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

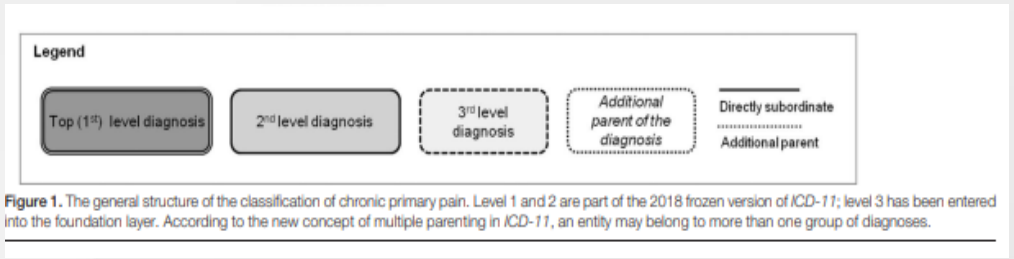

Figure 1. The general structure of the classification of chronic primary pain. Level 1 and 2 are part of the 2018 frozen version of *ICD-11*; level 3 has been entered into the foundation layer. According to the new concept of multiple parenting in *ICD-11*, an entity may belong to more than one group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

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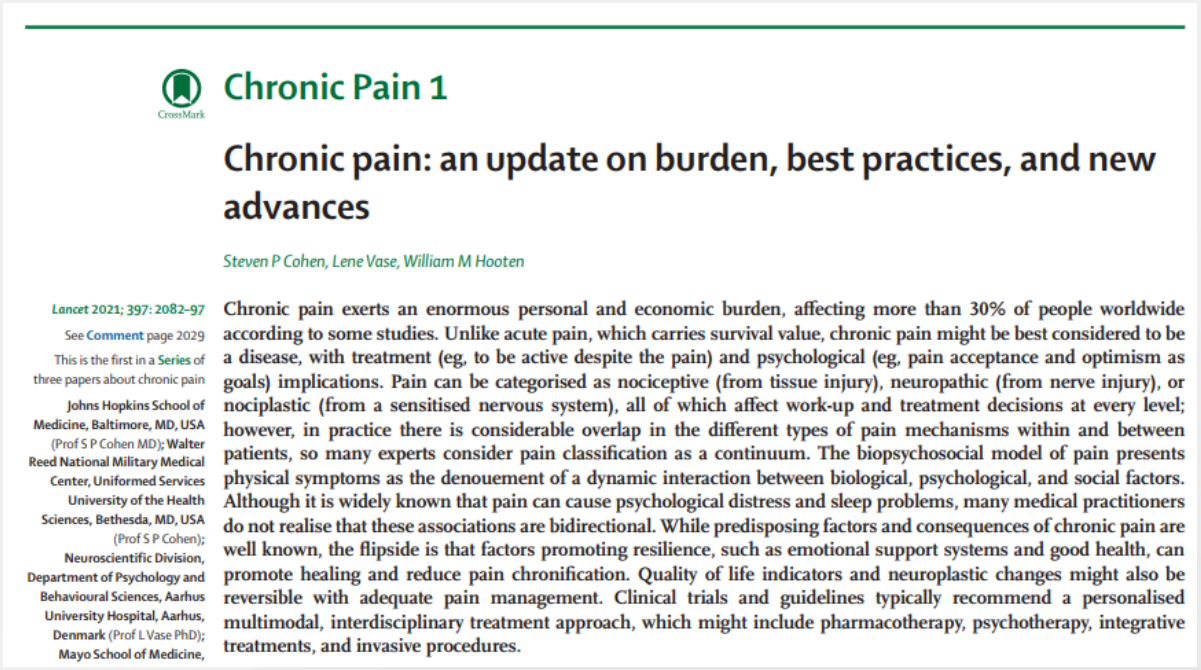
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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.

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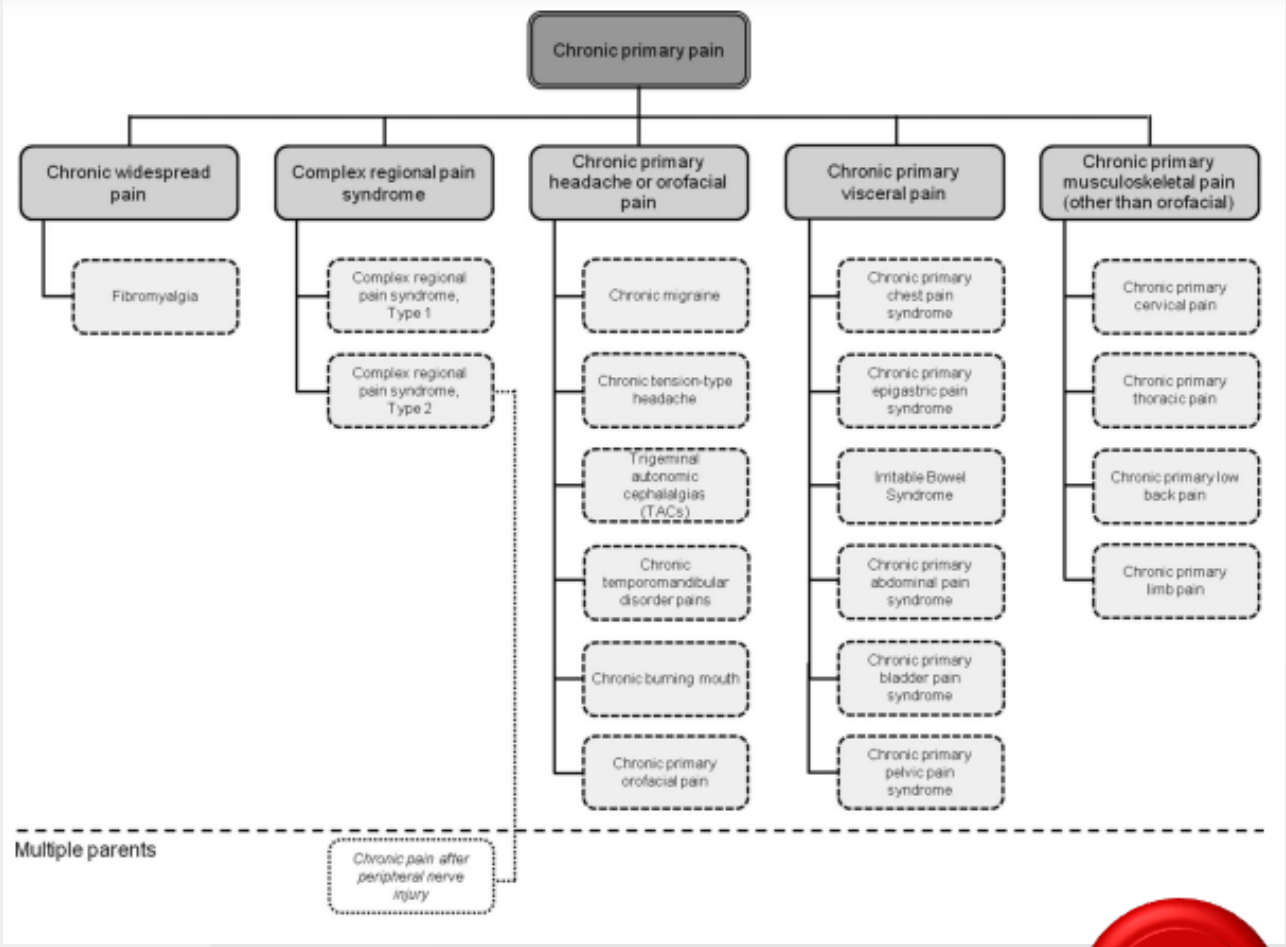
CERTA RESPOSTA!

A dor crônica primária é a doença propriamente dita. Seu mecanismo neurofisiológico predominante é o nociplástico. Normalmente se observa quando não existe história pregressa do paciente, ou seja, não há um evento detectável ou uma alteração estrutural que justifique sua dor.



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21 - De acordo a inclusão da dor crônica na CID-11 e segundo atualização da IASP, ela está associada a um aumento da excitabilidade do sistema nervoso e uma diminuição de seu controle inibitório nocivo difuso. São fatores de risco que aumentam o risco de desenvolver dor crônica:

A) Dificuldades de sono, fadiga, alterações psicológicas e sedentarismo;

B) Alterações psicológicas, fator genético, envelhecimento e desidratação;

C) Sedentarismo, cirurgia prévia, marcador biológico ativo e dieta restritiva de carboidratos;

D) Dor aguda não tratada corretamente, marcador biológico ativo, sedentarismo e fibromialgia.

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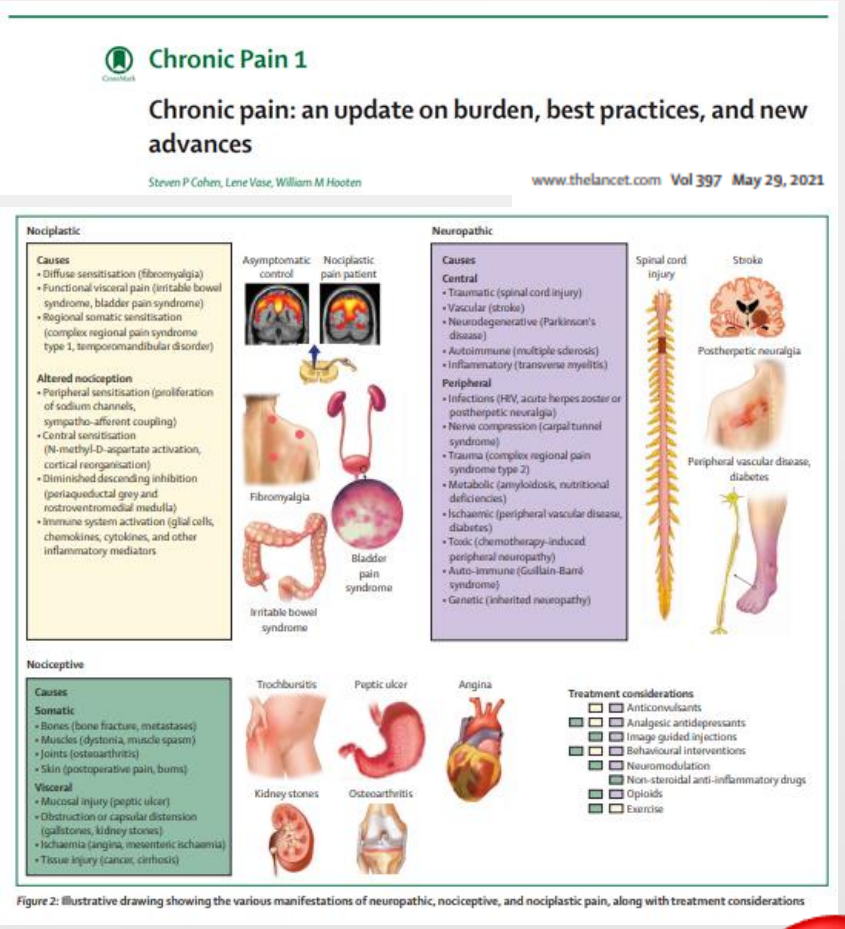

Chronic Pain 1

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

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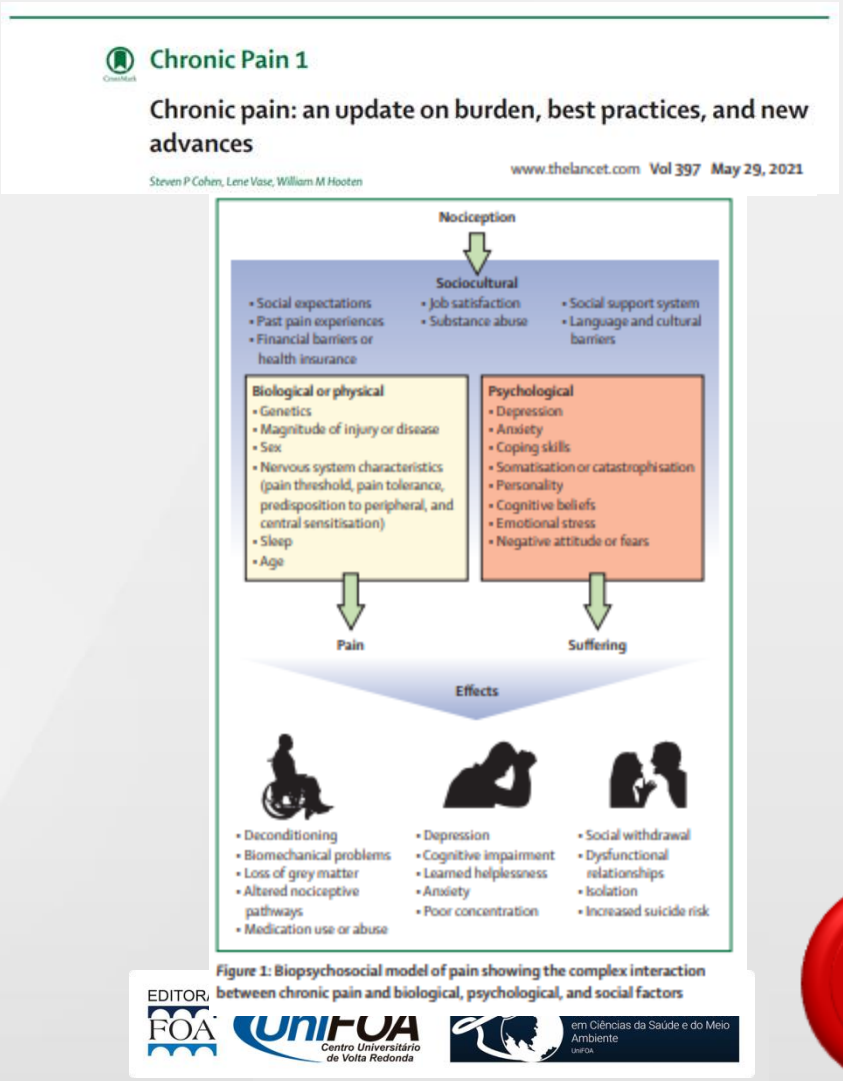
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



Chronic Pain 1

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

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Steven P Cohen, Lene Vase, William M Hooten

Nociception

Sociocultural

- Social expectations
- Past pain experiences
- Financial barriers or health insurance
- Job satisfaction
- Substance abuse
- Social support system
- Language and cultural barriers

Biological or physical

- Genetics
- Magnitude of injury or disease
- Sex
- Nervous system characteristics (pain threshold, pain tolerance, predisposition to peripheral, and central sensitisation)
- Sleep
- Age

Psychological

- Depression
- Anxiety
- Coping skills
- Somatisation or catastrophisation
- Personality
- Cognitive beliefs
- Emotional stress
- Negative attitude or fears

Pain

Suffering

Effects

- Deconditioning
- Biomechanical problems
- Loss of grey matter
- Altered nociceptive pathways
- Medication use or abuse
- Depression
- Cognitive impairment
- Learned helplessness
- Anxiety
- Poor concentration
- Social withdrawal
- Dysfunctional relationships
- Isolation
- Increased suicide risk

Figure 1: Biopsychosocial model of pain showing the complex interaction between chronic pain and biological, psychological, and social factors

EDITOR FOA UniFOA Centro Universitário de Volta Redonda em Ciências da Saúde e do Meio Ambiente UniFOA

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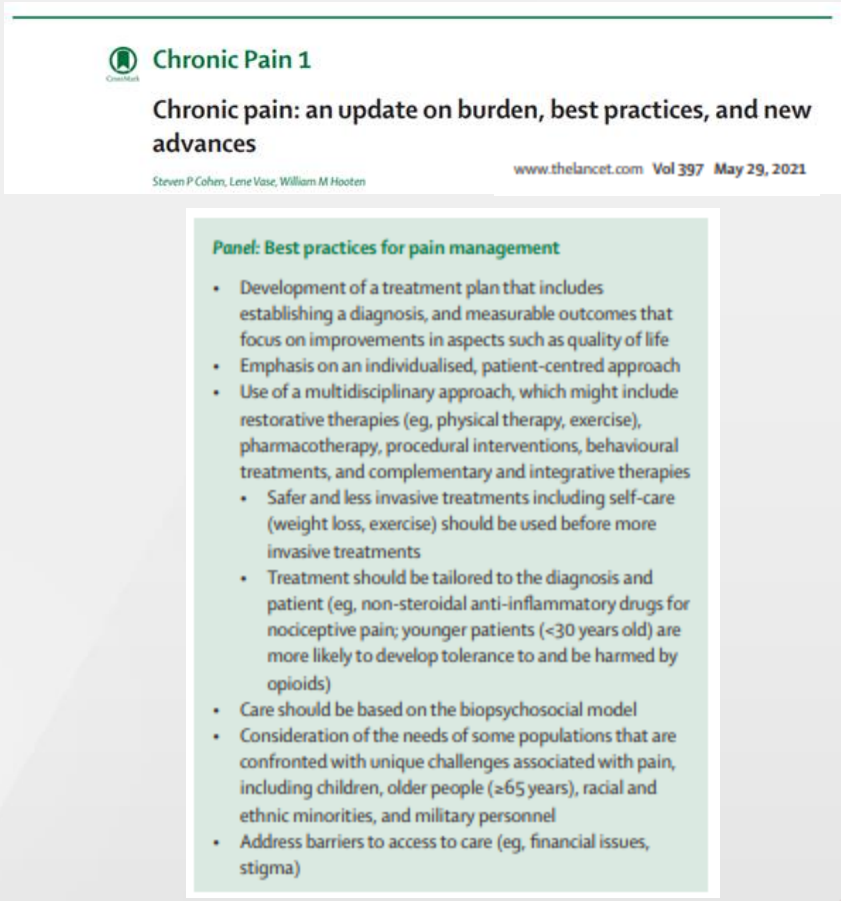
QUESTIONADOR PODCAST



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)

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CERTA RESPOSTA!

O modelo biopsicossocial postula dor e incapacidade como interações multidimensionais e dinâmicas entre fatores biológicos, psicológicos e sociais que reciprocamente influenciam uns aos outros. Características como depressão, ansiedade, sono e condições sociais adversas podem ser o resultado de dor crônica. É menos conhecido que esses fatores predispõem os indivíduos à dor crônica.

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.

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22 - No capítulo "Neurobiologia da Dor e Analgesia", escrito pela renomada pesquisadora Kathleen A. Sluka, no livro "Eletroterapia Aplicada a Reabilitação – Dos Fundamentos às Evidências" (Richard E. Liebano, 2021), o sistema nervoso central equilibra a atividade excitatória ou inibitória por meio de estrutura e vias nociceptivas. As metas do tratamento são reequilibrar a atividade das vias envolvidas na nocicepção e dor, portanto:

A) Aumentando o estímulo da via direta motora e inibindo o estímulo da via indireta somestésica;

B) Estimulando a via sensitiva e via motora (trajeto sensório-motor);

C) Reduzindo a atividade das vias excitatórias (facilitatórias) e aumentando a atividade das estruturas inibitórias;

D) Estimulando a atividade das vias excitatórias (facilitatórias) e reduzindo a atividade das estruturas inibitórias.

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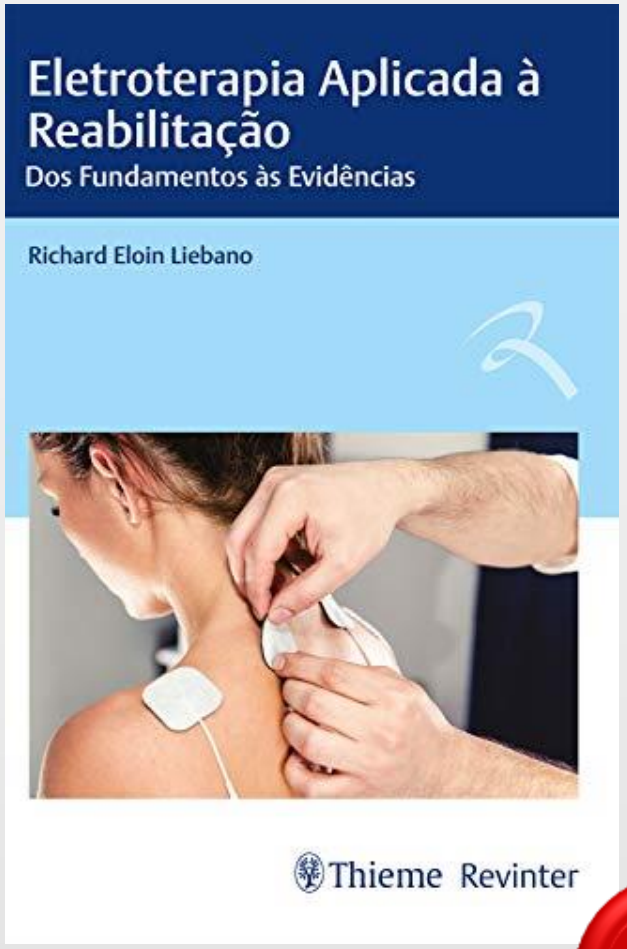
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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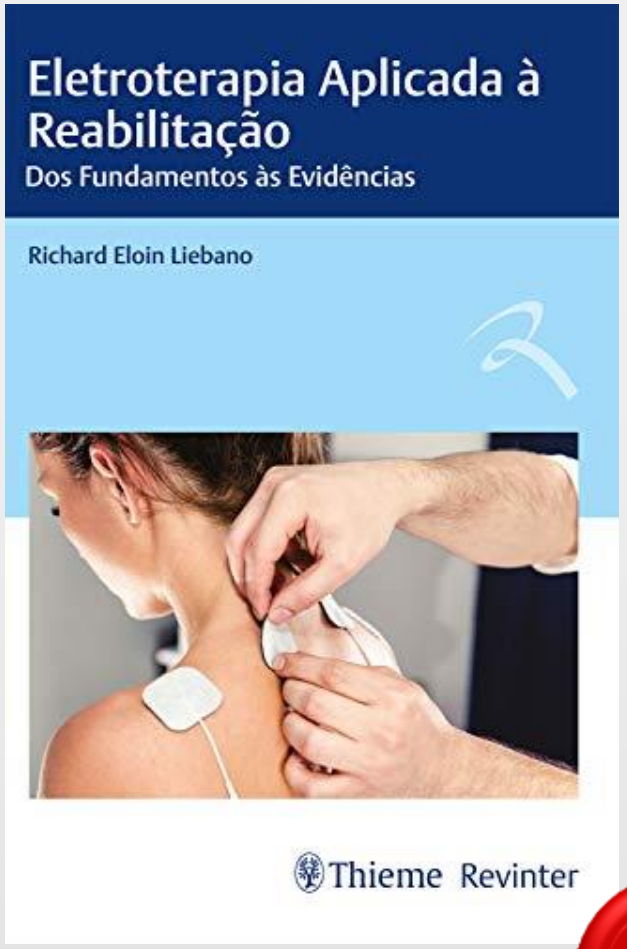

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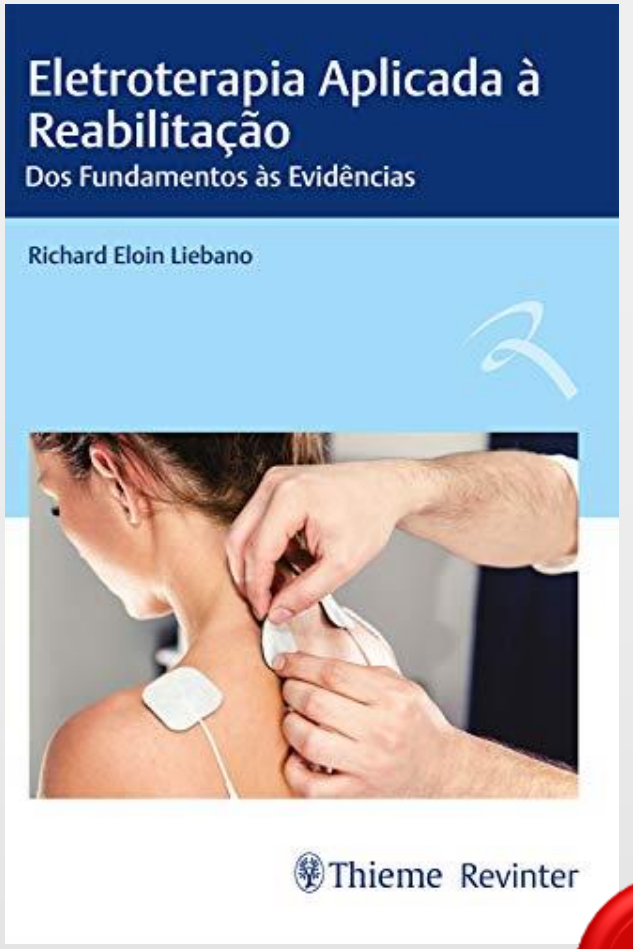
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



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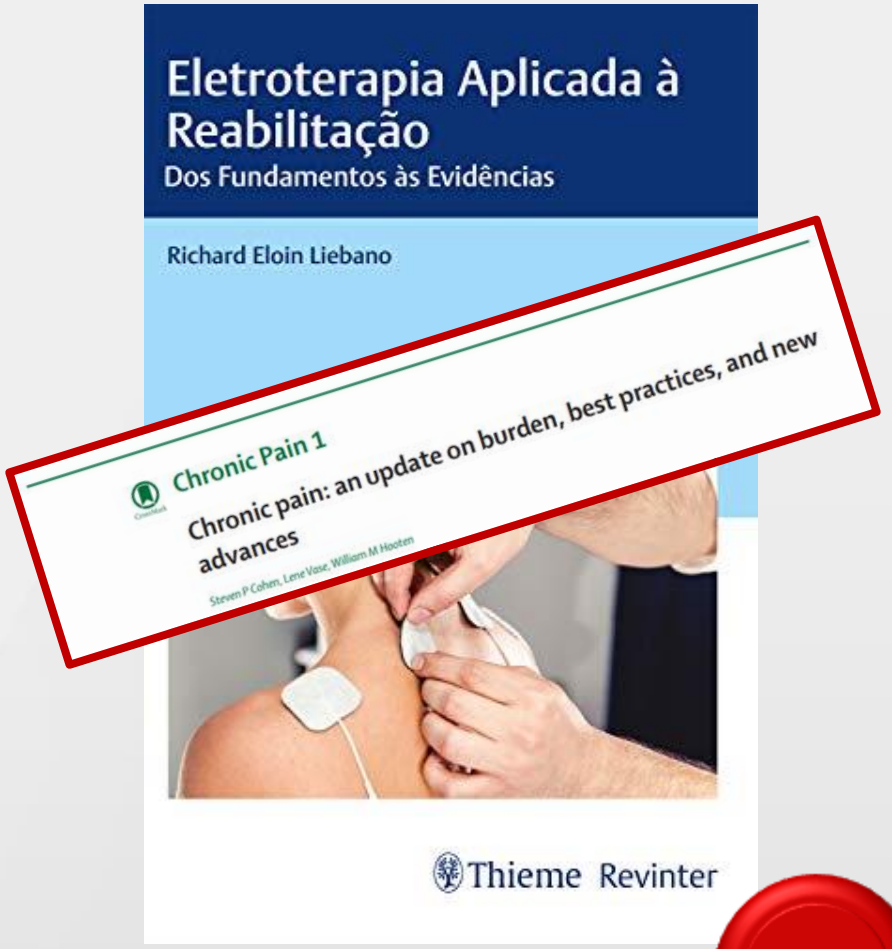
CERTA RESPOSTA!

Independentemente do local da dor (região do corpo onde dói), de qual sistema acometido (somático, visceral ou neural), do tecido envolvido (nervos, músculos, cartilagem, tecido visceral) até mesmo os estímulos passivos de não serem de detectados e os passivos de detecção. Esses mecanismos são filtrados por 2 (duas) espécies de controle ou caminhos. Um caminho descendente (top-down) e um caminho ascendente (bottow-up). Porém independente do sistema ou caminho são em sua totalidade influenciados e modulados por fatores endógenos, mas também por fatores exógenos (aspectos psicológicos e sociais da dor). Porém a repercussão ao final é bioquímica e neurofisiológica, sendo a nível molecular e de difícil diagnóstico.



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23 - São questionários e escalas utilizados na avaliação do paciente com dor:

A. Escala Tampa de Cinesiofobia, Inventário de Sensibilização Central (CSI), Escala de Pensamentos Catrastóficos, Pain Detect, Douleur Neuropathique 4 Questions;

B. Critério de Ottawa, WOMAC, IKDC;

C. CADE-Q SV, ELSA, DASI;

D. Inventário de Sensibilização Central (CSI), Pain Detect, DASI, WOMAC.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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:

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- 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 relacionada ao câncer. Rev Brasileira de Oncologia Clínica 2014 Outubro/Novembro/Dezembro; Vol. 10 (38): 132 – 14

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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

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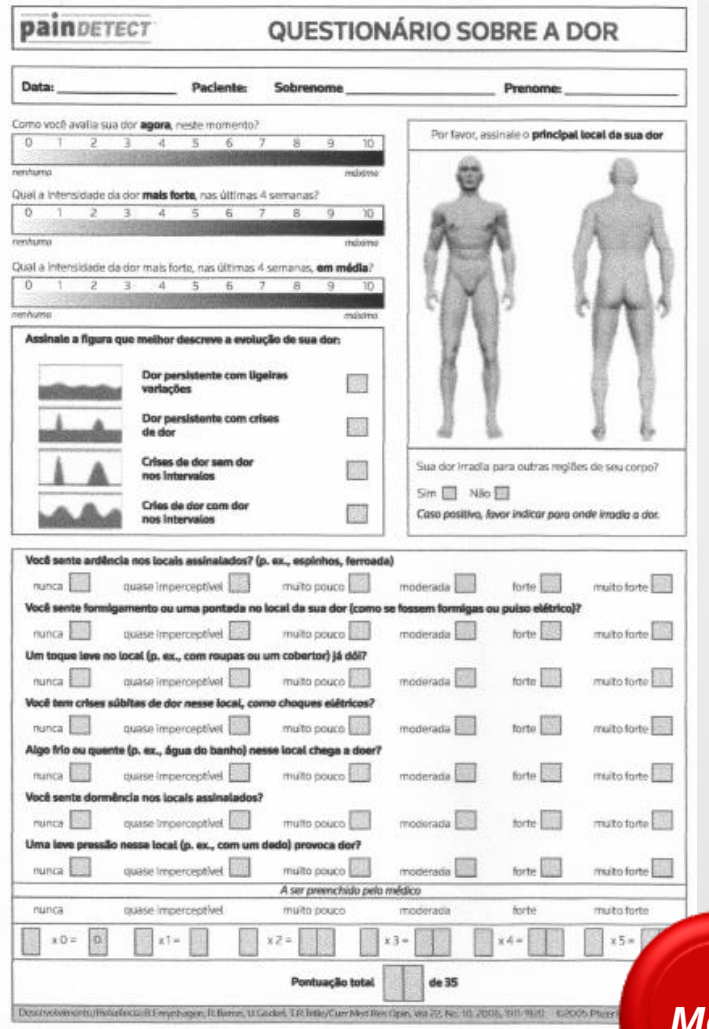
Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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/Validação: R. Freynhagen, R. Baron, U. Gockel, T.R. Tölle/Curr Med Res Opin, Vol 22, No. 10, 2006, 1911–1920 ©2005 Pfizer

retornar

Menu

CERTA RESPOSTA!

A dor é uma condição subjetiva, portanto, difícil de mensurar. Os numerosos questionários sobre dor são classificados e orientados para permitir uma avaliação de forma mais objetiva e reprodutível dos múltiplos fatores que estão associados a dor. Os questionários são preenchidos pelos próprios pacientes e revelam o nível e a qualidade da dor, fatores como depressão, ansiedade, medo, insegurança, incapacidade e catastrofismo.

ARTIGO ORIGINAL

http://dx.doi.org/10.1590/1984-0462/;2018;36;4;00014

TRADUÇÃO, ADAPTAÇÃO TRANSCULTURAL E AVALIAÇÃO PRELIMINAR DA *PAIN CATASTROPHIZING SCALE-PARENTS* PARA USO NO BRASIL

Translation, cross-cultural adaptation and preliminary evaluation of the Brazilian version of the pain catastrophizing scale-parents

Julianna Amaral Cavalcante[a], Karolline Alves Viana[b], Paulo Sucasas Costa[b,*], Luciane Rezende Costa[b]

Pain Medicine

Pain Medicine 2012; 13: 1425–1435
Wiley Periodicals, Inc.

Original Research Article

Cross-Cultural Adaptation and Validation of the Brazilian Portuguese Version of the Pain Catastrophizing Scale

QUESTIONÁRIO PARA DIAGNÓSTICO DE DOR NEUROPÁTICA DN4

painDETECT QUESTIONÁRIO SOBRE A DOR

DOR & NEUROMODULAÇÃO – ISCPA/CNPq

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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;

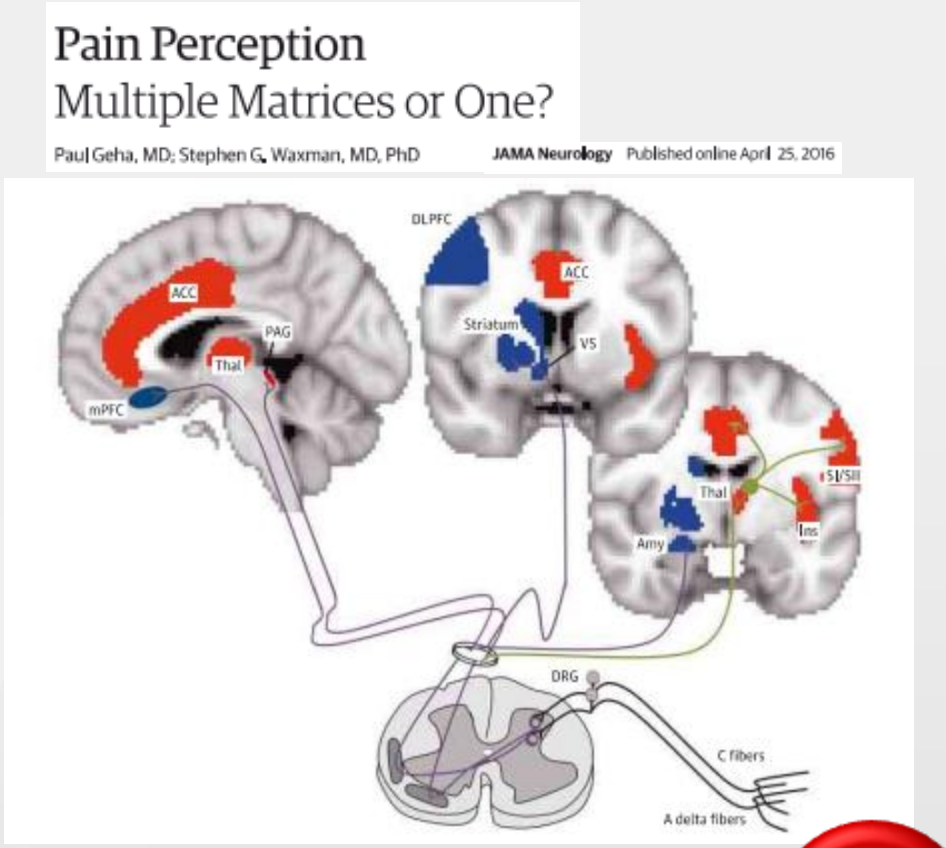
B) Massa neuronal, precisão neural e neuroplasticidade;

C) Neuroplasticidade, persistência e precisão neural;

D) Neuroplasticidade, cognição e neuroplasticidade.

Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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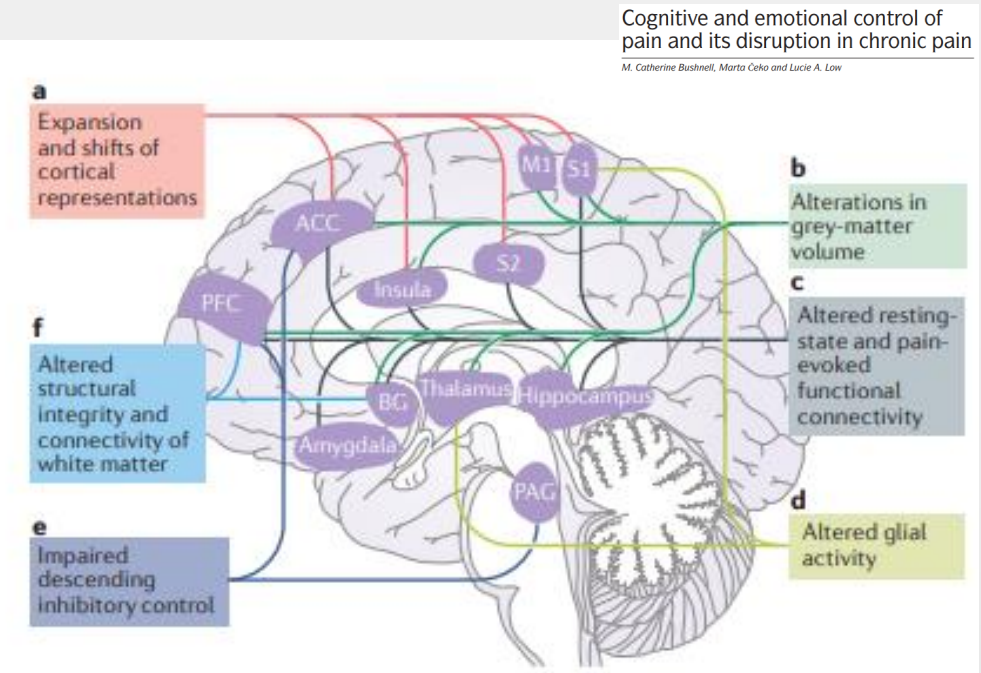

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Menu

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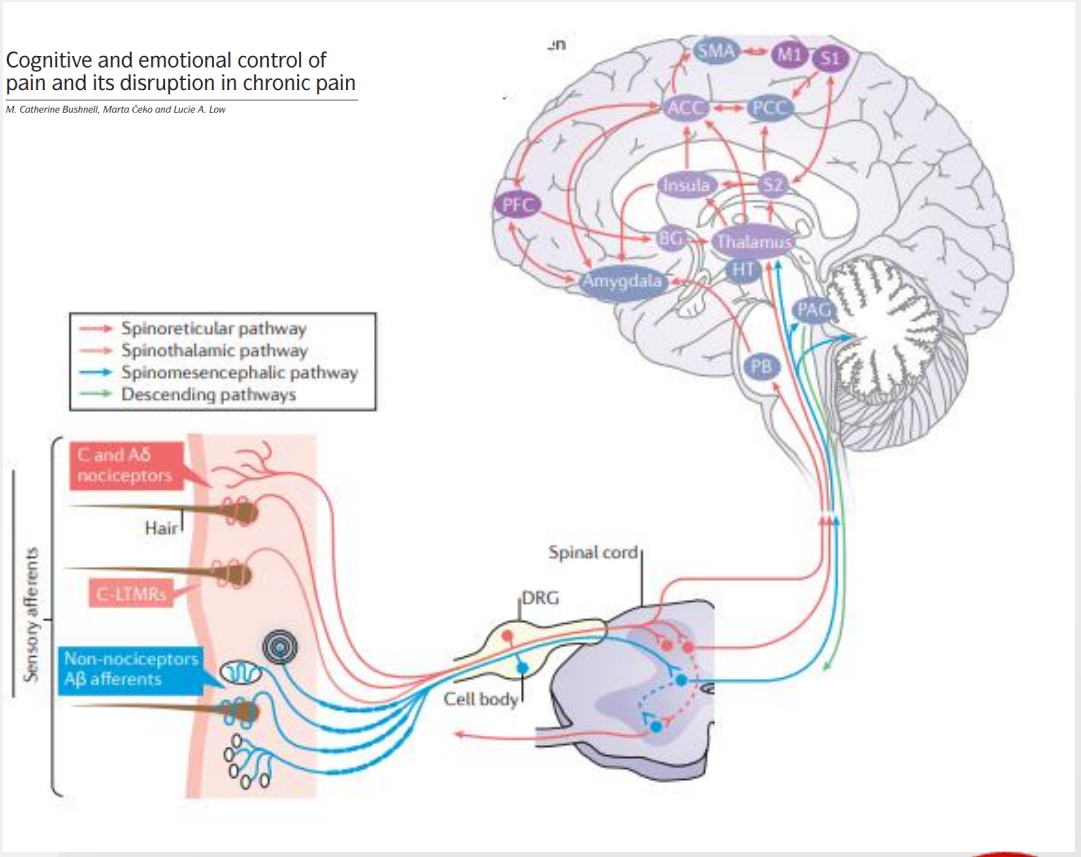

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Menu

CERTA RESPOSTA!

O processamento cíclico e a síntese de impulsos nervosos impõem uma característica padrão de estímulos ou “assinatura neural” (*neurotags*). Três princípios relacionam-se com a intensidade dessas assinaturas e de plasticidade cerebral. São eles: (1) massa neuronal, relacionando-se ao número de células cerebrais integrantes em uma determinada *neurotag* e sua eficácia sinápticas entre elas; (2) precisão neural, ou seja, o quanto consegue-se inibir as células cerebrais não integrantes; e (3) neuroplasticidade, é a propriedade do sistema nervoso central de sofrer alterações estruturais e funcionais em respostas as atividades.

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

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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

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Menu

25 - O que é a *neuromatrix* da dor?

A. Uma área específica do cérebro que está ativada durante estimulação nociceptiva;

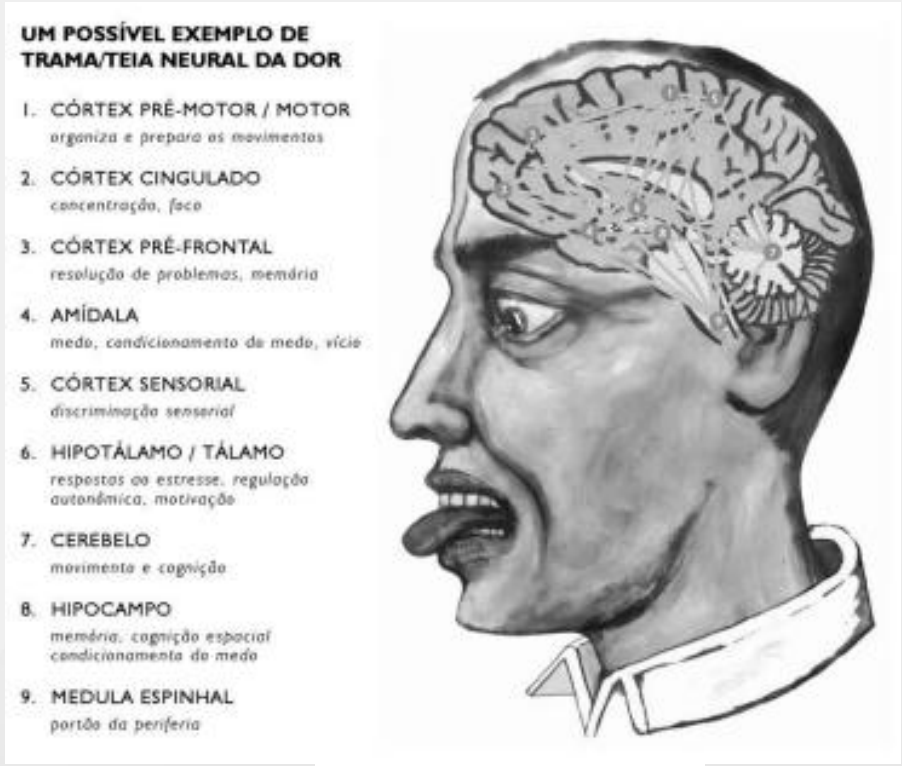
B. A região do córtex somatossensorial 1 e 2 responsáveis pelo processamento da dor;

C. As regiões associadas do neocórtex responsáveis pelo processamento da dor;

D. O conjunto de diferentes regiões do cérebro que estão ativadas durante estimulação nociceptiva.

Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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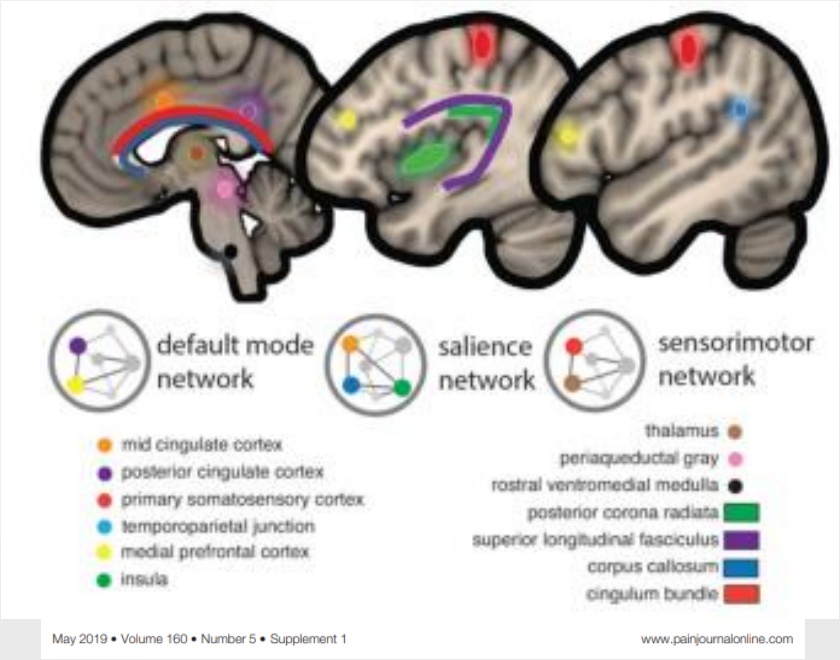

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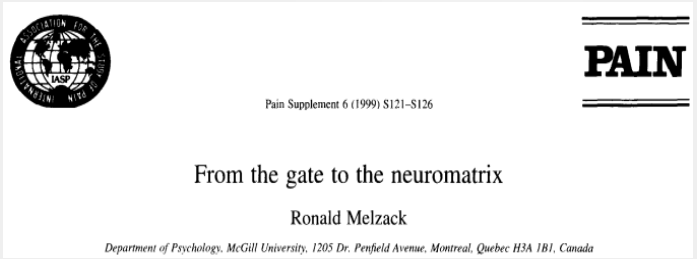
Menu

CERTA RESPOSTA!

A neuromatrix da dor é o conjunto de diferentes regiões do cérebro que estão ativadas durante estimulação nociceptiva. As descrições dos estudos da neuromatrix relacionam-se a nove áreas corticais e subcorticais. Essas diferentes áreas interagem com suas demandas e suas diferentes tarefas. Os estímulos divergem para permitir processos paralelos em diferentes componentes da neuromatrix e convergem para permitir interações entre produtos de processamento de saída. O processamento cíclico e a síntese de impulsos nervosos impõem uma característica padrão de estímulos ou “assinatura neural” (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

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Menu

26 - Uma neurotag de dor (pain neurotag) é descrita, como: “uma rede funcional de neurônios em diferentes áreas do cérebro envolvidas no processamento da dor.” Elas compartilham informações de:

A) estímulos nociceptivos, motores, emocionais, do sistema nervoso autônomo simpático e parassimpático;

B) estímulos sensoriais de ameaça e proteção da dor;

C) estímulos motores de proteção e vigilância da dor;

D) estímulos nociceptivos centrais e do sistema nervoso periférico.

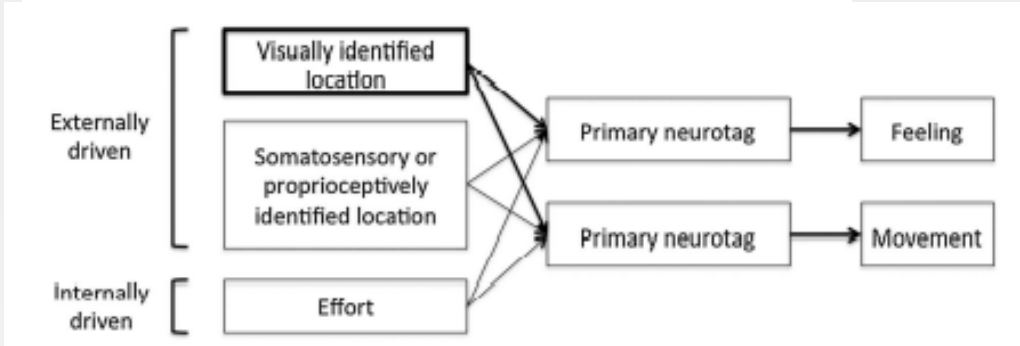
Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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



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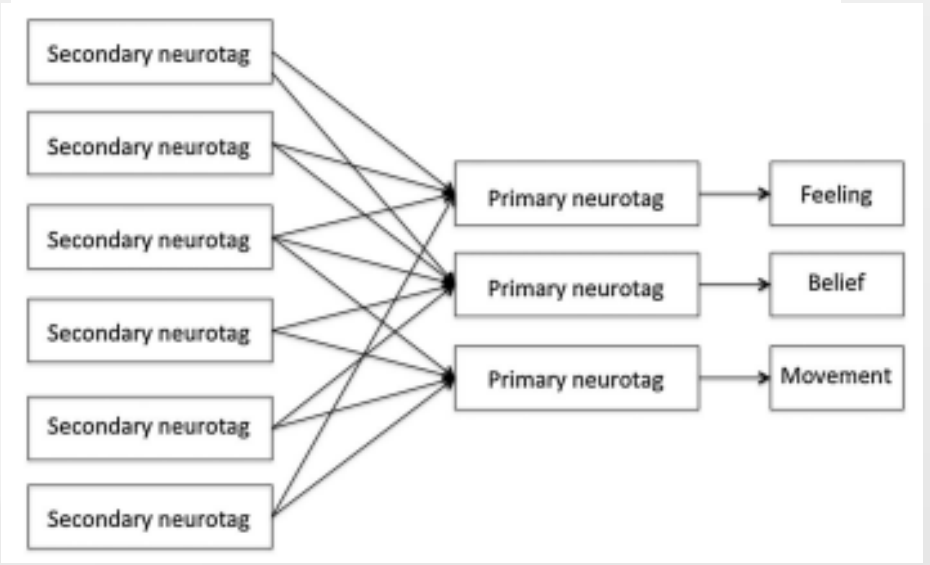
Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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



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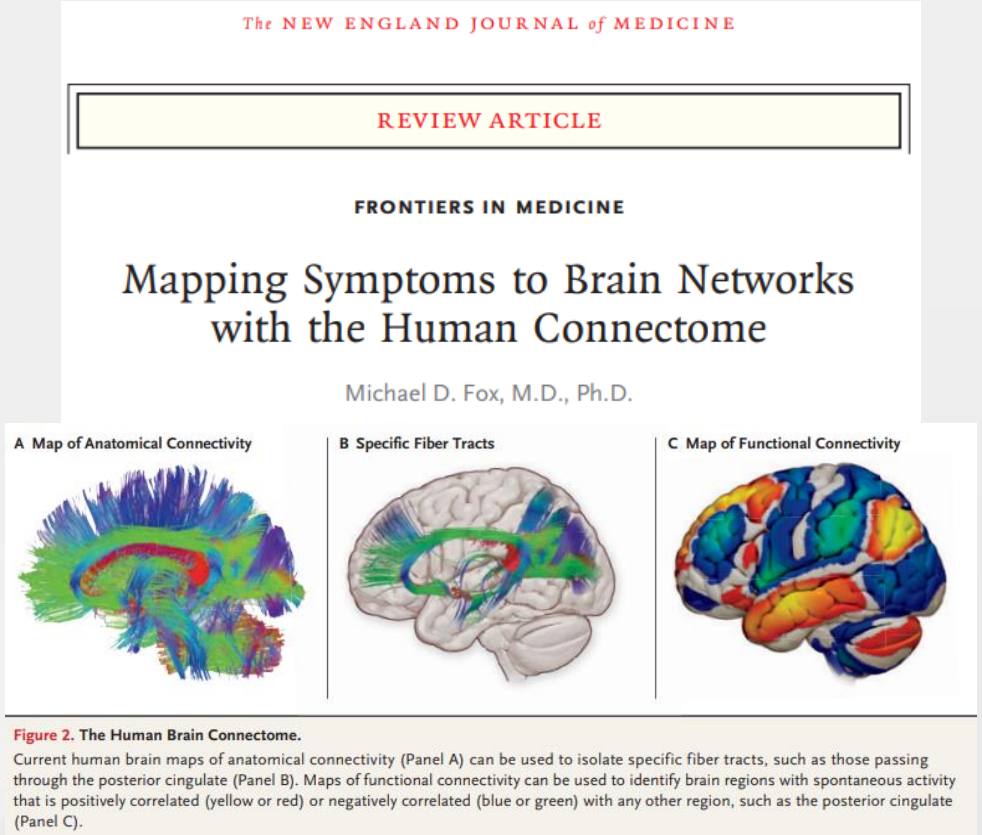
RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST



The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

FRONTIERS IN MEDICINE

Mapping Symptoms to Brain Networks with the Human Connectome

Michael D. Fox, M.D., Ph.D.

A Map of Anatomical Connectivity

B Specific Fiber Tracts

C Map of Functional Connectivity

Figure 2. The Human Brain Connectome.

Current human brain maps of anatomical connectivity (Panel A) can be used to isolate specific fiber tracts, such as those passing through the posterior cingulate (Panel B). Maps of functional connectivity can be used to identify brain regions with spontaneous activity that is positively correlated (yellow or red) or negatively correlated (blue or green) with any other region, such as the posterior cingulate (Panel C).

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Menu

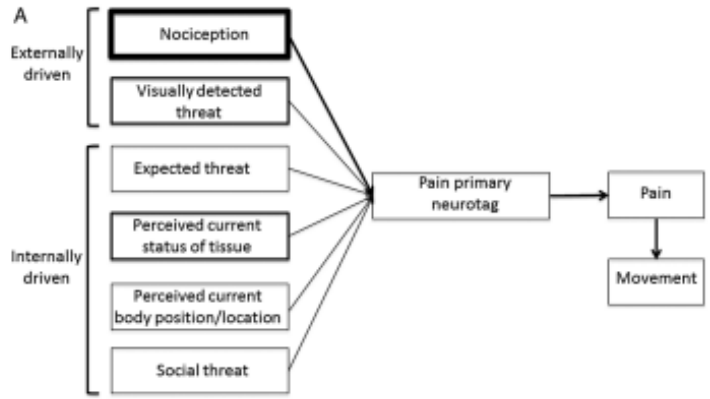
CERTA RESPOSTA!

A intensidade da dor pode ser dissociada da magnitude da resposta na matriz da dor. Os estímulos nociceptivos podem provocar respostas corticais com uma configuração espacial semelhante à da neuromatrix. Sendo assim, uma neurotag de dor (pain neurotag) é descrita, como: “uma rede funcional de neurônios em diferentes áreas do cérebro envolvidas no processamento da dor.” São regiões do cérebro que ao mesmo tempo compartilham informações de estímulos nociceptivos, motores, emocionais, do sistema nervoso autônomo simpático e parassimpático, podendo trabalhar de maneira conjunta ou isolada. Ou seja, a dor é resultado da ativação de várias entradas sobre o SNC.

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



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Menu

27 - São fenômenos de uma plasticidade facilitadora da informação transmitidas da periferia aos centros superiores:

A) Sensibilização alterada e borramento;

B) Hiperalgesia e alodinia;

C) Hiperalgesia e sensibilização alterada;

D) Sensibilização difusa e hipersensibilidade.

Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Quantitative Sensory Testing in Chronic Musculoskeletal Pain

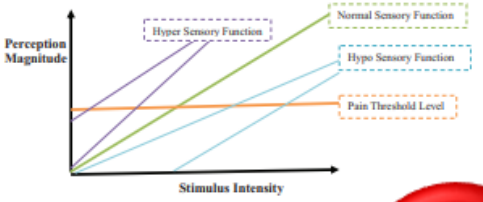
Zakir Uddin, BScPT, MSc, PhD*,† and
Joy C. MacDermid, BSc, BScPT, MSc, PhD*,‡

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.



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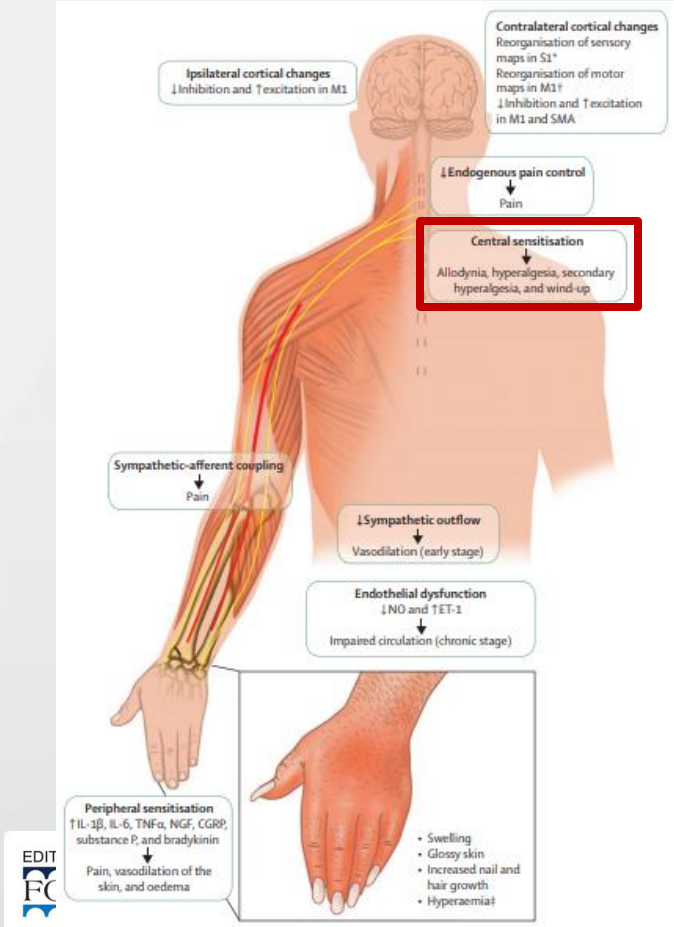
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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



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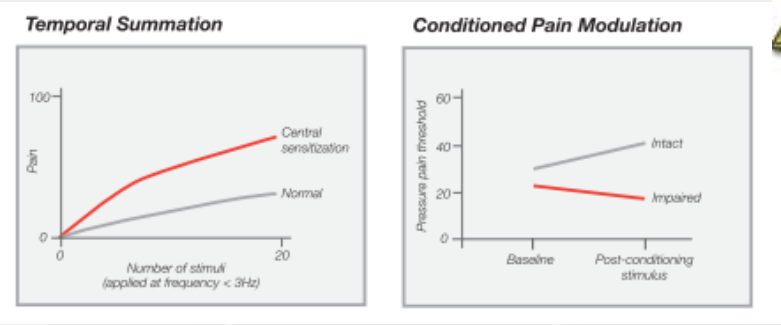
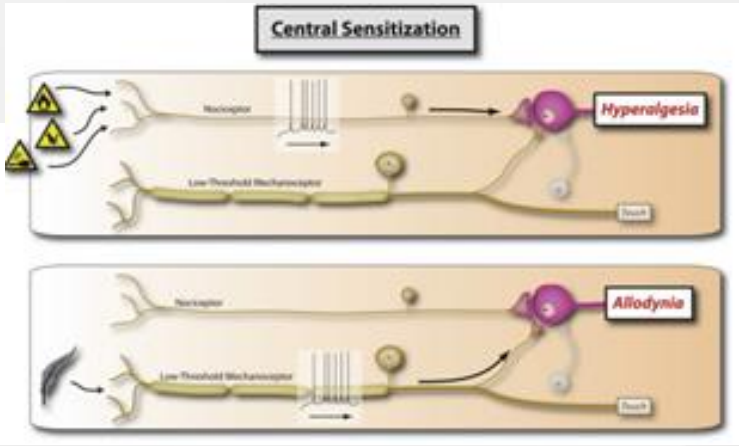
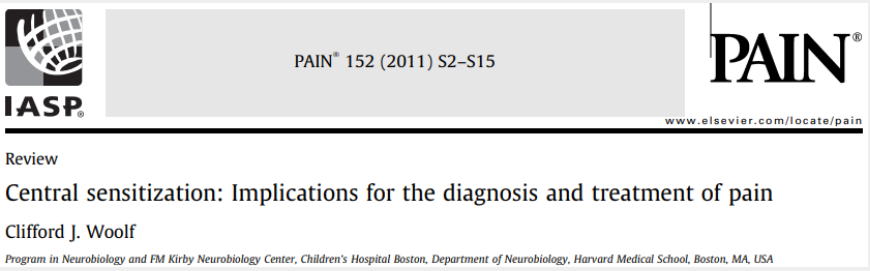
Menu

CERTA RESPOSTA!

A facilitação do sinal de forma que a informação retransmitida para centros superiores, não esteja acoplada a intensidade ou duração do estímulo periférico, podendo resultar em fenômenos neurofisiológicos de hiperalgesia (dado que o estímulo é doloroso, ele é percebido como mais doloroso) e alodinia (perceber um sintoma como doloroso, quando na verdade não é). Esses dois fenômenos serão apresentados em qualquer tipo de sensibilização: que é uma amplificação de uma resposta neuronal, de uma via sensorial (no caso a via nociceptiva), uma vez que se entra em contato com o estímulo..






PAIN® 152 (2011) S2–S15

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

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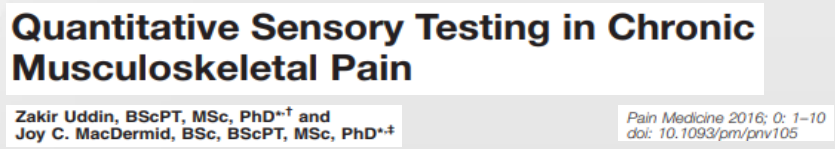
Mechanisms of chronic pain – key considerations for appropriate physical therapy management

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

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Menu

28 - Podemos avaliar sinais de deficiência do sistema somatossensorial a partir de materiais de baixo custo, através de estímulos simples como:

A. Toque leve (bola de algodão ou gaze), picada (clipe de papel ou alfinete) e temperatura (tubo de água a 40 graus C);

B. Toque leve (bola de algodão ou gaze), reflexo tendíneo (martelo neurológico de buck) e estetoscópio;

C. Reflexo tendíneo (martelo neurológico de taylor), diapasão e lente de maddox;

D. Otoscópio, lanterna clínica e esfigmomanômetro.

Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

[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…

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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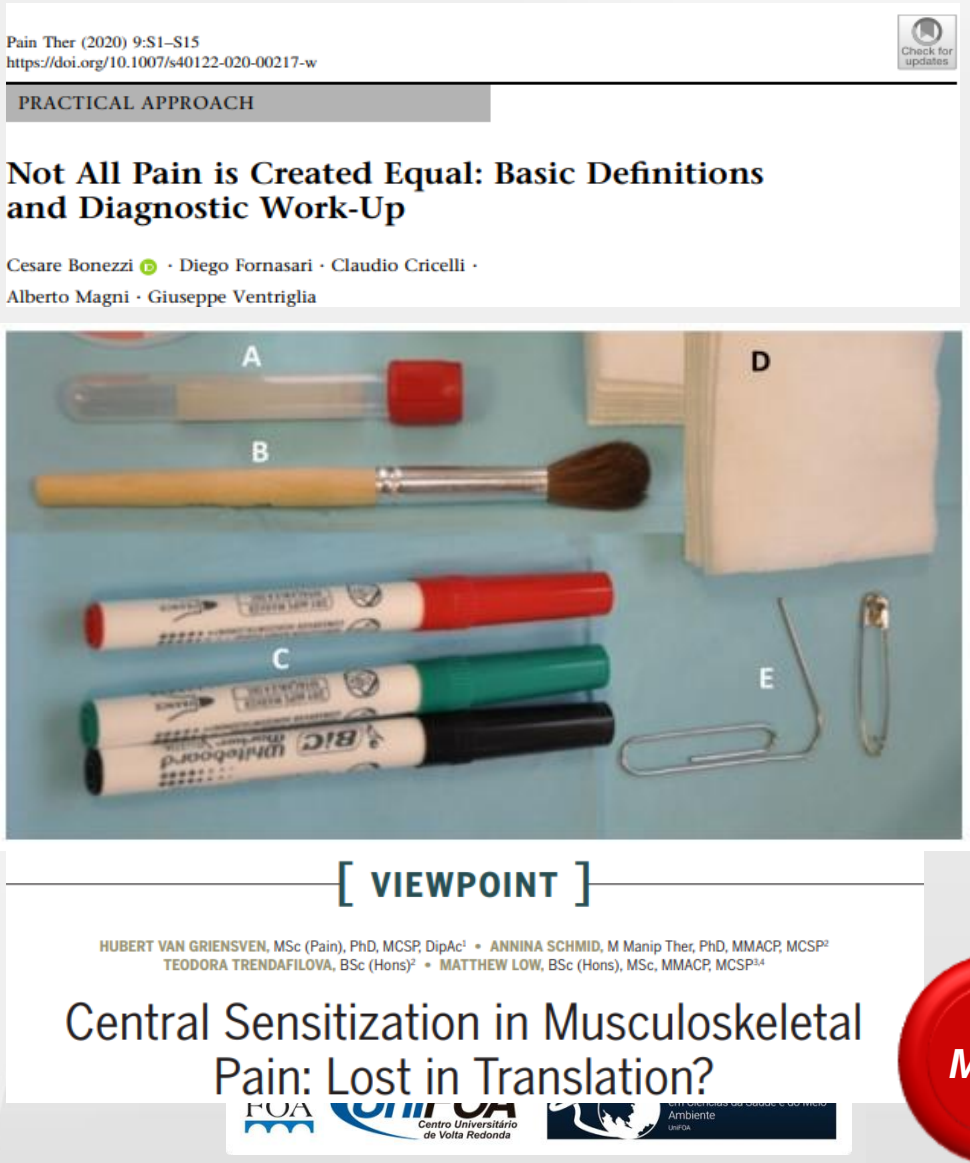
CERTA RESPOSTA!

Três estímulos são usados para testar a sensibilidade à discriminação de toque leve com uma bola de algodão ou gaze, picada com uso de um clipe de papel e/ou alfinete e temperatura com um tubo de água a 40 ° C. A partir desta avaliação, podemos determinar se o paciente apresenta ou não déficits sensoriais, possibilitando avaliar a resposta do sistema nervoso aos estímulos exógenos somáticos nocivos e inofensivos, e eventualmente a dor. Os fisioterapeutas devem estar atentos à sensibilização central e considerar potencias top-down e bottow-up no contexto de uma abordagem biopsicossocial centrada na pessoa.



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29 - Podemos avaliar quantitativamente o limiar de dor que podem estar associados a processos de sensibilização central e/ou periférica em âmbito clínico através de qual aparelho?

A Esfignomamômetro;

B Algômetro de pressão;

C Baropodômetro;

D Dinamômetro.

Menu

GUSTAVO FERRAZ
Fisioterapia Integrada

QUESTIONADOR

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

[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

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RESPOSTA ERRADA! CLIQUE NO PODCAST, INTERAJA E TENTE OUTRA VEZ!

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)	↓	→	→

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

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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]

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CERTA RESPOSTA!

Podemos avaliar o limiar de dor à pressão com o uso de um algômetro de pressão que nos fornece informações em Kg/cm2. A algometria de pressão pode ser utilizada em diversos segmentos corporais (ventre muscular, tendão e superfícies ósseas). O aparelho valida e viabiliza medições quantitativas de limiares de dor na rotina clínica, beneficiando a avaliação principalmente na atenção primária. A comprovação atual é que um algômetro de pressão de baixo custo é válido e confiável o suficiente para ser considerado um equipamento padrão para avaliar o limiar de dor à pressão. Índices de testes quantitativos sensoriais de hipersensibilidade à dor podem ajudar a desenvolver intervenções direcionadas com o objetivo de melhorar os resultados em uma variedade de condições musculoesqueléticas.

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]

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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]

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30 - A analgesia endógena e o funcionamento do sistema anti-nociceptivo pode ser investigada em âmbito clínico através do teste de:

A. Teste de disdiadococinesia;

B. Teste de sensibilidade dolorosa epicrítica;

C. Teste de CPM (conditioned pain modulation);

D. Teste de palestesia ou sensibilidade vibratória.

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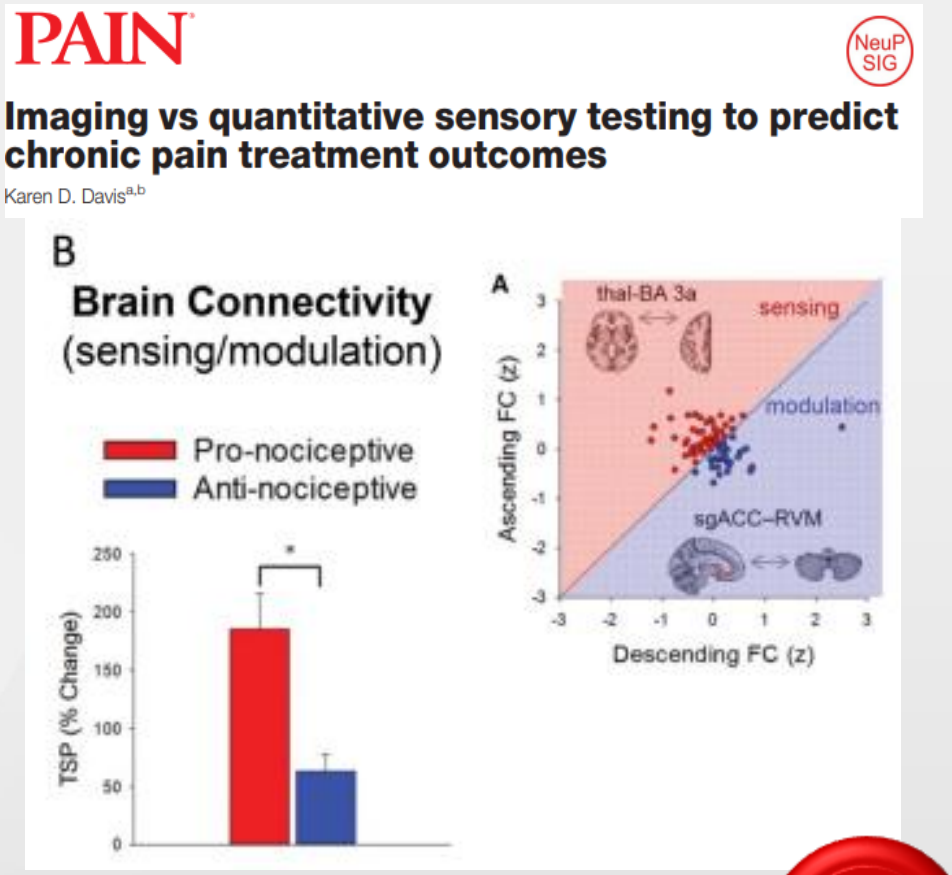

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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

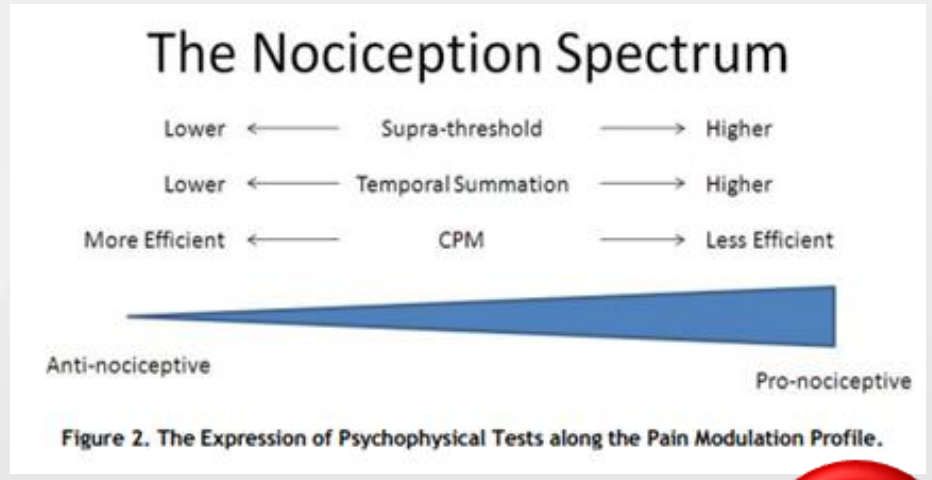

Figure 2. The Expression of Psychophysical Tests along the Pain Modulation Profile.

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

QUESTIONADOR PODCAST

American Pain Society
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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.

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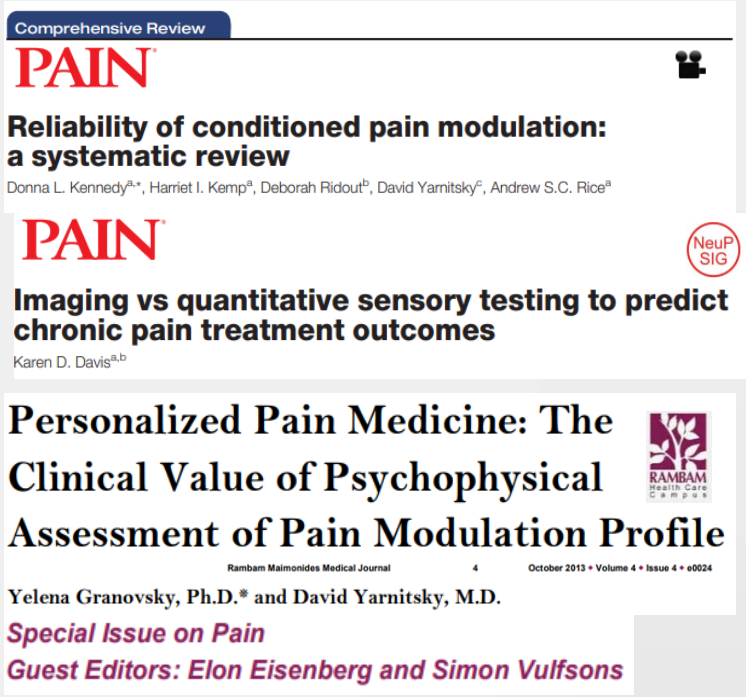
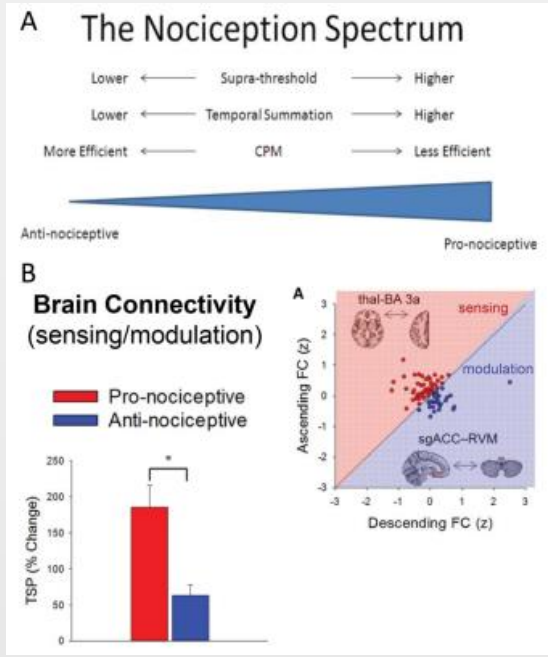
CERTA RESPOSTA!

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);





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31 - A busca de inibir a dor por estímulo condicionante pelo teste do CPM passivo, que pode ser feita de qual forma?

A — Eletroneuromiografia e dinamometria;

B — Balde de gelo e esfigmomanômetro;

C — Eletromiografia e estesiometria;

D — Baropodometria e estabilometria.

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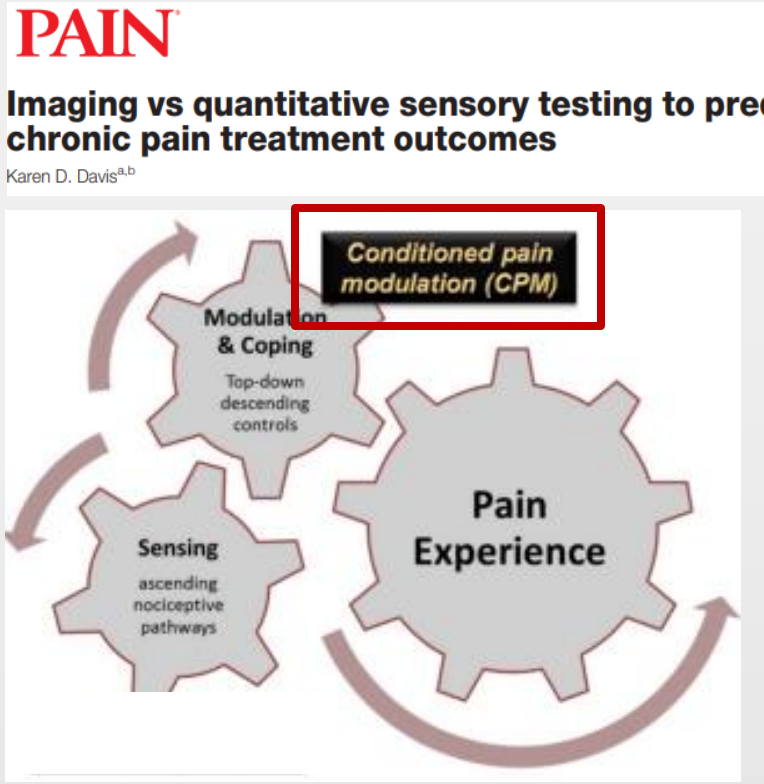
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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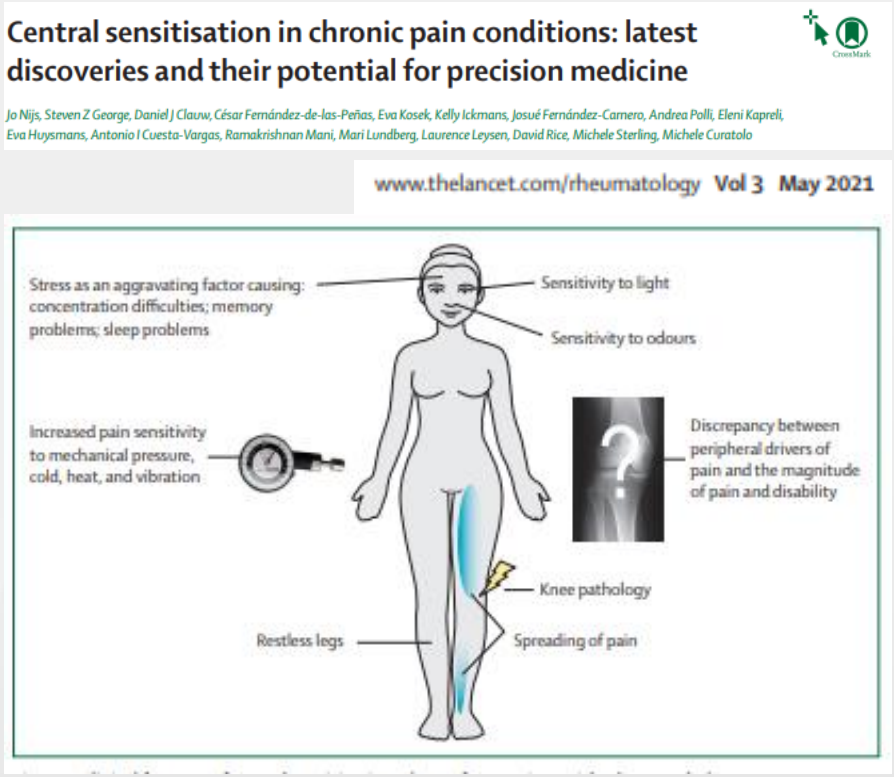

Central sensitisation in chronic pain conditions: latest discoveries and their potential for precision medicine

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

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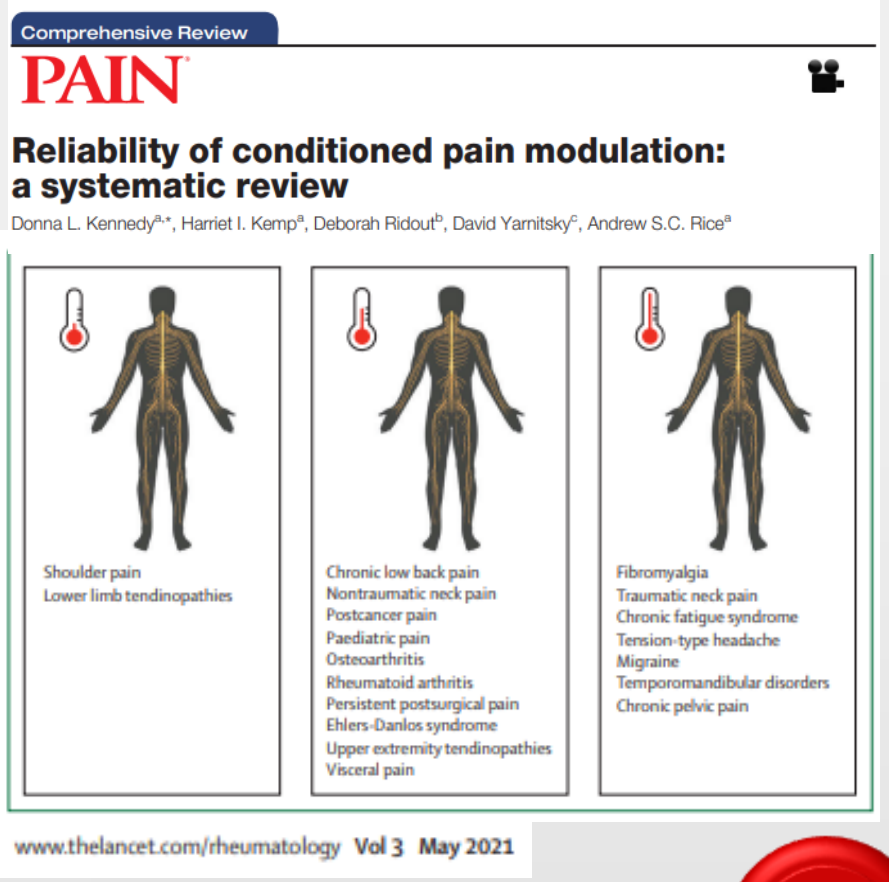
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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CERTA RESPOSTA!

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

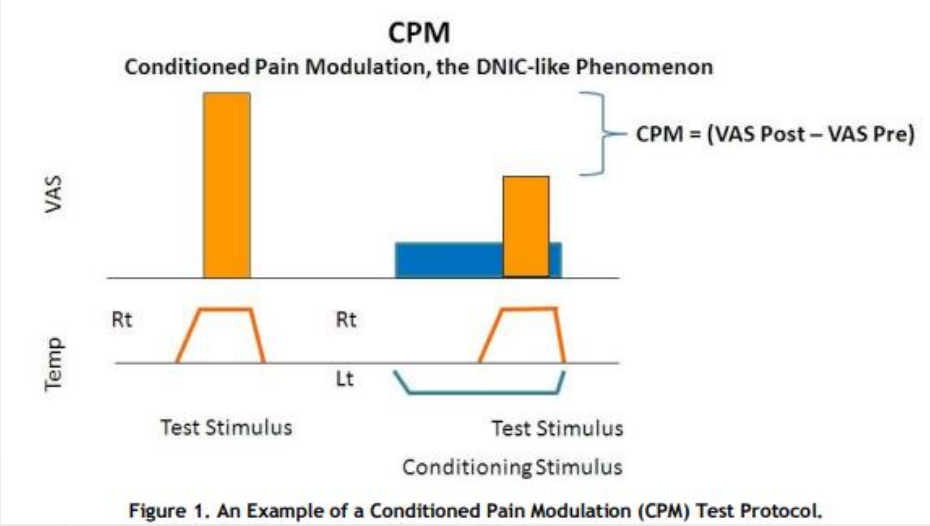

Figure 1. An Example of a Conditioned Pain Modulation (CPM) Test Protocol.

O teste do CPM passivo consiste em 3 (três) fases: *pré-estímulo*: avaliar o limiar de dor à pressão 3 locais anatômicos diferente com o algômetro de pressão. O *estímulo condicionante* que pode ser aplicado pela imersão de um dos membros num balde com gelo ou através de esfigmomanômetro. A descrição de carga pelo manguito é de 20 em 20mmHg até que a percepção de dor aconteça, e após esse primeiro limiar o paciente deve relatar uma EVA média de 5 pontos. A partir desse estímulo condicionante aguardamos 2 minutos em teste. *Pós-estímulo*: reavaliamos o limiar de dor à pressão nos 3 pontos anatômicos ainda sob efeito do estímulo através do esfigmomanômetro. Escores positivos com: (limiar pós – limiar pré) = são indicativos de uma função anti-nociceptiva preservada. Ou seja, o sistema nociceptor auxilia com o mecanismo endógeno.

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Menu

32 - Podemos avaliar de forma quantificada um estado de pró-nocicepção aumentado na prática clínica, através de qual fenômeno?

A Wind-Up (somação temporal);

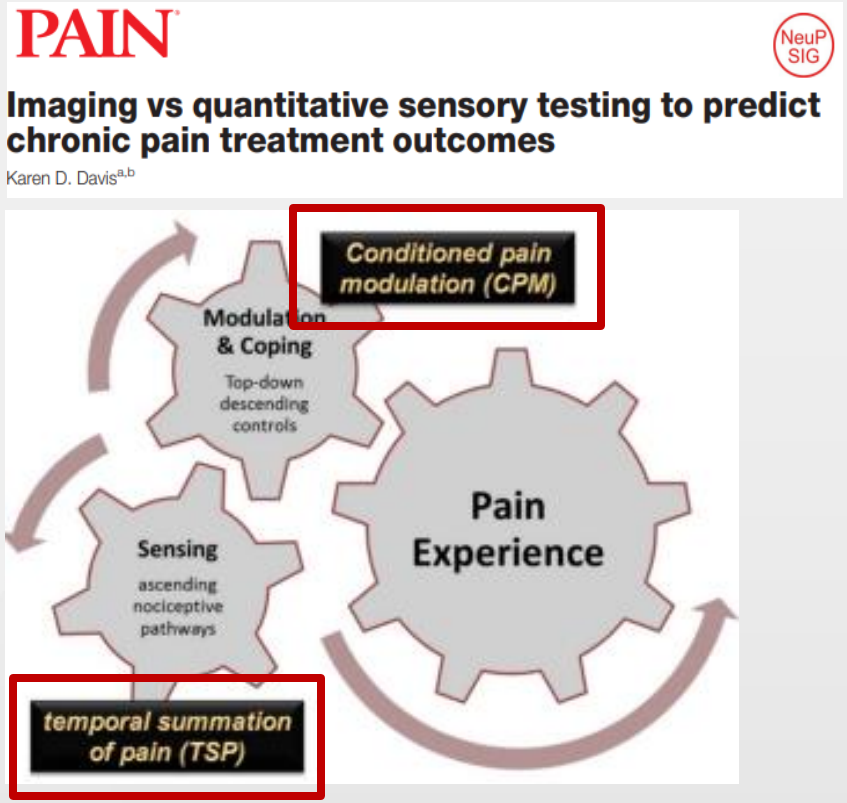
B Parestesia;

C Paresia;

D Hipoestesia.

Menu

RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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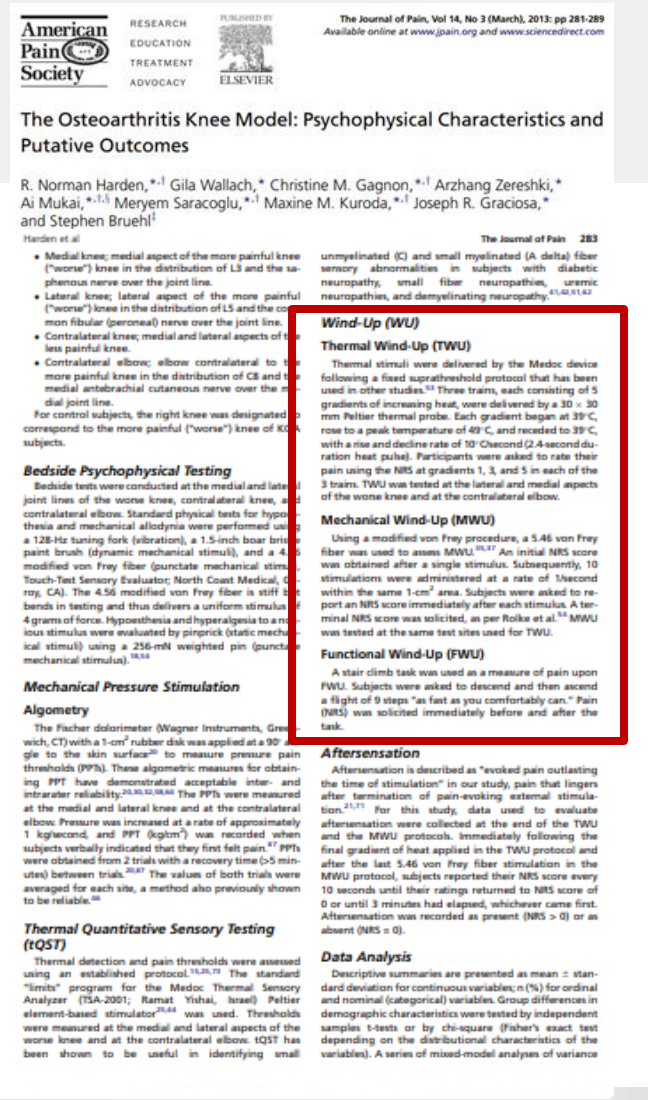

American Pain Society — RESEARCH EDUCATION TREATMENT ADVOCACY — PUBLISHED BY ELSEVIER

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‡

- 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

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

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CERTA RESPOSTA!

A presença de somação temporal (Wind-Up) é um dos sintomas que podem indicar um estado de pró-nocicepção aumentado. Com a utilização de um algômetro de pressão de baixo custo, podemos quantificar a avaliação. O primeiro passo é identificar o limiar de dor à pressão do paciente utilizando o algômetro. O protocolo de somação temporal (Wind-Up) é descrito com a realização de 10 (dez) repetições do estímulo pressórico no paciente, retornando ao zero (ou negativo do aparelho). Durante a quinta (5) e a décima (10) repetição solicitamos ao paciente que aponte novamente sua EVA. Como já realizamos 5 pressões até esse momento do teste, a tendência é que o paciente perceba um pouco mais o limiar já avaliado, com um pouco mais de intensidade. Dessa forma teremos 3 (três) valores: no início do protocolo quando avaliamos o limiar de dor analógica do paciente. Durante o 5 (quinto) momento e ao 10 (décimo) momento do teste. O resultado do protocolo de somação temporal é a diferença entre a escola visual analógica na 10 (décimo) estímulo e no estímulo inicial. Interpretação do teste: (escore positivo = pró-nocicepção aumentada).

European Journal of Pain (2000) **4**: 5–17
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Review Article

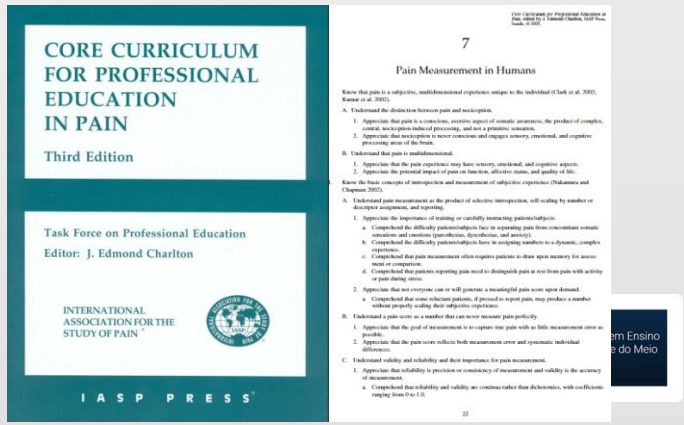
Wind-up and the NMDA receptor complex from a clinical perspective

Per K. Eide

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

Comentários: mecanismos anormais periféricos - somação temporal

"os resultados dos exames clínicos com doentes com dor crônica, sugerem que o receptor NMDA pode apresentar um novo alvo para a modulação somação anormal da dor."



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33 - Paciente sexo feminino, chega à consulta apresentando dor lombar há cerca de 6 (seis) anos, de origem lenta e progressiva, sem eventos traumáticos descritos, levando a uma baixa probabilidade de lesões teciduais, e maiores possibilidades de eventos de sensibilidade aumentada nos tecidos. Sedentária e histórico de depressão. Testes quantitativos sensoriais (QST) apresentado hiperalgesia e alodinia. Dessa forma, uma possível hipótese com as descrições da anamnese nos leva ao mecanismo:

A — Neuropático;

B — Nociceptivo;

C — Nociplástico;

D — Neuropático com sobreposição nociceptiva.

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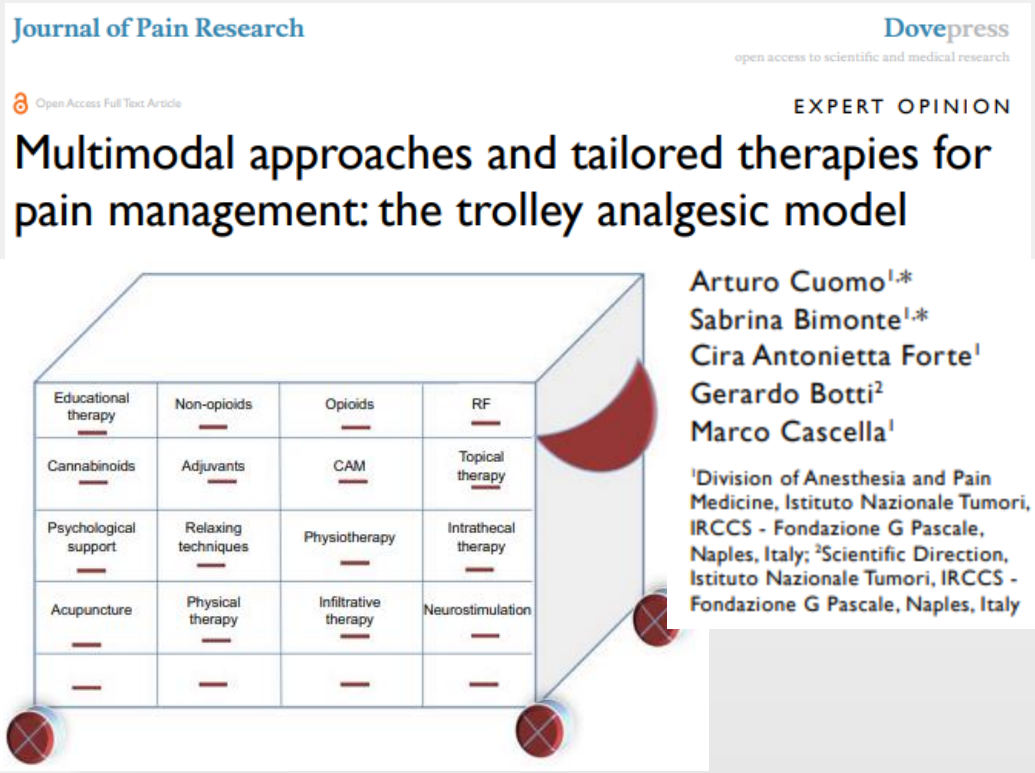
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Perspective

A Mechanism-Based Approach to Physical Therapist Management of Pain

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

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

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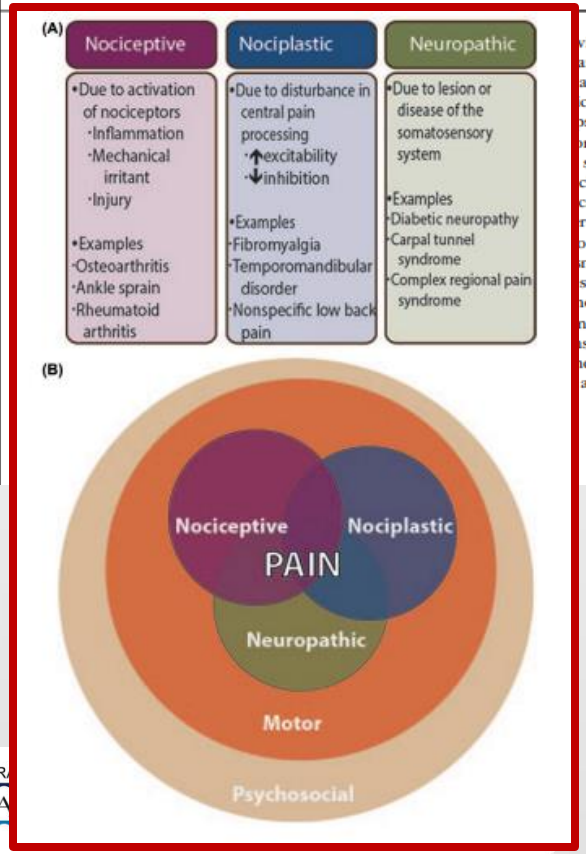

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

Perspective

A Mechanism-Based Approach to Physical Therapist Management of Pain

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

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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

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CERTA RESPOSTA!

Dor de origem lenta e progressiva, sem eventos traumáticos descritos, levando a uma baixa probabilidade de lesões teciduais, e maiores possibilidades de eventos de sensibilidade aumentada nos tecidos (sensibilização). **Dessa forma, uma possível hipótese com as descrições da anamnese nos leva ao mecanismo neurofisiológico nociplástico**. Associado aos testes quantitativos sensoriais, com utilização do algômetro de pressão, avaliando a hiperalgesia/alodinia mecânica, seguindo um padrão mais local ou difuso (como relatado pela paciente) sem uma área neuro anatômica específica.

Utilizamos uma carga de 1kgf por 1 segundo, dentro da área relatada dolorosa. Solicitamos o relato do paciente do momento ao qual a pressão comece a incomodar. Depois avaliamos o limiar no lado contralateral a queixa dolorosa. Observando a sensibilidade comparada a pressão, o lado doloroso apresenta um limiar mais baixo. Quando progredimos com os testes para área torácica, observamos um limiar de dor mais predominante em um dimídio corporal. Ou seja, sem padrão neuro anatômico definido. Quando avançamos com avaliação dos testes quantitativos sensoriais (QST) com estímulos de picada com a agulha, e comparamos o dedo médio da mão direita com a coluna lombar à direita, observamos sinais sensoriais aumentados e possível hiperalgesia. Quando continuamos a exploração do teste, observamos sinais de hiperalgesia e alodinia espalhados em todo dimídio direito e sugestiona uma possível pró-nocicepção aumentada em regiões superiores das vias nociceptivas. Continuando a avaliação através do teste de Wind-Up ou Somação Temporal, avaliamos a repetição do estímulo várias vezes e a tendência é que a dor piore para o mesmo estímulo, levando a um resultado positivo no teste. Quando chegamos na quinta repetição do teste, apresentou mais dor relatada pela EVA (de 5 foi para 7) e na décima repetição novamente mais dor (EVA de 7 foi para 8), o que indica Wind-Up positivo.

O teste positivo remete sinais de pró-nocicepção, sendo assim, o aumento da via nociceptiva está presente. Seguimos o exame clínico através do CPM avaliando o funcionamento da via analgésica endógena, induzindo dor na paciente. Padronizamos a região do trapézio contralateral ao lado doloroso, inflando o esfigmomanômetro até a sensação dolorosa de EVA média. Aguardamos por 2 minutos, repetimos a algometria sobre a mesma área testada inicialmente, ainda sobre efeito do estímulo condicionante (esfigmomanômetro) apresentando um aumento do limiar ao final do teste, o que mostra uma via analgésica funcionante. As abordagens que podem contribuir com o auxílio de progressão do quadro doloroso, estão em exercícios que irão ativar o sistema de opioides endógenos auxiliando na neuromodulação da dor através de uma cascata de efeitos analgésicos e anti-inflamatórios.

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34 - A partir dos dados coletados na anamnese e exame clínico, o paciente apresentou os testes de Wind-Up (Somação Temporal) positivo e CPM (Modulação Condicionada da Dor) funcionante. Sendo assim, optou-se por conduta fisioterápica através de terapia manual e cinesioterapia. Com o Wind-Up (Somação Temporal) positivo, teríamos algumas possibilidades por intervenção através de terapia manual. . Quando observamos pelo CPM (Conditioned Pain Modulation – Modulação Condicionada da Dor) um sistema anti-nocicepção preservado, a escolha por exercícios terapêuticos passa a ser uma boa estratégia. A escolha por essa conduta pode se beneficiar então:

A mobilização articular grau 2 associada a exercícios localizados;

B manipulação articular (thrust) associada a exercícios sistêmicos;

C manipulação articular (thrust) associada a exercícios localizados;

D mobilização articular grau 4 associado a exercícios sistêmicos.

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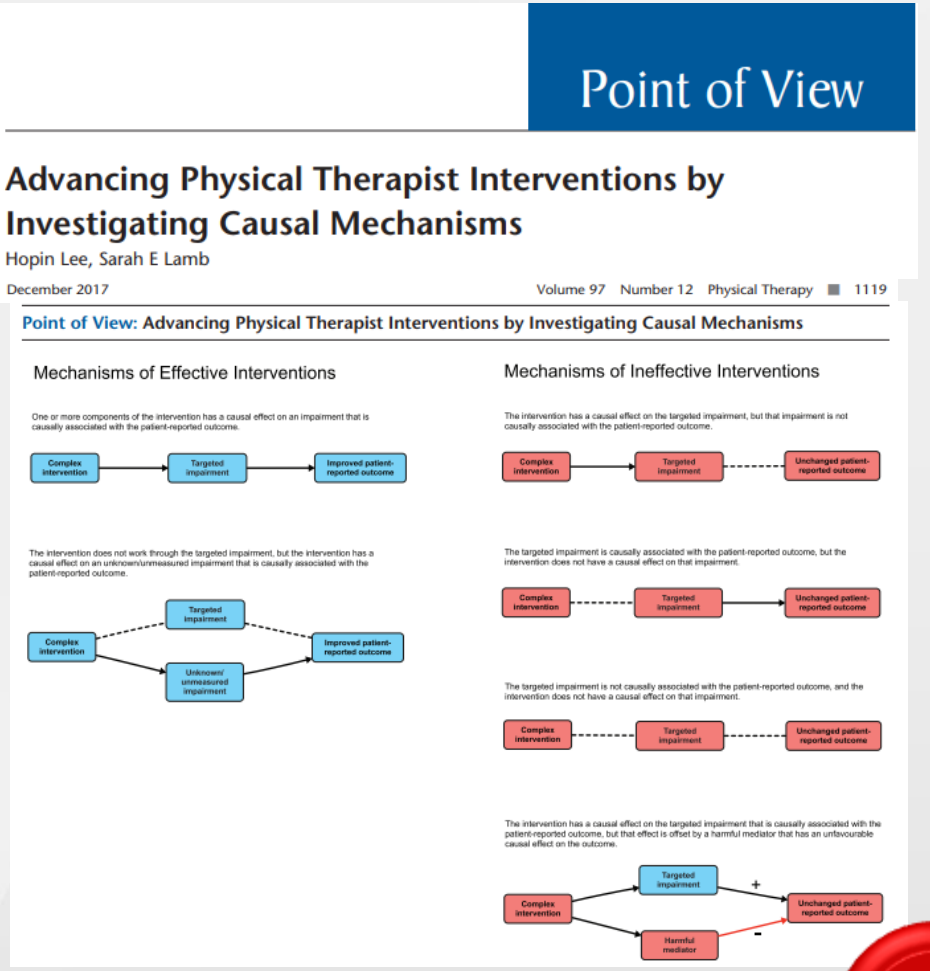
RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!



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 (-)

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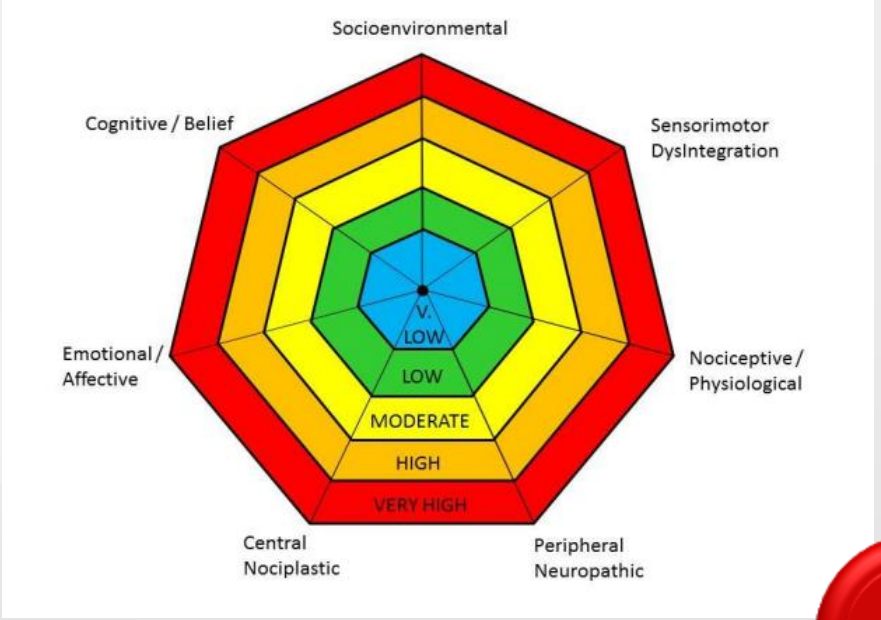

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RESPOSTA ERRADA! CLIQUE NO VÍDEO, INTERAJA E TENTE OUTRA VEZ!

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]

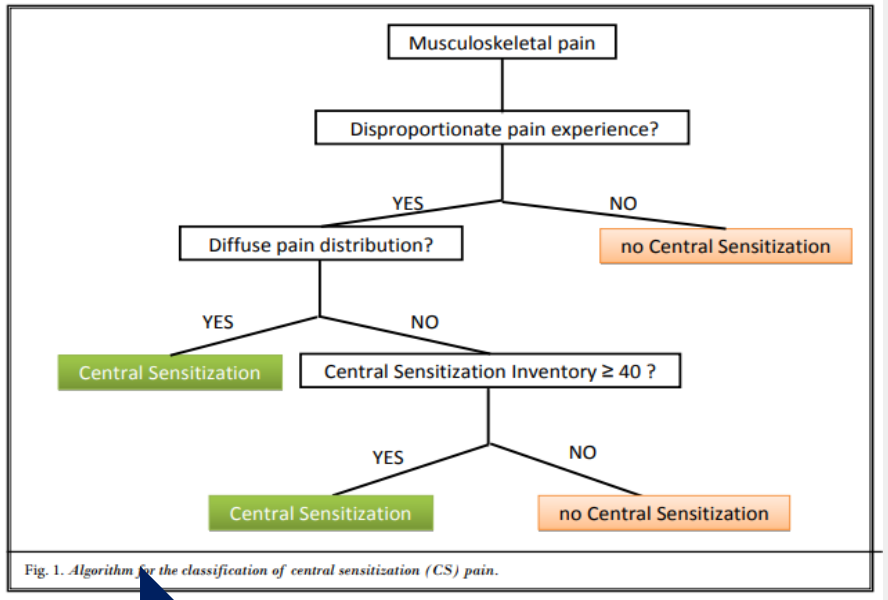

Fig. 1. *Algorithm for the classification of central sensitization (CS) pain.*

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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[‡,§,¶,‖‖]

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UniFOA Centro Universitário de Volta Redonda
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CERTA RESPOSTA!

Com o Wind-Up (Somação Temporal) positivo, teríamos algumas possibilidades por intervenção através de terapia manual. Manipulação articular (thrust) ou mobilização articular, gerando o mesmo efeito terapêutico.

A escolha pela manipulação se baseia que um estímulo único não reproduzirá a somação de estímulos que a mobilização articular repetidamente pode gerar sobre o sistema nervoso da paciente, o que pode contribuir para aumento de sensibilidade, não conseguindo gerar efeitos analgésicos usando o teste como referência para a escolha do tipo de manobra utilizada. A prescrição de exercícios também se baseia nessa hipótese.

Quando observamos pelo CPM (Conditioned Pain Modulation – Modulação Condicionada da Dor) um sistema anti-nocicepção preservado, a escolha por exercícios terapêuticos passa a ser uma boa estratégia. Porém como a paciente apresenta Wind-Up positivo, pode não responder bem a exercícios com foco local na área dolorosa, pela somação de estímulos. Portanto a escolha de exercícios que não sejam analíticos e sim sistêmicos, como exercício aeróbico de baixa-moderada intensidade, pode ajudar mais estimulando o sistema endógeno descendente de neuromodulação da dor.

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Quando pensamos em um diálogo que contemple e auxilie todo conteúdo que aprendemos até aqui, ele deve ser pautado em exercícios direcionados a cognição. Através de uma comunicação que transfira a teoria da Pain Neuroscience Education à prática clínica.

Chegamos ao final do quiz Moderna Dor, e nessa última questão apresento um diálogo prático entre fisioterapeuta e paciente, transcrito e narrado como na seção Box 1, 3 e 4, do artigo "Applying contemporary neuroscience in exercise interventions for chronic pain: treatment protocol", publicado em 2017 na BjPT.

Diálogo

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.''*

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CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 1!

Fisioterapeuta

Paciente

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CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 1!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 1!

retornar

Fisioterapeuta

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 2!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 2!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 2!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 2!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 3!

Fisioterapeuta

Paciente

retornar

Menu

CLIQUE NO VÍDEO E INTERAJA COM A COMUNICAÇÃO ATRAVÉS DE EXERCÍCIOS DIRECIONADOS A COGNIÇÃO – DIÁLOGO 3!

Fisioterapeuta

Paciente

retornar

Menu

MUITO OBRIGADO!

Menu