Escuela de Osteopatía de Madrid Internacional

INTESTINO
BIOQUÍMICA POR ALIMENTACIÓN
Parte 4

DOLOR SOMÁTICO, INTESTINO Y NERVIO VAGO

AL ENFRENTARNOS AL ESTUDIO Y TRATAMIENTO DEL SNV:

- Y .. ¿ cómo puede afectar la inflamación intestinal a una rodilla ? , por ejemplo.
- La actividad de las aferentes vagales que inervan el intestino Delgado (proyectándose sobre las ramas celiacas de los nervios vagos abdominales) es importante en la modulación refleja de procesos inflamatorios (como en la articulación de la rodilla) y una respuesta hiperalgésica a la sobrecarga mecánica (debido a la sensibilización de nociceptores) en tejidos a distancia vía sistema nervioso simpatico-suprarenal y el eje hipotálamo-hipofisario-suprarenal.

Eur J Neurosci. 1998 Feb;10(2):435-44.

Modulation of bradykinin-induced mechanical hyperalgesia in the rat by activity in abdominal vagal afferents.

Khasar SG[1], Miao JP, Jänig W, Levine JD.

Author information

1 Department of Anatomy, University of California at San Francisco, 94143-0452, USA.

Abstract

Bradykinin-induced plasma extravasation and mechanical hyperalgesia are sympathetic-dependent components of inflammation. Noxious stimulation has been found to inhibit bradykinin-induced plasma extravasation by activating the hypothalamo-pituitary-adrenal axis. The sensitivity of this nociceptive-neuroendocrine feedback control of inflammation is modulated by activity in subdiaphragmatic vagal afferents. In the present study, we tested the hypothesis that activity in the subdiaphragmatic vagus also modifies bradykinin-induced mechanical hyperalgesia in the rat, using the Randall-Selitto method. Following subdiaphragmatic vagotomy, the baseline paw-withdrawal threshold to mechanical stimulation decreased and bradykinin-induced mechanical hyperalgesia was enhanced. Mechanical hyperalgesia produced by prostaglandin E2, a direct-acting hyperalgesic agent, was not significantly affected by vagotomy. The effect of subdiaphragmatic vagotomy on bradykinin-induced hyperalgesia, but not on baseline paw-withdrawal threshold, was mimicked by coeliac branch vagotomy. Indomethacin blocked the hyperalgesia in normal rats, but not in vagotomized rats, suggesting that bradykinin-induced hyperalgesia in normal rats is mediated by prostaglandins, whose role was unexpectedly diminished after vagotomy. Bradykinin-induced hyperalgesia in normal rats was abolished by lumbar sympathectomy but not by sympathetic decentralization (cutting the preganglionic axons). In rats that were both vagotomized and sympathectomized, hyperalgesia induced by low-dose bradykinin was no longer present. These results demonstrate that vagotomy induces a decrease in baseline mechanical paw-withdrawal threshold and an enhancement of bradykinin-induced mechanical hyperalgesia and suggest that these phenomena are generated by actions in peripheral tissues.

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MODULACIÓN VAGAL = ANALGESIA + MOTILIDAD

ESTE ESTUDIO EXPERIMENTAL SUGIERE QUE LA MODULACIÓN DEL TONO VAGAL INCREMENTA LA MOTILIDAD GASTROINTESTINAL Y REDUCE EL DOLOR SOMÁTICO.

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Modulation of vagal tone enhances gastroduodenal motility and reduces somatic pain sensitivity

J. B. FRØKJÆR,*,† S. BERGMANN,*,† C. BROCK,‡,§ A. MADZAK,* A. D. FARMER,‡,¶,** J. ELLRICH†† & A. M. DREWES†,‡

*Mech-Sense, Department of Radiology, Aalborg University Hospital, Aalborg, Denmark
†Department of Clinical Medicine, Aalborg University, Aalborg, Denmark
‡Mech-Sense, Department of Gastroenterology & Hepatology, Aalborg University Hospital, Aalborg, Denmark
§Department of Drug Design and Pharmacology, University of Copenhagen, Copenhagen, Denmark
¶Department of Gastroenterology, University Hospitals of North Midlands, Stoke on Trent, UK
**Centre for Digestive Diseases, Blizard Institute, Wingate Institute of Neurogastroenterology, Barts and the London School of Medicine & Dentistry, Queen Mary University of London, London, UK
††Department of Health Science and Technology, Aalborg University, Aalborg, Denmark

Key Points

- The aim was to explore the effect of combined electrical and physiological modulation of vagal tone on musculoskeletal pain thresholds and gastroduodenal motility.
- Eighteen healthy subjects were included in a subject-blinded, sham-controlled, cross-over study with an active protocol including transcutaneous electrical vagal nerve stimulation and deep slow breathing.
- Recording of cardiac derived parameters including cardiac vagal tone, moderate pain thresholds to muscle and bone pressure algometry, conditioned pain modulation using a cold pressor test, and a liquid meal ultrasonographic gastroduodenal motility test were performed.
- Cardiac vagal tone, thresholds to bone pain, frequency of antral contractions, and gastroduodenal motility index all increased during active treatment compared to sham.
- The presented noninvasive approach with combined electrical and physiological modulation of vagal tone enhances gastroduodenal motility and reduces somatic pain sensitivity.
- These findings warrant further investigation in patients with disorders characterized with chronic pain and gastrointestinal dysmotility such as functional dyspepsia and irritable bowel syndrome.

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LA VAGOTOMÍA PRODUCE HIPERALGESIA

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Vagotomy-Induced Enhancement of Mechanical Hyperalgesia in the Rat Is Sympathoadrenal-Mediated

Sachia G. Khasar,[1] Frederick J.-P. Miao,[1] Wilfrid Jänig,[2] and Jon D. Levine[1]

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Abstract Go to:

We have recently shown that subdiaphragmatic vagotomy enhances bradykinin-induced hyperalgesic behavior and decreases baseline paw withdrawal threshold to mechanical stimulation of the hindpaw skin in rats by a peripheral mechanism. To elucidate the underlying mechanism, we studied whether lesions of efferent neuroendocrine pathways could prevent or reverse the potentiating effect of vagotomy. In groups of sham-vagotomized or vagotomized rats, we surgically removed or denervated the adrenal medulla. Bradykinin was injected intradermally into the skin of the dorsal surface of the rat hindpaw. Threshold of paw withdrawal to mechanical stimulation of the skin was measured.

Vagotomy induced a decrease in mechanical baseline paw withdrawal threshold and enhancement of bradykinin-induced mechanical hyperalgesic behavior, both of which were maintained over the 5 week testing period. Adrenal enucleation or denervation of the adrenal gland by suprarenal ganglionectomy prevented vagotomy-induced decrease in baseline paw withdrawal threshold and enhancement of bradykinin-induced hyperalgesia. In animals that had a demonstrated decrease in baseline paw withdrawal threshold and enhancement of bradykinin-induced hyperalgesia 2 weeks after vagotomy, additional denervation of the adrenal medulla significantly reversed these effects over a 3 week period.

Y estimular el vago, es analgésico??? sí.

La estimulación eléctrica de las aferentes abdominales vagales inhibe la transmission de impulsos nociceptivos en las astas posteriors de la médula y deprime la nocicepción.

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ELSEVIER

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Vagal modulation of nociception

G.F. Gebhart *, A. Randich †

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The role of vagal afferents in cardiopulmonary and gastrointestinal function is well-established. An appreciation of their role in the modulation of pain, however, is relatively recent. The authors present studies that investigated the hypothesis that physiological stimuli activate vagal afferents and engage CNS structures integral to the modulation of pain. Electrical stimulation of vagal afferents produces biphasic modulation of spinal nociceptive reflexes and of spinal nociceptive transmission, effects that descend from the brainstem and are mediated by spinal monoamine and opioid receptors. The studies also have revealed that intravenous morphine-produced antinociception and inhibition of spinal nociceptive transmission is mediated in part by vagal afferents. In the aggregate, the studies reviewed document a role for vagal afferents in pain modulation and suggest that these "visceral" afferents are an integral component of endogenous pain control systems.

Resumen: Respuestas a irritación aferentes vagales, respuestas de enfermedad.

Cualquier patógeno como virus, bacterias, isquemia, lesion tisular o atrapamiento neural activa al macrófago (en un hígado, un corazón, un pulmón, un músculo, la piel,…), el cual libera citokinas como TNFalfa e IL -1 que, al margen de activar la respuesta inflamatoria neutrofílica y fibroblástica activan aferentes vagales que activan neuronas secundarias en el núcleo del tracto solitario, lo cual activa respuestas de enfermedad como hiperalgesia, dolor (por activación del núcleo paraventricular del hipotálamo y Sistema límbico) y facilitación medullar descendente. (*After Goehler et al. 2000).*