

SISTEMA NERVIOSO AUTÓNOMO: DOLOR SOMÁTICO Y SNV

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 vágales 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 hiperalgesia a la sobrecarga mecánica (debido a la sensibilización de nociceptores) en tejidos a distancia vía sistema nervioso simpático-suprarrenal y el eje hipotálamo-hipofisario-suprarrenal.



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

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

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.

LA VAGOTOMÍA PRODUCE HIPERALGESIA

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

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Abstract

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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.



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¿¿¿Y estimular el vago, es analgésico??? si.



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Vagal modulation of nociception

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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.

La estimulación eléctrica de las aferentes abdominales vágales inhibe la transmisión de impulsos nociceptivos en las astas posteriores de la médula y deprime la nocicepción.

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

Cualquier patógeno como virus, bacterias, isquemia, lesión tisular o atrapamiento neural activa al macrófago (en un hígado, un corazón, un pulmón, un músculo, la piel), el cual libera citoquinas como TNF e IL -1 que, al margen de activar la respuesta inflamatoria neutrofílica y fibroblástica activan aferentes vágales 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 medular descendente. After Goehler et al. (2000).



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