



# Pain: Is It All in the Brain or the Heart?

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## Abstract

**Purpose of Review** Scientists have reported that pain is always created by the brain. This may not be entirely true. Pain is not only a sensory experience, but also can be associated with emotional, cognitive, and social components. The heart is considered the source of emotions, desire, and wisdom. Therefore, the aim of this article was to review the available evidence about the role of the heart in pain modulation.

**Recent Findings** Dr. Armour, in 1991, discovered that the heart has its “little brain” or “intrinsic cardiac nervous system.” This “heart brain” is composed of approximately 40,000 neurons that are alike neurons in the brain, meaning that the heart has its own nervous system. In addition, the heart communicates with the brain in many methods: neurologically, biochemically, biophysically, and energetically. The vagus nerve, which is 80% afferent, carries information from the heart and other internal organs to the brain. Signals from the “heart brain” redirect to the medulla, hypothalamus, thalamus, and amygdala and the cerebral cortex. Thus, the heart sends more signals to the brain than vice versa. Research has demonstrated that pain perception is modulated by neural pathways and methods targeting the heart such as vagus nerve stimulation and heart-rhythm coherence feedback techniques.

**Summary** The heart is not just a pump. It has its neural network or “little brain.” The methods targeting the heart modulate pain regions in the brain. These methods seem to modulate the key changes that occur in the brain regions and are involved in the cognitive and emotional factors of pain. Thus, the heart is probably a key moderator of pain.

**Keywords** Brain · Emotion · Heart · Hurt · Pain

## Abbreviations

CNS central nervous system  
HRV heart rate variability  
VNS vagus nerve stimulation

## Introduction

Current pain research involves the slogan “No brain, no pain.” Professor Lorimer Moseley [1] reported that “Pain is always – 100% of the time – created by your brain in an attempt to make you do something to protect your body.” There is

considerable evidence regarding the role of the central nervous system (CNS), particularly higher levels of the brain, in processing innocuous information and pain [2]. However, current evidence has shown the role of the heart in the modulation of acute and chronic pain [3, 4]. Most research has focused on the heart and the responses of the human body to the commands of the brain. Few scientists have examined the signals that the heart sends to the brain and response of the brain to these signals. Therefore, this paper proposes the statement “No heart, no hurt,” meaning that the heart is a key moderator of pain. The following discussion is intended to support this argument. Therefore, the aim of this article was to review the available evidence about the role of the heart in pain modulation.

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## Pain Definition

The International Association for the Study of Pain defines pain as:

*an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.* [5]

Many people report pain, particularly chronic pain, in the absence of any pathophysiological cause or tissue damage. Pain has several dimensions: sensory, emotional, cognitive, and cultural [5]. Several studies demonstrated that chronic pain patients display key changes in several brain regions, particularly the regions involved in cognitive and emotional factors of pain. These changes include the pain neuromatrix, structural and functional changes in the brain, and alteration of descending antinociceptive pathways [2]. Cognitive and emotional factors critically influence pain perception [5]. The heart plays a major role in creating emotions. It was recently shown that the cardiovascular system and the heart send more signals to the brain than the brain sends to the heart (see below) [4••].

### “Heart Brain”

In many cultures, such as Hebrew, Christian, Chinese, Hindu and Islamic, the heart is considered the source of emotions, desire, and wisdom [6]. Dr. J. Andrew Armour, in 1991, first introduced the concept of “heart brain,” “little brain,” or “intrinsic cardiac nervous system.” This “heart brain” is composed of approximately 40,000 neurons that are able to sense, feel, learn, and even remember. These neurons in the heart are alike the neurons in the brain [6]. Cardiac afferent neurons are located in both the dorsal root and nodose ganglia as well as in the intrinsic cardiac and intrathoracic ganglia. The intrinsic cardiac neurons can generate spontaneous activity independent of inputs from central and other intrathoracic neurons [7]. Thus, the heart has its own nervous system. In this perspective, the heart is a system that is complex and self-organized. It maintains a constant two-way communication with the brain and the whole body [7].

### Heart-Brain Communication

Literature has demonstrated that the heart communicates with the brain in many methods: through nerve impulses (neurologically), via hormones (biochemically), through pulse waves (biophysically), and through electromagnetic fields (energetically) [4••]. In comparison to the brain, the heart produces 40–60 times more electrical power and 5000 times more electromagnetic power. Consequently, the heart can align and synchronize all body systems to create physiological coherence [6]. The heart produces and secretes several hormones.

For example, atrial peptide or atrial natriuretic peptide, which inhibits the release of stress hormones, reduces sympathetic outflow and influences motivation and behavior [8]. Moreover, the main source of the brain natriuretic peptide is the cardiac ventricle rather than the brain. The heart also manufactures and secretes oxytocin, the so-called “love” or “social bonding” hormone, which is involved in cognition, tolerance, trust, etc. [8].

The vagus nerve is the tenth cranial nerve, which carries information from the heart and other internal organs to the brain. It terminates in the brainstem, particularly in the medulla, and solitary nucleus [8]. The vast majority (80%) of the vagus nerve fibers are afferent (ascending). This means, as previously mentioned, that the heart sends more signals to the brain than vice versa [4••]. Interestingly, signals from the “heart brain” transmit to the head brain via afferent neurons in the spine as well as the vagus nerve, where the signals redirect to the medulla, hypothalamus, thalamus, amygdala, and then to the cerebral cortex [4••]. There is evidence that a pathway from the dorsal vagal complex and cardiovascular afferent signals travels directly to the frontal cortex [8].

### Emotions and the Heart

As previously explained, pain experience is not only sensory but also emotional, cognitive, and cultural. Alterations in the forms of afferent input to the brain result in significant changes in emotion, cognition, perception, behavior, and physiological function. The heart sends afferent input that influences the homeostatic control areas in the brain. Interestingly, this input from the heart also affects the activity of higher brain areas comprised in the processing of emotion, cognition, and perception [8]. Recently, research at the Institute of HeartMath has shown that stress and emotions are highly reflected by heart rate variability (HRV), which is a product of the dynamic interaction of several systems of the body. Beat-to-beat variability in both heart rate and blood pressure is mainly produced and augmented by the interaction between the heart and brain. This is demonstrated by increased vagal afferent information sent from the cardiovascular system and heart to the brain. In addition, this interaction is mediated by afferent and efferent neural pathways of the autonomic nervous system through its sympathetic and parasympathetic branches. Emotion and physiological synchronization is directly related to heart rhythm, rather than the rate. Positive emotions like appreciation result in more coherent heart rhythms—a stable form of repeating sine waves. On the other hand, heart rhythms associated with negative emotions such as anxiety, worry, and frustration are more incoherent—highly variable and erratic. This indicates less synchronization

in the alternate action of the parasympathetic and sympathetic nervous systems [4••, 8].

## Pain and the Heart

Research on humans and animals has demonstrated that pain perception is modulated by neural pathways and methods targeting the heart. Recently, Frangos et al. [3•] reviewed 23 studies that investigated invasive and non-invasive vagus nerve stimulation (VNS). The authors concluded that VNS has positive effects on mechanical and/or thermal pain perception and psychological factors associated with pain experience in patients with various conditions such as low back pain, chronic pelvic pain, fibromyalgia, and headache [3•]. The aforementioned findings are consistent with other studies that used heart-rhythm coherence/HRV feedback techniques. Heart-rhythm coherence feedback aims at facilitating skill acquisition of self-regulation to develop a greater awareness of the connection between emotions, physiology, and behavior [4••]. The HeartMath Institute uses these techniques to reduce patients' pain, often to the extent that they can minimize the use of pain medications. Similar to VNS, these techniques have proven effective in patients with numerous conditions, including visceral, cutaneous, and chronic musculoskeletal pain; fibromyalgia; serious burns; and traumatic brain injury [4••, 8].

Several mechanisms may be referred to the vagal modulation of pain. In a review, De Couck et al. [9] attributed the influence of vagal modulation of pain to five mechanisms: inflammation, sympathetic nervous system, oxidative stress, brain activity, and opioids. The vagus nerve in general can inhibit inflammation, especially in a pain condition. Afferent input from VNS can modulate sympathetic input and stress via modulating descending noradrenergic and serotonergic neurons and, consequently, reduce pain. Vagal nerve activity may inhibit oxidative stress, which occurs when there is an imbalance between prooxidants and antioxidants and contributes to pain. Interestingly, VNS can alter the activity of the pain neuromatrix of brain areas that are commonly active during pain. These regions include, but are not limited to, the periaqueductal gray, hippocampus, amygdala, thalamus, putamen, insula, primary somatosensory, prefrontal cortex, and anterior cingulate cortex. Moreover, studies on animals and humans have revealed that VNS modulates the ascending and descending pain inhibitory pathways, primarily the spinothalamic tract, by activating the solitary nucleus, raphe nuclei, locus coeruleus, and nucleus cuneiformis [3•]. The vagus nerve may modulate pain via the opioid and cannabinoid receptors present on the sensory fibers of this nerve [9]. De Couck [9] postulated that the triggers of pain (namely, psychological stress, inflammation, and tissue injury) change

activation of the vagus nerve, which consequently modulates any of the aforementioned five mechanisms and potentially increases the excitability of the CNS. It has been demonstrated that left VNS inhibits approximately 60% of the spinothalamic cells [8].

## Conclusion and Future Recommendation

The recent scientific evidence revealed that the heart is not just a pump, has its neural network or “heart brain,” and is the most powerful system that generates signals in the human body. In addition, the methods targeting the heart modulate the perception of pain. These methods seem to modulate the key changes that occur in the brain regions and are involved in the cognitive and emotional factors of pain. Therefore, the proposition of the current article “No heart, no hurt” may be plausible to emphasize that the heart is a key moderator of pain, irrespective of the role of the brain in the modulation of pain. Although there appears to be growing foundation to consider this proposition, further clinical trials with robust methodologies are needed.

## Compliance with Ethical Standards

**Conflict of Interest** The authors declare no conflicts of interest relevant to this manuscript.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

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