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Pain-Related Responses

Abubakar Imam*

Department of Physical and Rehabilitation Medicine, Algarve Hospital Center, R. Leão Penedo, Faro, Portugal

Introduction

The sympathetic nervous system (SNS), neuro-endocrine system, and immunological system, as well as emotions, all respond to pain through complex and linked physiological mechanisms.

Description

Sympathetic Nervous System

The SNS is involved in the body's rapid response to emergencies, such as severe and acute pain; the 'fight or flight' response is the body's response to pain or panic. The SNS causes brainstem cells that govern descending pain mechanisms to discharge noradrenaline, serotonin, and endogenous opioids into the dorsal horn when it is activated.

Because sympathetic nerves have stimulating effects on the heart (enhancing circulation) and respiratory system, the SNS is concerned with the control of vascular tone, blood flow, and blood pressure (increasing oxygen intake). As a result, pain raises heart rate, blood pressure, and breathing rate. Ischemic damage can occur if these physiological responses are sustained, especially in a person with low physiological reserves [1].

The SNS also inhibits digestion by lowering or stopping the secretion of digestive enzymes in the alimentary canal and peristaltic activity in the gut wall. As a result of the pain, the body's capacity to digest food is harmed, which can result in nausea, vomiting, or constipation [2].

Neuro-Endocrine System

The pituitary gland, located near the base of the hypothalamus, connects the endocrine and neurological systems. Some of the body's pain responses are mediated by the nervous and endocrine systems, primarily through the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathomedullary pathway, and involve the release of mediators like cortisol, adrenaline, and noradrenaline, as well as growth factors and cytokines.

Adrenaline, noradrenaline and cortisol

Pain increases the creation of corticotrophin-releasing hormone (CRH) in the hypothalamus, which is then transported to the anterior pituitary gland, where it activates the SNS and stimulates the production of adrenocorticotrophic (ACTH). The SNS also causes the adrenal medulla to secrete adrenaline and noradrenaline, both of which have different effects.

ACTH travels through the bloodstream to the adrenal cortex, where it stimulates the production of cortisol, which mobilises glucose and functions as an anti-inflammatory by suppressing prostaglandin formation [3]. Cortisol levels in the blood act as a feedback mechanism to the hypothalamus, preventing excessive release.

When this process is working properly, it decreases discomfort and prevents the inflammatory response from spiralling out of hand. Long-term pain and stress, on the other hand, can impair the body's ability to control inflammation. The continual synthesis of cortisol under long-term stress and/or pain causes glucocorticoid receptor resistance. As a result, input to the hypothalamus is disrupted, and cortisol loses its ability to modulate inflammation. Inflammatory mediators are found in increased concentrations in the blood of some persons with chronic pain, which can lead to despair, anxiety, and sleep issues [4].

Growth hormone

Growth hormone (GH), which is produced by the anterior pituitary gland, has a direct impact on cellular activity as well as protein, carbohydrate, and fat metabolism. Pain causes a rise in GH secretion, which leads to an increase in blood glucose levels and insulin resistance [5]. Muscle weakness and exhaustion are indications of a GH shortage, which are also symptoms of the pain syndrome fibromyalgia. GH levels have been discovered to be lower in people with fibromyalgia, and GH treatment has improved pain and quality of life [6].

Cytokines

A range of peripheral cells local to the lesion (including macrophages, fibroblasts, and monocytes) as well as cells in the dorsal horn of the spinal cord and brain generate cytokines in response to injury and pain (glial cells). Tumour necrosis factor alpha (TNF), nerve growth factor (NGF), interleukin 6 (IL-6) and interleukin 1 beta (IL-1) are all pro-inflammatory cytokines. Interleukin 10 (IL-10) and interferon alpha (IFN) are anti-inflammatory cytokines.

TNF and IL-1 sensitise sensory nerve terminals and stimulate the generation of unpleasant mediators shortly after an injury (for example, substance P). They promote the production of pain neurotransmitters (such as substance P, CGRP, and glutamate) in the spinal cord and increase the number of receptors for these molecules on secondary neurons.

TNF and IL-1, on the other hand, decrease the activity of cells that help to suppress pain (interneurons that produce GABA and glycine). As a result, they play a significant role in pain amplification. Pro-inflammatory cytokines also stimulate the hypothalamus, stimulating the HPA axis and generating fever; however, anti-inflammatory cytokines like IL-10 and IFN counteract this effect. This is a complicated relationship that is influenced by the pain's circumstances [7].

Conclusion

The first clinical trials of anti-TNF medications were undertaken in the 1990s, and blocking the activity of pro-inflammatory cytokines can

***Corresponding author:** Abubakar Imam, Department of Physical and Rehabilitation Medicine, Algarve Hospital Center, R. Leão Penedo, Faro, Portugal, E-mail: imam_a12@gmail.com

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have profound impacts on pain. Monoclonal antibodies and biologics have been demonstrated to be effective in the treatment of a variety of painful disorders [8,9] - a significant step forward in the treatment of difficult pain.

Acknowledgement

None

Conflict of Interest

None

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