IJAYUSH
International Journal of AYUSH
AYURVEDA, YOGA, UNANI, SIDDHA AND HOMEOPATHY
http://internationaljournal.org.in/journal/index.php/ijayush/

International Journal
Panacea
Research library
ISSN: 2349 7025

ReviewArticle | Volume 9 Issue 4 | Oct – Dec 2020

A REVIEW ON MODE OF ACTION OF AGNIKARMA BY VIRTUE OF PAIN MODULATION THEORY

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Abstract:

Many disorders like *Ghridhrasi* (Sciatica), *Avabahuk* (Frozen Shoulder) etc.*Agnikarma* found to be effective in relieving pain. Unfortunately, due to lack of scientific validation in its mechanism of actionof this effective therapeutic technique, it is very difficult to convince the patient for it. Although, Ayurveda therapy is extremely effective; appropriate mode of action, pharmacokinetics, pharmacology, and pharmacovigilance of several important Ayurvedic parasurgical procedures are still not fully discovered. Hence, literary research which is the base for the applied research is highly needed for global recognition and acceptance of Ayurveda. As physicians, whether serving individual patients or populations, we always have sought to base our decisions and actions on the best possible treatment. According to Sushruta, the *Agnikarma* is stated as more important than *Ksharkarma, Bheshaja* and *shastrakarma* becauseofnon-recurrenceofdisease.Buttoday*Agnikarma*seems tobemostneglectedpartoftreatment, soitshouldbehighlighted withproperscientificdirectioninthismoderneraoflifesciences. The purpose of the following literature study is to assimilate the literature on the neurobiological pathways within the central, autonomic, and peripheral nervous systems that mediate pain processing, and discuss how thermal stimulation interact with anatomy and physiology to modulate the pain.A greater understanding of the mechanism of action of ***Agnikarma*** through "**Gate control theory**", provides clue to get therapeutic action of Agnikarma against chronic pain state.

Key Words: Agnikarma, Mechanism of action, pain modulation and gate control theory

INTRODUCTION

All superlative Medical Sciences with several principles and fundamentals are trying their best to achieve Health for community. To attain this goal the science must be able to treat the ailment and that to be without any side effects. Many therapeutic procedures have been suggested by ancient texts in the management of each and every disease. But their effectiveness needs to re-establish by systematic and intensive studies.

Agnikarma found to be effective in many disorders but how it exactly works is the main question. In this era of evidenced based medicine, we have to prove the mode of action of *agnikarma* in the scientific language. It is the need of an hour to convince the patient for it, to avoid the jeopardy of analgesic drugs. For understanding the mode of action of *agnikarma* the knowledge of pain pathway and pain modulation pathway is essential. Human body is the most conspicuous thing made by the nature and the most appreciable system in human body is analgesic system in our body. Though it is very complicated but it can give us answer of the question i.e. how Agnikarma exactly work? It shows how excellent is our science of Ayurveda Agnikarma.

According to Sushruta, *Agnikarma* is stated as more important than *ksharkarma* [burning with alkalies], *bheshaja* [medications] and *Shastrakarma* [surgery] because of non-recurrence of diseases.[1]But today *agnikarma* seems to be more ignored part of treatment, so it should be highlighted with scientific direction in this modern era of life sciences. The purpose this review is to amalgamate the literature on the neurobiological pathways within nervous system that intercede pain processing, and converse about interaction of thermal stimulation with physiology to control the pain. A better understanding of the mechanisms that transduce and transmit neural processes of encoding and processing noxious stimuli i.e. Nociception, also those that basis of the endogenous pain modulatory systems, expectantly will indicate the advancement of new therapeutic molecules against chronic pain conditions.

AIMS & OBJECTIVES

Aim:

To find the mode of action of *Agnikarma* with help of Pain Modulation Theory.

Objectives:

1. To understand the pain modulation theory.
2. To know the mode of action of *agnikarma*.

MATERIALS AND METHODS

This is a literary research.

ATERIAL:

1. Literature of *agnikarma*.
2. Literature of pain pathway and pain modulation pathway.

LITERATURE OF AGNIKARMA

***Agnikarma* Definition:**

The word *Agnikarma* comprises of two words i.e. *Agni*(fire) and *karma*(procedure); collectively give the meaning procedure performed with the help of *fire or heat*.

Agnikarma, a para-surgical procedure has attained importance as a treatment for several complex diseases during the period of Sushruta. Sushruta, father of surgery has earmarked the *Agni* as supreme in all the para surgicalprocedures.[1]A separate chapter in text Sushrut Samhita having details about every aspect of *Agnikarma* denotes its importance in the treatment, during that period. A number of diseases and conditions have been explained in text where *agnikarma* as therapeutic measure has been indicated and in many disorders like *Gridhrasi, Avabahuk, shiriroga* etc.[2, 3, 4]*Agnikarma* found to be very effective to relieve pain.

Literature of pain pathway [5]

Pain is undoubtedly the utmost ultimate and primal sensation. To know the mode of action of Agnikarma we should know following things-

Pain

Pain is a distressful sensory and emotive experience accompanying with actual or possible tissue injury, or defined in terms of such impairment.[6]

When harmful impetuses intervene upon the body from internal or external sources, information regarding the damaging impact of these stimuli on bodily tissues is

transduced using available neural pathways and transmitted via peripheral nervous system toward the central and autonomic nervous systems. This process of transmitting information is known as nociception.

Pain sensation is transmitted through three stages-

1. First order neuron
2. Second order neuron
3. Third order neuron

First order neuron:

Nociceptive fibres (fibres that carry pain sensation) have free nerve endings. These available nerve endings unite to form a dense network having multiple branches that are regarded as nociceptors that is the sensory receptors for pain. Pain sensations are transmitted by two types of fibres which form first order neuron in pain pathway.[7]

1. **Slow fibres i.e. DRC (Dorsal Root C) Fibres-** unmyelinated and having slow rate of conduction i.e. 0.5-2 m/sec, diameter of 0.4-1.2 μm.[8]
2. **Fast fibres i.e. Aδ Fibres-** Myelinated, having fast rate of conduction i.e. 12-30 m/sec, diameter of 2-5 μm.[9]

According to the conduction velocity pain has been classified into two types-

1. **Slow pain** carried by **DRC (dorsal root C) Fibres**
2. **Fast pain** carried by **Aδ Fibres**

These first order neuron i.e. primary afferent fibres terminate in dorsal horn of spinal cord and enter the spinal cord through dorsal roots of spinal nerves. Dorsal root fibres contain numerous molecules, which are either known or suspect, to fulfil a neurotransmitter or neuromodulator like glutamic acid, substance P etc.

Second order neuron:

Spinothalamic tract consists of second order neurones which convey pain sensation to the somato-sensory region of thalamus.

The dorsal root fibres (axon of first order neuron) of all the segments after entering the spinal cord end around the cells of substantia gelatinosa of Rolando. The second order neuron from these cells crosses in the anterior white commissure, obliquely to the

opposite side of the same segment and ascends in the lateral white column form spinothalamic tract.

The lateral spinothalamic tract is cited in the lateral funuculus (lateral white column), lying medially to the ventral spinocerebellar tract.

These neurons respond maximally to the noxious, mechanical and thermal cutaneous stimuli. On reaching the lower brain stem the lateral spinothalamic tract continue as a spinal leminiscus. The spinal leminiscus continue in the medulla and pons. In the midbrain, fibres in the spinal leminiscus conveying pain and temperature sensation from lower limb extend dorsally, while those from the trunk and the upper limb are more ventrally placed.

The spinothalamic tract neurones are divided into three separate groups on the basis of laminar site, functional properties and specific thalamic termination they are as follows

1. Lamina I- apical cells of dorsal grey column.
2. Lamina IV-VI- deep dorsal column cells
3. Lamina VII-VIII- cells in ventral grey column

LaminaI spino-thalmic tract receives input from **Aδ and C Fibres.** LaminaI spino-thalmic tract neurons projects preferentially to the ventral posterolateral nucleus of thalamus, with limited projection to centro-lateral or mediodorsal thalamic nuclei and at higher brain stem level send several collators into reticular formation (spino-reticular pathway) and tegmentum before ending in the thalamus.

Third order neuron:

Third order neurones start from thalamus and axons of these neurons terminates in the postcentral gyrus of the cerebral cortex through the posterior limb of internal capsule.

Pain modulation pathway:

The activity of spinothalamic tract neurons may be selectively modulated by the pathway descending from the brain to spinal cord. The areaspersuading such effects correspond to a number of midbrain and rhomben-cephalic nuclei in the brain stem which, with their connections, constitute an **endogenous analgesic system.**

The endogenous analgesic system consists of –

In midbrain – periaqueductal grey matter (PAG), dorsal raphe nucleus

In rhombencephalon- nucleus raphe Magnus and medial reticular column i.e.

Neurons in these sites contain serotonin (5-HT), ϒ- aminobutyric acid (GABA), encephalin and dynorphin.

Gate control theory:

The 'The Gatecontrol Theory'(Melzac and Wall 1965) proposed a mechanism of modulation of information along nociceptive afferent pathway.[10]The theory suggests that the high frequency volley of impulse from large **Aδ fibres close the gate for pain transmission.**

The implementers of this pathway are identified as **neurotransmitters.** Endogenous chemical messenger's i.e. Neuro-transmitters disseminate signals across a chemical synapse, from one neuron to another "target" neuron, muscle cell, or gland cell. Some of them are inhibitory neurotransmitters, obstructing the transmission, while others are excitatory, simplifying transmission of messages. These chemical communications are vital in the modulation of pain.

V anilloid receptor (VR-1, TRPV1) is situated on the free nerve endings of both C and Aδ fibres which reacts to raised levels of heat (>43 °C).

Chemicals associated with pain mechanism are:

Substance P [SP] is intricated in sensory and, especially in nociceptive pathways. In the periphery, substance P has been identified in **C-type sensory nerve endings** and is a neuropeptide that acts **as a mediator of pain** transmission in the central nervous system and during neurogenic inflammation in the periphery. SP carries the sense of pain by secretions from nerve, exudates from inflammatory cells. It acts by affixing to neurokinin-1 receptors (NK-1R) which arepositioned on the nociceptive neurons on unmyelinated primary afferents, known as C fibres, to the dorsal horn of the spinal cord.

Glutamed pass on the pain sensation by binding to 5-subunit ligand-gated ion channels situated on nociceptive neurons on **myelinated pain afferents A-delta [belonging to the class of afferent axons]** that conduct to the dorsal horn of the spinal cord.

Glutamate is generally involved in the fast neurotransmission of acute pain, such as with mechanical stimuli or temperature stimuli producing quick, sharp pain.

Means neurotransmitter for slow pain is **'substance P'** and for fast pain is **'Glutamed'**. This Glutamed stimulate transmit the pain quickly to the cortex and stimulate the periaqueductal grey matters in midbrain through spinomesencephalic, spinoreticular pathway. And stimulate **endogenous analgesic system** which sends **efferents to the sunstantia gelatinosa of Rolando and close the gate for pain neurotransmitters by secreting** neuromodulator **sretonin(5-HT), ϒ- aminobutyric acid (GABA)**which secrets opioids receptors **like encephalin, enkaphaline and dynorphin.** These opiates inhibit the neurotransmitters like **Substance P** in peripheral nervous system and block the pathway for pain through spinothalamic tract.

This gate theory is the answer for the mode of action of Agnikarma. *Agnikarma* stimulate **Aδ fibers which carry** fast pain stimulus and activate endogenous analgesic system and close the gate for dull pain carried by the Substance P through Small C-fibres and resulting in relieving pain in the patients.[11, 12]

CONCLUSION:

Pain is a Subjective feeling, precision of intensity is difficult to generalize.[11][12] *Agnikarma* act over cutaneous receptor and first cause sharp pain through ascending pain pathway and activate the **descending** pathway i.e. **endogenous analgesic system (**periaqueductal grey matter(PAG), dorsal raphe nucleus, nucleus raphe Magnus and medial reticular column) which involves various chemicals i.e. neurotransmitters which are the basis of **Gate Control Theory**.

Exogenous and endogenous opioids from central or peripheral termini of nociceptive afferent fibres comprise opiate receptors may act to modulate the capacity to transmit nociceptive information. Opiate receptors are concentrated in peri-aqueductal gray (PAG), dorsal raphe (DR) in the rostral ventral medulla, and nucleus raphe magnus (NRM).

Thus, Agnikarma act same like that of opioids analgesic drug and that too without any adverse effect and is also cost effective.

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