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ORO FACIAL PAIN -AN OVERVIEW

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ABSTRACT

The article is an overview of the current concepts regarding the properties and behavior of pain. Pain is now being recognized as a subjective feeling more than a mere physical sensation. It is no longer viewed as a single sensory event signaling tissue damage. Thus, simultaneously the role of psychological factors in the experience of pain has also been highlighted to apply this knowledge to oral pain conditions.

Pain is a complex perception that differs enormously among individual patients. It is for this reason why the word 'pain' has not been defined in a manner agreeable to all, despite its long history. The understanding of pain, to its fullest, requires an adroit clinician, one who is cognizant in the integration of neuroanatomy, psychology and pharmacology. A broader goal is to further the dentist's understanding of physiology of pain to aid in the diagnosis of chronic orofacial pain and its successful management.

Keywords: Nociceptor; Orofacial pain; Odontalgia; Biopsychological.

INTRODUCTION

There are several difficulties in understanding the current concepts of pain. There are numerous research works being published with such a wide array of information available, sometimes conflicts in opinions do arise. In addition there are many theories which are vague and unfamiliar to clinicians regarding the mechanisms of pain.

As said by **Sir William Osler:-**

"**What You Don't Know – You Won't Diagnose**"

Thus, the purpose of this review is to summarize the understanding of physiology of pain and the role of psychological factors in pain perception to aid clinicians in the successful diagnosis.

Pain is the cynosure of basic and clinical sciences. Every individual has experienced pain in some form or the other in their lifetime. It is that one symptom which brings even the most reluctant patient to a doctor. Pain even in its benign form warns an individual that something is wrong, and that he/she should take medications or see a doctor. So, it is spoken as also having a protective mechanism. As dentists, we assume a great responsibility in the management of pain in the orofacial region. Failure to do so will lead a patient to skepticism and fear to return. Therefore, a successful clinician would be one who can relieve pain by administering treatment with least amount of trauma.

Historical perspective:

The word pain is derived from the name of the Greek goddess of revenge "Poine" (Poine in Greek means a fine or penalty). Aristotle was the first to distinguish the five physical senses, and he considered pain to be "Passion of Soul" that results from intensification of other sensory experience.[1]

In the Middle Ages and Renaissance evidence began to accumulate on the sensory component of pain. Leonardo Da Vinci in the 15th century developed the idea that brain is the central organ responsible for sensation. He also developed the idea that spinal cord transmits pain sensations.

In 1664 the French philosopher René Descartes described what to this day is called a "Pain Pathway". He illustrated how particles of fire, in contact with the foot, travel to the brain and he compared pain sensation to the ringing of a bell[1]. However, in the 19th century, pain came to dwell under a new domain – science paving the way for advances in pain therapy. As the 21st century unfolds we have seen tremendous advances in pain research and a future that includes a better understanding of pain, along with greatly improved treatments to keep it in check.

Definitions of Pain:

No simple definition of the word 'pain' will be acceptable to all nor is any definition likely to be an endearing one. Rapid developments and discoveries in the field of pain research will mandate periodic updating and redefinition.

Dorland's Dictionary defines pain as "A more or less localized sensation of discomfort, distress or agony resulting from the stimulation of specialized nerve endings. It serves as a protective mechanism as it induces the sufferer to remove or withdraw from the source".

This definition implies to say that pain is a protective mechanism against injury and being external to the body, the presumed noxious agent could be avoided by proper evasive action. Evidence from the clinic and laboratory demonstrates that pain cannot be divided simply into either structural (organic) or psychological (non organic) components. Current models view pain as a complex experience that by its nature includes psychological components.

Pain is now being defined by the International Association for the Study of Pain (IASP), as "unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage"[2,3].

It is clear from this definition that pain represents a subjective psychological state rather than an activity that is induced solely by noxious stimulation. It should be noted that, if the subject regards his / her experience as pain, it should be accepted as pain. This definition is currently accepted as it incorporates the physical and psychological aspect of pain.

Changing Concept of Pain:

Pain is now being recognized as being more of an experience than only a sensation. The sensory dimension, registers the nature of the initiating stimulus including its quality, intensity, location and duration.

The three sensory dimensions of pain are:

1) Cognitive
2) Emotional
3) Motivational

Pain perception is therefore the sum of the interactions between psychological and physical functions communicated by the "Biopsychological Model of Pain" [4] (WHO) (Fig 1).

Neuroanatomy of a Pain Pathway:

Structural unit of a pain pathway is a nerve cell. Afferent and efferent conducting impulses from this neuron travel towards central nervous system, relaying the pain sensation. Sensory receptors are the distal terminals of afferent nerves, specialized receptors that respond to physical and chemical stimuli are located. Once

these receptors have been adequately stimulated, an impulse is generated in the primary afferent neuron that is carried centrally into the CNS. Sensory receptors are specific for certain types of stimuli. They can be classified into three main groups as follows[5].

1. Exteroceptors

These are sensory receptors that are stimulated by the immediate external environment and are appropriately fashioned and located so as to be exposed to the organism's environment. These receptors provide information from the skin and mucosa (the envelope). Some examples of this type of receptor are:-

a. Merkel's corpuscles: Tactile receptors in the sub mucosa of tongue and oral mucosa.
b. Meissner's corpuscles: Tactile receptors in skin.
c. Ruffini's corpuscles: Pressure and warmth receptors.
d. Krause's corpuscles or end-bulbs: Cold receptors.
e. Free nerve endings: Perceive superficial pain and touch.

2. Proprioceptors

These are the sensory receptors that provide information from the musculoskeletal structures concerning the pressure, position and movement of the body. Most part sensations conducted by there are below conscious levels. Examples of these are muscle spindles, golgi tendon organs, pacinian corpuscles, periodontal mecahnoceptors and free nerve endings.

3. Interoceptors:

These are receptors that are located in, and transmit impulses from the viscera (supply system) of the body. Sensation from these receptors, for the most part are involved in voluntary functioning of body and is below conscious level.

Examples are pacinian corpuscles for pressure sensation and free nerve endings.

Peripheral and Central Mechanisms of Nociception

Nociception, in other words pain perception involves peripheral and central pathways and relayed by receptors that respond to harmful (noxious) stimuli .They are called as **nociceptors**[6]. The most conspicuous part of the body with no receptors is brain. Morphologically they are free nerve endings. They differ with respect to their axons are (A-fibres) myelinated or unmyelinated and their responsiveness to particularly forms of noxious stimuli. These nociceptors are present within a receptor field which is an area of the skin or its equivalent part from where information is gathered by single receptor. Two types of nociceptors are seen mainly:

The peripheral mechanism of pain is a complex interplay of nociceptors and inflammatory mediators causing pain. Damaged tissue release a cocktail of chemicals, the inflammatory agents that have vascular and neural effects. The vascular effects produced are vasodilation (causing color and rubor) and increased vascular permeability (causing tumor). Neural effects, involve either activation or sensitization of nociceptors, thus relate to pain (Dolor). These agents include Potassium ions, hydrogen ions, serotonin, prostaglandins, neuropeptides, adenosine, nor adrenalin and various other cytokines.

Nociceptor Types[7]:

1. A- Fiber nociceptors:

All myelinated nociceptor may be categorized as A-delta fibres, i.e. the thinnest myelinated nerves. Depending on the kind of stimuli they respond to the A – delta nociceptors are divided into 3 types: A - δ mechano - nociceptors – respond only to noxious mechanical stimuli.A - δ polymodal – nociceptors – respond to all types of noxious stimuli i.e mechanical, thermal and chemical. Other A δ nociceptors – respond only

to cold, or to hot and chemical but not mechanical noxious stimuli has also been reported.

2. C-Fiber nociceptors:

Most of the unmyelinated nociceptors are also polymodal, responding to strong mechanical stimuli, intense heat or cold and various pain-producing chemicals. C-fiber polymodal nociceptors are the most numerous and arguably the most important nociceptors in the human body.

Diameter 0.5 – 1 μm	Velocity 0.5 – 2 m/s
Pain → Aδ	and C-Fibres
↓	↓
Rapid, pricking pain (1^{st} PAIN) [8]	Slow,burning pain (2^{nd} PAIN) [8]

Receptors can be specialized organs or structures or can be just bare nerve terminals. Pain receptors are "free nerve endings" (bare nerve). They are described as naked, as the have no capsule surrounding them. They form a network that is especially dense in cutaneous layers of mucous membranes and periodontium. Since pain receptors respond to wide variety of stimuli, they are called as 'polymodal'. Fast pain receptors are those that are sensitive to mechanical and thermal stimuli of noxious strength. Whereas slow pain receptors are sensitive not only to noxious thermal or mechanical stimuli but also associated with a wide variety of chemicals associated with inflammation like serotonin, bradykinin etc.

Neural Pathways of Pain

Fields (1987)[9] described that the subjective experience of pain arises by four distinct processes: transduction, transmission, modulation and perception

1. Transduction

It is the process by which noxious stimuli lead to the electrical activity in the appropriate sensory nerve endings. These noxious stimuli can be in the form of thermal and mechanical stimuli, noxious chemicals, noxious cold, endogenous algesic chemical substances (inflammatory mediators). They are released in the body in response to tissue injury. The body has several types of sensory organs (receptors) that initiate process of nociception.

2. Transmission

This refers to the neural events that carry the nociceptive input into the CNS for proper processing. There are three basis components for transmission.

i) **Peripheral afferent nerve or first order neuron (Dubner and Bennett 1983)[10]**

These are cells in the posterior nerve root ganglia. These neurons receive impulses of pain sensation (nociceptive input) from pain receptors (sensory organ) through dendrites and their axons reach the spinal cord. These first order neurons include the A fibers- Aα, Aβ, Aγ, Aδ and C fibers. Each sensory receptor is attached to a first order or primary afferent neuron that will carry the impulse to central nervous system. Three classes of nociceptive afferent neurons provide the input whereby the brain perceives pain.

1. A – Delta mechano thermal afferents:

- They are primarily A – delta fibers that conduct at a velocity of 12 to 30 m/s and respond to intense thermal and mechanical stimuli.
- Provide high degree of discriminative information.
- Peculiar to primates.

2. C-Polymodal nociceptive

- Conduct much slowly at velocity of 0.5 m/s and respond to mechanical, thermal and chemical stimuli in all mammals. At this rate it takes an impulse 2 seconds to go from big toe to spinal cord.

3. High threshold mechanoceptive afferents (A - δ)

- Chiefly A – delta fibres respond to intense mechanical stimuli in all mammals.
- These can also be sensitized by alogenic substances or repeated noxious stimulation to respond to noxious heat also. Only the first two classes of nociceptive afferents normally respond to noxious heat.

4. Silent Nociceptor or sleeping afferents

- These are insensitive afferents normally unresponsive to transient excessive stimulation, but become sensitive to mechanical stimuli in presence of inflammation.

ii) Second order neurons (Okeson)[11]

The primary afferent neuron carrying impulse into the CNS synapses with the second-order neurons. This is also referred to as interneuron or transmission neuron since it transfers the impulse on to the higher centers. Synapse of primary afferent and second-order neuron occurs in the dorsal horn of the spinal cord. There are three specific types of second-order neurons that transfer impulses to higher centers. They are named according to the type of impulses they carry. Low threshold mechanosensitive neurons (LTM) transfer information of light touch, pressure and proprioception. Nociceptive specific neurons (NS) exclusively carry impulse related to noxious stimulation. Wide dynamic range neuron (WDR) respond to wide range of stimulus intensities ranging from non-noxious to noxious. Nociception is primarily carried by the nociceptive specific and wide dynamic range neurons. Under normal conditions, it is not thought that the low threshold mechanosensitive neurons are involved in the transfer of nociception.

iii) Third order neurons[11]:

There are the neurons of thalamic multi reticular formation, rectum and gray matter around the aqueduct of sylvius. Axons from these neurons reach the sensory area of cerebral cortex. Some fibers from reticular formation reach hypothalamus (limbic system). The third order neurons mostly constitute the ascending pathways like:

a. Neo-spinothalamic tract (fast pain)

b. Paleo spinothalamic tract (slow and chronic pain)

c. Trigeminal pathway (oro facial pain).

3. Modulation: It refers to the ability of the CNS to control pain transmitting neurons. This involves the descending Inhibitory systems in the brain. When the pain modulating system is active, noxious stimuli produce less activity in the pain transmission pathway. Several areas of the cortex and brain stem have been identified that can either enhance or reduce nociceptive input arriving by the way of transmitting neurons.

4. Perception:

This is the final step in subjective experience of pain. At this step the brain perceives the pain. When the nociceptive input reaches the cortex, perception occurs, which immediately initiates a complex interaction of neurons between higher centers of the brain. At this point suffering and pain behavior begins. This is least understood and is also variable aspect between individuals[12]. In a study done by Koyama T et al (2005)[13] it was observed that expectations of decreased pain profoundly reduce both the subjective experience of pain and pain-related brain activation.

Adequate stimulation of the primary receptors generates impulse in the primary afferent neuron that is carried centrally into the central nervous system.

Central Pain Mechanisms

Constitute nerve tracts ascending to brain; pain signals to brain take two different paths.

1. Neospinothalamic Tract – For fast pain, includes A-delta fibers that transmit acute pain. Nerve fibres terminate in lamina I (Lamina Marginalis) of dorsal horn of spinal cord.
2. Paleospinothalamic Tract – For slow pain, this is primitive tract, involves C-fibers. Relay chronic slow pain. Nerve fibers terminate in lamina II and III (Substantia Gelatinosa) of dorsal horn of spinal cord. Fibers terminate widely in brain stem –intra laminar nuclei. Only 1/10 th to 1/4 th of fibers pass all the way to thalamus, instead most of the fibers end in one of the three areas of the brain stem.

- Reticular Nuclei – of medulla, pons, and mesencepahalon.
- Tectal Area – of mesencephalon.
- Periaqueductal Gray Region – surrounding aqueduct of sylvius.

Trigeminal Pain Pathway:

Somatic impulses from face and oral structures do not enter spinal cord by way of spinal nerves. Instead they are carried by way of trigeminal nerve.

Clinical Significance of Trigeminal System[14]:

An appreciation of the arrangement of the trigeminal nociceptive system provides insight into the interesting pain and referral pattern that are encountered in the head and neck region.

The **nucleus caudalis** corresponds to the substantia gelatinosa of the rest of the spinal dorsal horn and is the most caudal portion of trigeminal spinal tract nucleus. The arrangement of trigeminal nerve fibers within this nucleus is significant and the fact that the nucleus descends as low as the third and fourth cervical vertebra (C 3-4) in the spinal cord. Fibers from all three trigeminal branches are found at all levels of the nucleus, arranged with the mandibular division highest and ophthalmic division lowest.

In addition, they are arranged in such a manner that fibers closest to the midline of the face synapse in the most **rostral** portion of the tract. The more lateral the origin of the fibers in the face, the more **caudal** the synapse in the nucleus. This "**Laminated**" arrangement helps to explain why maxillary molar toothaches may be perceived as pain in **mandibular molar** on the same side (referred pain) but not in an incisor[4].

Role of experience in referred pain:

Obviously, knowledge of referred pain and the common sites of pain referral from each of the viscera are of great importance to physician. Best known example, is referral of cardiac pain to the inner aspect of the left arm. Other examples in dentistry include, referred pulpal pain e.g. pain from maxillary premolars may refer pain to mandibular premolars. Experience also plays an important role in referred pain. Pain originating in the maxillary sinus is usually referred to nearby teeth, but in patients with a site of previous surgical operation, trauma, or localized pathological process, such pain is regularly referred to these previously traumatized teeth. This is true even when the teeth are a considerable distance away from the sinus. This phenomenon was discussed by **Ruch and Fulton** as Habit reference **[14].**

Pain Theories:

There were various theories put forth since the classical description by Rene Descartes in 1664. Specificity theory (Von Frey), Summation theory by Goldsheider 1894 and Sensory interaction theory by Noordenbos.[15] But the most widely accepted theory is Modified Gate Control Theory by Melzack and Wall 1965.[16,17]

According to this two factors regulate transmission of pain:

1. A gating control mechanism situated in a specific area of grey matter in spinal cord, called substantia gelatinosa. Pain impulses are regulated by opening and closing of the gate. Large diameter fiber when relaying an impulse closes the gate to pain.

Small diameter fibers relaying impulses open the gate to pain.

2. Descending control from the brain's intrinsic mechanism.

Clinical significance of gate control system (Weine) [18]

1. Relief of pain through rubbing massage techniques and application of ice packs.
2. Counter irritation.
3. Acupuncture, Transcutaneous electronic nerve stimulation (TENS)

Intentional stimulation of large diameter fibres (A-α) closes the gate to pain.

4. Higher Centre Modulaton

Gate control mechanism can be further modulated by intrinsic brain mechanisms and through emotional, psychic, visual as well as past learned experiences.

ORO FACIAL PAIN

Dentists should be skilled in treatment of acute orofacial pain because it often accompanies even meticulous clinical care. On the basis of differential diagnosis, the orofacial pain has been classified broadly into four groups [19]:

Typical Facial pain: Pain of extracranial origin, e.g dental, ocular, ear-nose-throat, salivary gland, temporomandibular joint, anginal pain, etc.

I. Primary Neuralgias: Trigeminal, glossopharyngeal, geniculate and post herpetic neuralgias
II. Secondary Neuralgias: Mental nerve neuralgia, causalgia, auriculotemporal nerve syndrome etc.
III. Atypical Neuralgias: Pain of vascular origin.

Mechanisms of Orofacial Pain[20]:

Peripheral Pain Mechanism

- Cutaneous Pain
- Dentinal Pain
- Inflammatory Pain

Central Pain Mechanisms: Trigeminal pain pathway

Clinical significance of inflammatory pain:

The inflammatory response to tissue damage results in the production of pain, edema and local increased temperature, redness and loss of function. Unlike dentinal pain, pain associated with inflammation has a prolonged time course.

In the periphery **Substance-P, CGRP** (calcitonin gene- related peptide) neuropeptides are transported from the CNS to the periphery. The relationship of these short lived inflammatory mediators with the release of substance P and the process of plasma extravasations form a positive feed back loop continually refueling the inflammatory process. This explains the prolonged time course of the inflammation which far exceeds the initial stimulation of the dental procedure[21].

Pharmacological management of pain in the periphery[22]:

Pain perception: Physio anatomical process, where impulse is generated following stimulus which is transmitted to central nervous system. This varies very little.

Pain reaction: Psychophysiological process represents person's manifestation of process that just occurred. This varies among individuals. Determines how patient will react to unpleasant experience.

Pain reaction threshold is inversely proportional to perception. To control pain, both aspects must be controlled[23]. The control of pain is brought about by nonsteroidal anti inflammatory drugs (NSAID's) .These exert their action at the nerve ending by blocking prostaglandin synthesis.

Clinical application: Usage of drugs like ibuprofen or other NSAID'S before a surgical procedure. This is also called as preemptive therapy.

Rationale for this therapy: Blockade of enzymes before initiation of tissue damage. Pre treating patients with ibuprofen delays the onset and reduces the magnitude of postoperative pain.

This prescription strategy emphasizes how clinicians can improve patient care by knowledge of **pain physiology**.

"**Ceiling Effect**" – when an analgesic produces a maximum effect and beyond this no additional increment of drug will produce any significant analgesia. This 'ceiling effect' probably reflects the contribution of other inflammatory mediators which are unaffected by the aspirin-like-drugs [20]. The metabolism of arachidonic acid via lipoxygenase pathway produces leukotreins and hydroperoxyeicosatetranoic acid (5HPETE's). Aspirin like drugs are ineffective in blocking this pathway. In chronic temporomandibular joint pain, the lipoxygenase products are formed. This explains why in chronic temporomandibular pain NSAID'S are not effective[24, 25].

DIAGNOSIS OF OROFACIAL PAIN[26, 27, 28]

To understand the diagnosis and treatment of orofacial pain, one must know the peripheral and central mechanisms that are involved in production and management of pain. The diagnosis of orofacial pain can often prove quite difficult for the dentist. A successful diagnosis of orofacial pain depends upon

1. Accurate and detailed history
2. Detailed clinical examination
3. Through knowledge of conditions

The history is the most important means of diagnosing orofacial pain, to differentiate the different causes, importance to certain key points have to be noted[29].

Location - pain is either diffuse or localized. Patient usually points with one finger when describing pain of dental origin or trigeminal neuralgia, atypical facial pain is more diffuse which may radiate across the midline.

Character – sharp or dull. Aching or throbbing/shooting. If patient has difficulty in describing the pain, they can rate pain severity on a scale of zero (no pain) to 10 (most severe pain that patient has experienced), or ask them to mark on a line divided into ten sections (visual analogue scale). These 'tools' help to assess the severity of pain and in the monitoring of treatment. Patient can also be asked regarding the disturbance in normal sleep pattern regarding severity of pain.

Duration – average duration of each episode of pain. For example, pain from exposed dentin will last only for sometime compared to pulpitis pain that lasts longer. Atypical facial pain is persistent on the other hand.

Frequency and periodicity – pain occurs at what specific times/ related to specific events. This can be noted by asking the patient to maintain a 'pain diary'.

Precipitating, aggravating and relieving factors – ask leading questions if temperature, biting, posture, analgesics affect the pain.

Associated features – other associated features such as swelling, e.g. dental abscess, nausea, vomiting, migraine or nasal stiffness must be noted.

The cause of most orofacial pain is established mainly from the history, and examination findings are also helpful in aiding in the diagnostic process to exclude any local pathology. Psychological causes of pain must be kept in mind while diagnosing pain with no organic causes[30].

PSYCHOLOGICAL COMPONENT OF PAIN:

The various psychological factors involved in perception of pain and behavior may be visualized through an elaboration of the basic model by **Melzack 1986** [31](Fig 3).

It is clear from the above model that pain is no longer a unitary concept. The sensory processes do not take place in a linear one-to-one fashion. There is a complex interaction between the various components of pain. This explains the

inconsistent relationships between tissue damage and pain perception

CONCLUSION

Pain is a multidimensional experience involving not only sensation evoked by noxious stimuli but also reaction to it. A coherent and clinically useful picture of pain needs to be projected that is clinically useful. It should be based on current knowledge in pain research. This article attempts to provide such a picture to the dentist who is involved in managing patients with complex orofacial pain.

Today pain has become the universal disorder, a serious and costly public health issue and a challenge for family and health care providers who must give support to the individual suffering from the physical as well the emotional consequence of pain.

Furthermore, successful diagnosis of pain requires a sound knowledge its physio anatomical and psychological aspects.

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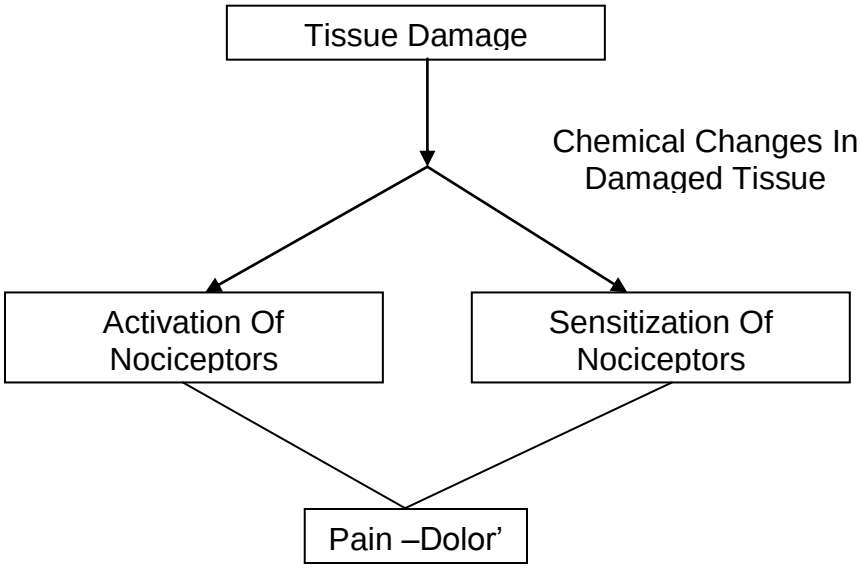
Fig 1: Biopsychological Model of pain (WHO) [4]

Fig2: Peripheral mechanisms of pain



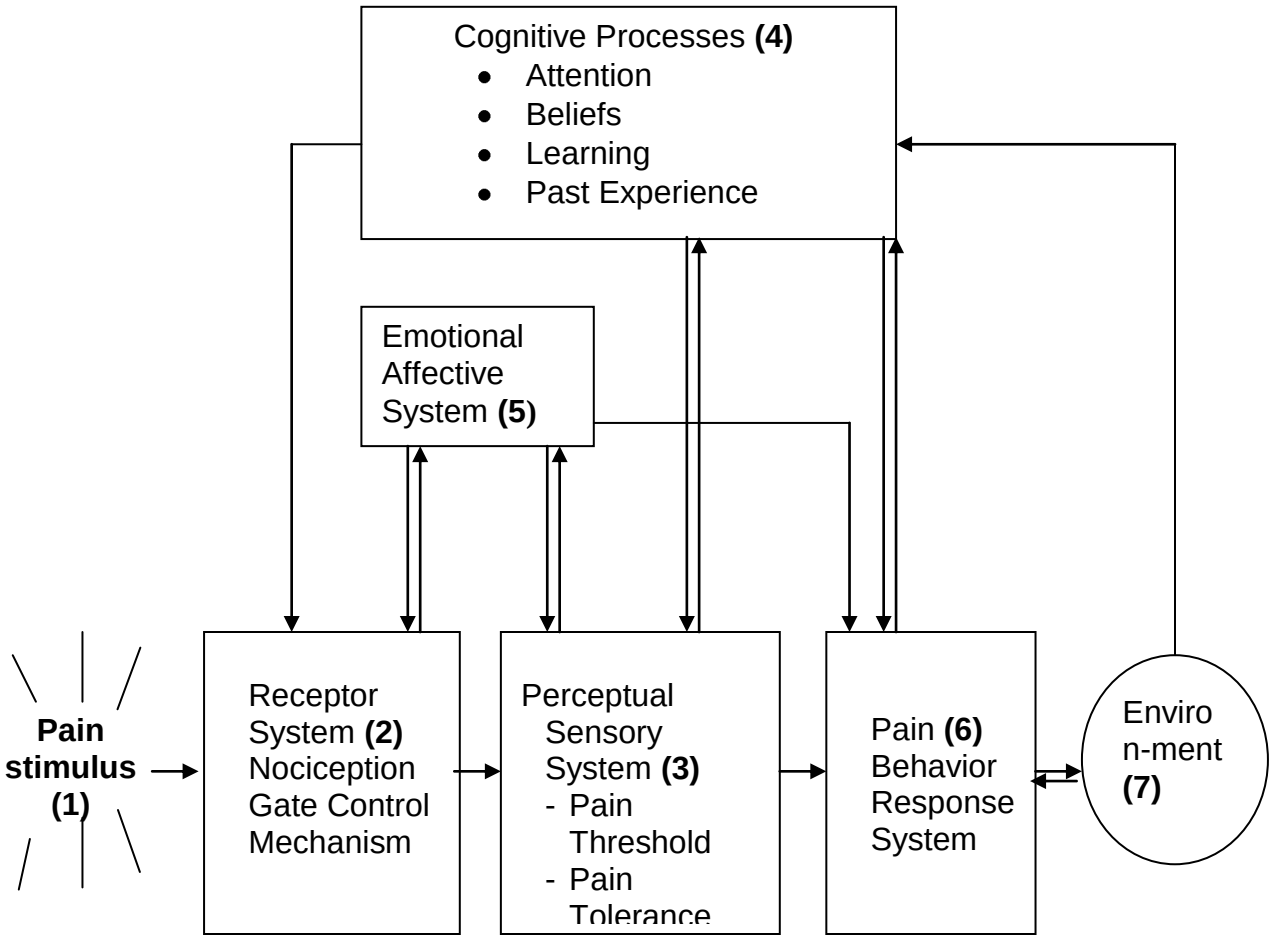


Fig 3: Model for interaction of components of pain experience.[24]