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شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



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لم ترد بالأصل



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**TRAMADOL AND /OR BUPIVACAINE FOR CAUDAL
BLOCK IN POSTOPERATIVE ANALGESIA FOR
PEDIATRIC SURGERY**

THESIS

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In

Anesthesiology

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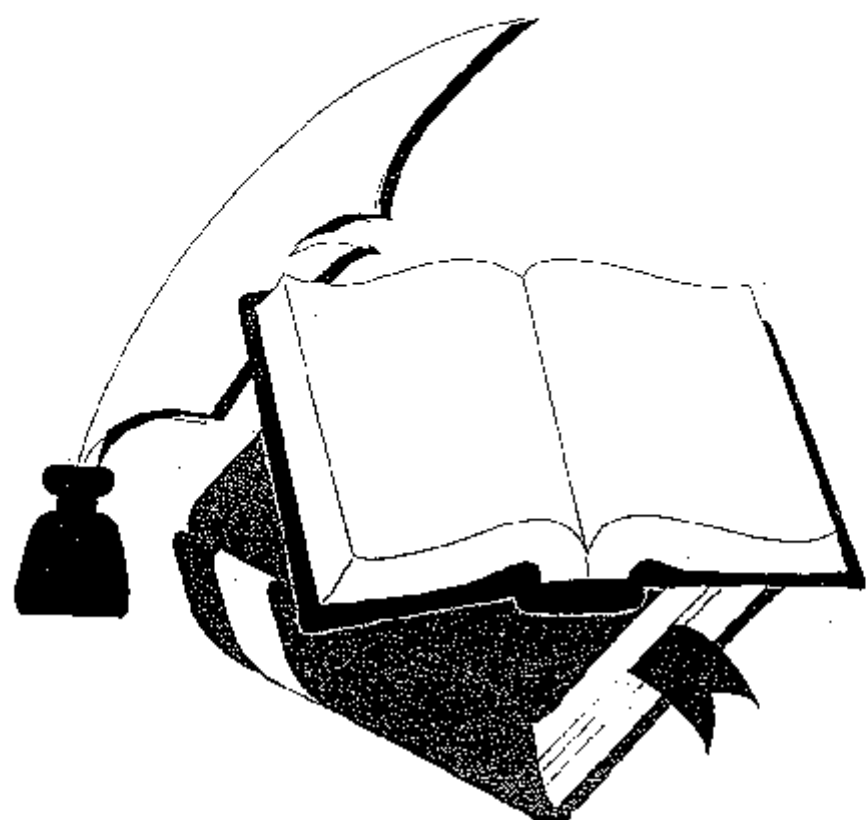
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*To
my Family*



REVIEW
OF
LITERATURE

REVIEW OF LITERATURE

PHYSIOLOGY OF PAIN

Pain is a protective mechanism for the body, it occurs wherever any tissues are being damaged and it causes the individual to react to remove the painful stimuli⁽¹⁾. Perception of pain like the perception of other sensations is a neuro-physiological process that has special structural, functional and perceptual properties and is accomplished by means of relatively simple neural, receptive and conductive mechanisms that are specific for pain⁽²⁾.

Pain has been classified into two different types, acute pain and slow pain. Acute pain occurs within about 0.1 second when a painful stimulus is applied, whereas slow pain begins only after a second or more and then increases slowly over a period of seconds and even minutes. The conduction pathways for these two types of pain are different⁽³⁾.

Pain receptors are specific and pain is not produced by overstimulation of other receptors. It has been suggested that pain is chemically mediated and stimuli which provoke it have in common- the ability to liberate a chemical agent which stimulates the nerve endings⁽³⁾.

Pain pathway:

Pain perception depends upon the transmission of impulses through pathways within the nervous system from the site of the stimulus to the higher centers of the brain and begins from pain

receptors present in skin, muscles and viscera along afferent nerve fibers which are of two types "A" delta and "C" fibers.

- * "A" delta fibers are 2-5mm in diameter, finely myelinated and conduct at a rate of 12-30 m/s.
- * "C" fibers are 0.4-1.2mm in diameter, myelinated and conduct at a rate of 0.5-2 m/s.

"A" delta fibers conduct sharp pain and are responsible for the withdrawal reflexes as their conduction are quick and well localized. "C" fibers are responsible for the more delayed and truly noxious burning or throbbing pain⁽⁴⁻⁶⁾.

The pain fibers enter the dorsal horn in the lateral division of the dorsal root. Thirty percent of the fibers enter through the ventral root in the gray matter of the spinal cord and cell bodies are arranged in eight laminae of Rexed⁽⁷⁾.

The majority of pain fibers synapse in the substantia gelatinosa with intermediate neurons which send projections to deeper layers. Cell bodies of nerve fibers lie in the dorsal root ganglia on entering the spinal cord⁽⁸⁾.

The pain fibers deviate laterally to form the ascending and descending branches of the tract of Lissaur at the tip of the posterior horn. After ascending one to three segments, these fibers synapse in the substantia gelatinosa on the tip of the posterior horn. The axons of the second neuron cross the midline in the anterior commissure to form the lateral spinothalamic tract which ascends and terminates in the lateral nucleus of the thalamus. From the thalamus, the third neuron

passes through the posterior limb of the internal capsule and is projected to the postcentral gyrus of the cerebral cortex where anatomical representation is reasonably precised⁽⁸⁾.

In addition to this, it has been suggested that collaterals from the spinothalamic tract are distributed to the ascending reticular system (spinoreticular pathways)⁽⁹⁾.

Pain impulses transmitted in this multisynaptic pathways pass via the reticular formation to be relayed over a wide and poorly localized area of the cerebral cortex⁽⁸⁾.

In the pain pathways, the thalamus is responsible for appreciation of pain while the cerebral cortex is responsible for localization and determination of the degree of pain. The responsibility of the reticular formation is to activate the cortex to help to maintain arousal and it also modifies the effective component of pain⁽¹⁰⁾.

There are various means by which pain transmission may be inhibited at the spinal levels:

A- Large primary fibers, afferents or ascending inhibitory pathways:

It is called a beta large low threshold mechanoreceptors primary afferents which enter in the medial portion of the dorsal root horn and pass without synapse up to the dorsal column. "AB" delta fibers from the skin give collaterals which enter the dorsal horn from the medial side and ramify in several laminae⁽¹¹⁾.

B- Inhibitory fibers (descending fibers):

Their cell bodies lie in the medullary raphe nuclei of brain stem and their fibers travel in the dorsolateral funiculus of the spinal cord. They excite the enkephalinergic interneurons in the spinal cord and can be blocked or reversed by the opiate receptor blocking drug "naloxone"⁽¹²⁾.

C- Opiate receptors:

It is suggested that there are highly specific opioid receptors which could be combined in a stereospecific manner with all known active opiate agonists. Opioid receptors are present in the gray mater of the brain stem, medial thalamus, posterior pituitary and substantia gelatinosa of spinal cord, while the cerebral cortex has a low density of receptors and cerebellum is devoid of these receptors⁽¹³⁾.

There is reasonably firm evidence for three major categories of opioid receptors in the central nervous system designated:

- 1- μ (m μ) receptors.
- 2- K (kappa) receptors.
- 3- Δ (delta) receptors.
- 4- ϵ (epsilon) receptors.
- 5- δ (sigma) receptors.

μ (m μ) receptors are responsible for supraspinal and spinal analgesia, physical dependence and euphoria. K (kappa) receptors are responsible for sedation and spinal analgesia⁽¹⁷⁾. Δ (delta) receptors play a prominent role in respiratory depression. ϵ (epsilon) receptors seem to play a role in

endogenous opioid (mainly beta-endorphin) neurotransmission and in the mediation of opioid effects especially suppression of stress response and acupuncture. δ (sigma) receptors are responsible for dysphoria, mydriasis and respiratory stimulation⁽¹⁴⁾.

Mechanism of action of opiate receptors:

The three classes of opiate receptors share the following characters:

- a- They all appear to function primarily by exerting inhibitory modulation of synaptic transmission in both the central nervous system and the myenteric plexus. Although their location varies, they are often found on presynaptic nerve terminals, where their action result in decreased release of excitatory neurotransmitters.
- b- They all appear to be coupled to guanine nucleotide binding regulatory proteins. Thus, it can be anticipated that opioids regulate the transmembrane signalling systems that are characteristically initiated by this type of receptors. The consequences of the activation of μ and delta receptors are usually very similar and are distinctly different from those resulting from activation of kappa receptors⁽¹⁵⁾.

Neuropharmacology:

In the periphery :

Prostaglandins of the E and I series sensitize pain receptors and prostaglandin E substance (PGES) are believed to be involved in the amplification of pain in the inflammatory process.