The role of Pre-emptive analgesia in the treatment of postoperative pain

Essay
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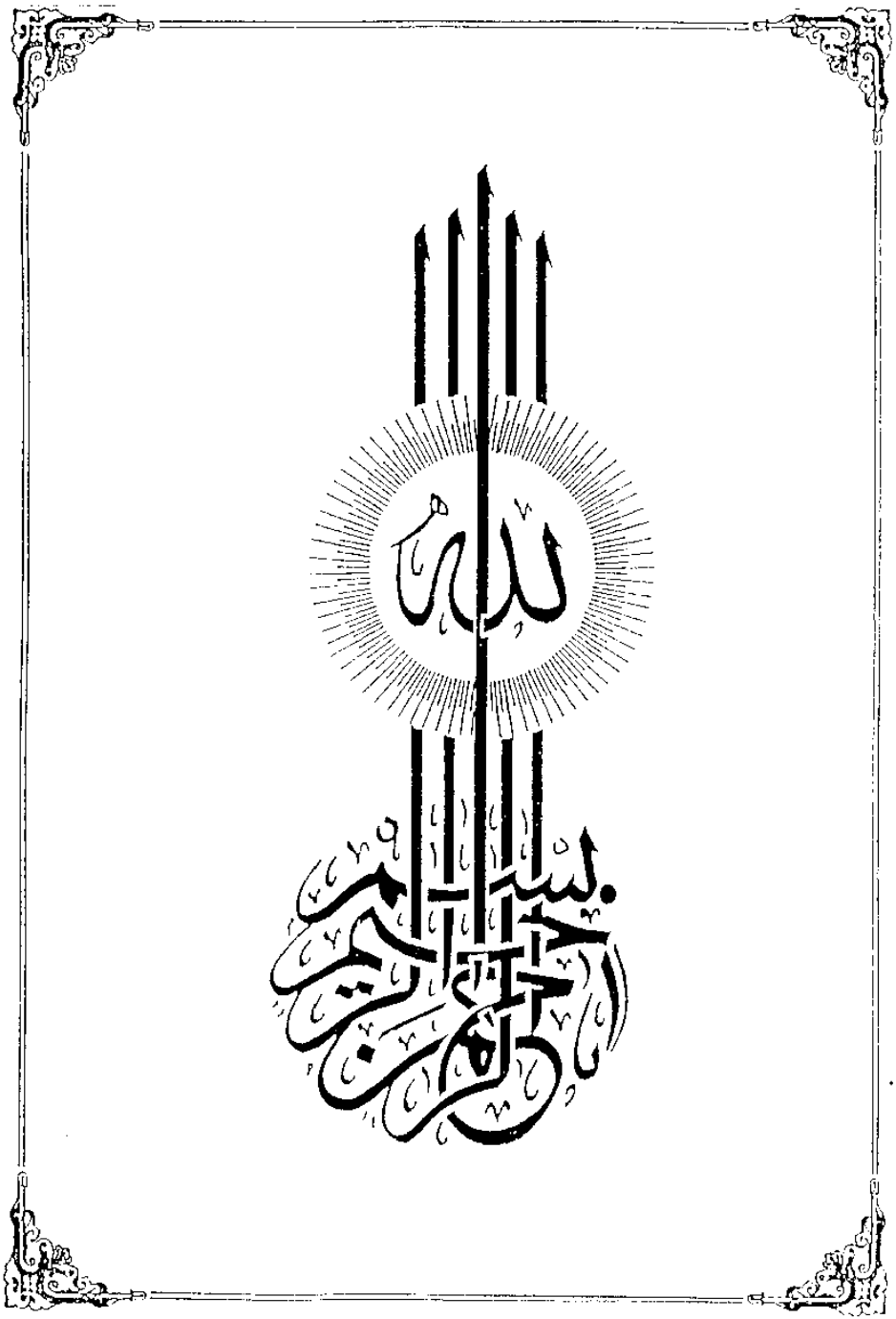
To My Wife and Family

Adel



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CHAPTER 1

INTRODUCTION

Introduction

Pain linguistically is derived from the latin "Ponea",a penalty or punishment, this means that we use the word when any sensation becomes disturbing, irritating or causes suffering to us.However, pain in physiological terms is the psychologic adjuvant to a protective reflex, the purpose of which is to cause the affected tissue to be withdrawn away from the potentially noxious stimulus *(Sherrington,1947)*. In 1979 the international association for the study of pain (IASP) describes pain as : "unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage". Pain in another way may be defined as the conscious awareness of tissue injury *(Bonica,1990)*.Its perception reflects activation of nociceptors,afferent transmission to the spinal cord,and relay via dorsal horn to higher centers.

On a teleologic level such behavior serves to avoid further injury and promote wound healing,however,other affective responses,including profound suffering,increased anxiety, release of stress hormones and catecholamines,may be less than desirable.After extensive dissection and visceral manipulation,such responses often incite pathologic changes that may adversely affect postsurgical outcome , particularly in high - risk patient population.Inadequately treated pain is a major cause of unanticipated hospital stay after surgery.Postoperative pain relief is associated with a more stable cardiovascular system,decreased neuroendocrine stress response,decreased respiratory complications,decreased thromboembolic complications and early ambulation(*Sinatra,1992*).

Preemptive analgesia aims at treating postoperative pain by preventing the central nervous system from reaching a hyperexcitable state in which it responds excessively to afferent

inputs.This approach for treating postoperative pain is developed as a consequence of advances in the physiology of pain and how clinical pain is generated.Preemptive treatment could be directed at the periphery,at inputs along sensory axons,and at central neurons by using opioids,non-steroidal antiinflammatory drugs and local anesthetics utilizing different routes of administration including the parentral,oral,transmucosal or intraspinal routes *(Woolf and Chong,1993)*.New drugs that block the excitatory aminoacids and neuropeptide transmitters that induce central sensory hyperexcitability may become available in the future,enabling a more direct treatment of injury-induced sensory hyperexcitability and therefore,a more effective treatment of postoperative pain*(Tverskoy et al.,1994)*.

CHAPTER2

PHYSIOLOGY OF POSTOPERATIVE PAIN

Physiology of Postoperative Pain

There are two theories explaining the neural organization for nociception. The specificity theory states that there is a sensory system specific for nociception which has its own receptors,pathways,subcortical and cortical areas *(Mense ,1983)*.On the other hand , the intensive theory proposes that all sensory elements can register pain if stimulated sufficiently *(Edmeads,1983)*. Most basic scientists and clinicians support the specificity theory for neural organization for nociception *(Woolf,1989)*.

There are two distinct types of pain , both ocurring after each other in response to a single stimulus , these are fast and slow pain.Fast pain is short,well localized,stabbing in character.It is matched in intensity to the intensity of the stimulus ,it begins and ends abruptly. Slow pain is longer in duration , diffuse,throbbing burning or aching in character.It is seldom matched to the intensity of the stimulus *(Stoelting ,1993)*. The temporal separation between the two types of pain depends on the distance between the site of stimulation and the brain , being greater when the stimulation is further from the brain *(Guyton and Hall,1996)*.

Nociceptors

Nociceptors are naked nerve endings of primary afferents of Aδ and C fibers.These nerve endings are widely distributed in the superficial layers of the skin, being more abundant in more sensitive areas.They are also found in the periosteum, joint surfaces,skeletal muscles,and tooth pulps.Nociceptors are classified according to their mechanism of stimulation into three types :

(1)Mechanosensitive nociceptors : these respond to mechanical deformation.Their impulses are transmitted via Aδ fibers.

(2)Mechanothermal nociceptors : these respond to mechanical deformation and thermal changes.Their impulses are also transmitted via Aδ fibers .

(3)Mechanothermochemical nociceptors: these respond to mechanical deformation,thermal changes and chemical factors such as substance P,potassium,bradykinin,prostaglandins,acetyl choline,histamine and serotonin.Their impulses are transmitted via C fibers *(Stoelting ,1993).*

All types of nociceptors are characterized by having a high threshold for impulse initiation.They do not adapt as opposed to other sensory receptors,this non-adaptability is protective in the sense that more tissue damage produces more pain signals as pain receptors do not stop sending impulses *(Stoelting ,1993).*

The Pain Pathway(Figures 2-1 and 2-2)

The bipolar nociceptor primary afferents unite to form axons which pass to cell bodies within the dorsal root ganglion and the trigeminal ganglion,these cell bodies send their dendrites to enter the dorsal horn of the spinal cord .

On entering the dorsal horn,nociceptor primary afferent fibers ascend or descend one to three segments in the tract of Lissaur which lies immediately posterior to the dorsal horn.From this point on there are two pathways for pain fibers (*Guyton and Hall , 1996*) .

The fast type Aδ pain fibers terminate at two points in the dorsal horn, in lamina I(lamina marginalis) and lamina V.In both

of these laminae the Aδ fibers excite second order neurons that send long fibers immediately to the opposite side of the cord in the anterior commissure and then upwards in the anterolateral pathway,to terminate on the reticular formation of the medulla,pons and mesencephalon.From these areas higher order neurones pass to the thalamus,hypothalamus and cerebrum.However a small proportion of nerve fibers transmitting fast pain pass directly to the ventrobasal complex and the posterior nuclear group of the thalamus,from here signals are transmitted to the sensory cortex. These fibers are important mainly for localizing pain not for interpreting it *(Ganong , 1995)*.

The much slower C fibers terminate in laminae II&III of the dorsal horn,an area called the substantia gelatinosa.These fibers synapse with one or more interneurons before eventually terminating in lamina V.Here the last neuron gives rise to long axons most of which join the fibers of the fast pain pathway crossing the cord in the anterior commissure and reaching the midbrain reticular formation,the thalamus and the crebral cortex via the lateral division of the anterolateral sensory pathway. However some fibers pass ipsilaterally to the thalamus via the anterior division of the anterolateral sensory pathway.In contrast to the fast pain fibers the slow pain fibers terminate almost entirely on the reticular formation of the brainstem and on intralaminar nuclei of the thalamus.Both areas are part of the reticular activating system,thus,the slow burning aching pain fibers have a very potent effect in activating essentially the entire central nervous system,this activating effect of the slow pain fibers cause the person to get excited,arise from sleep,thus promoting defence and aversion reactions designed to rid the person of the painful stimulus (*Guyton and Hall , 1996)*.

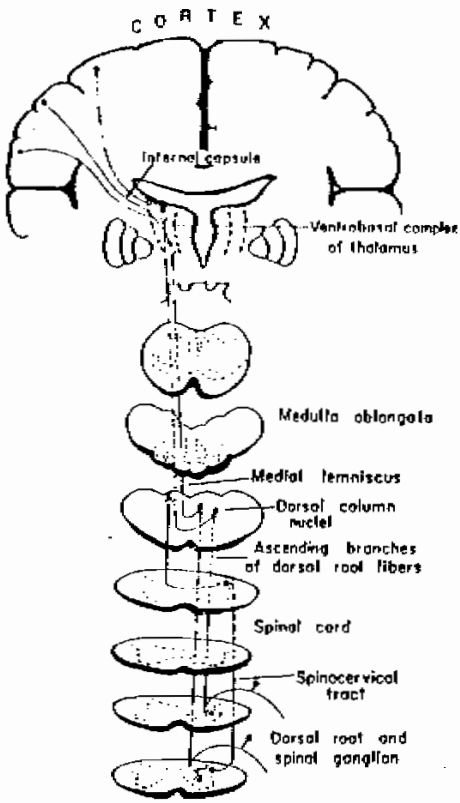

Figure (2-1):Dorsal column pathways *(after Guyton and Hall,1996).*

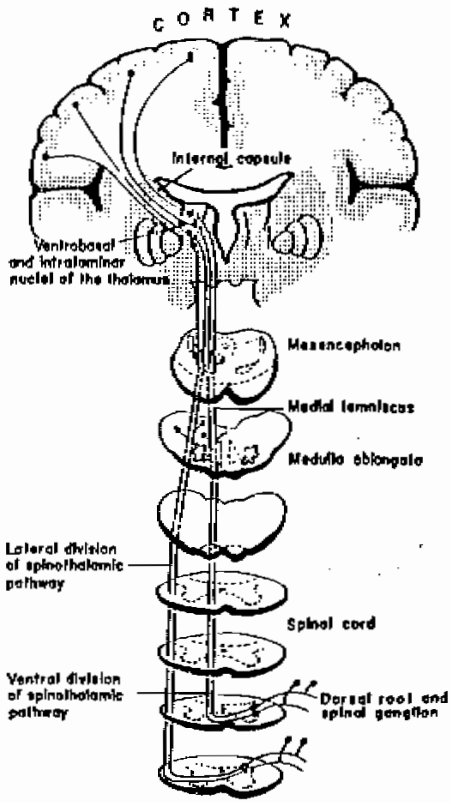

Figure (2-2):Anterolateral spinothalamic pathways*(after Guyton and Hall,1996).*

The cortical projection of the pain pathway comprise those of the thalamocortical projections,fibers from the ventrobasal lateral nuclei of the thalamus carrying fast pain signals pass to the somatosensory area of the postcentral gyrus.While fibers from intralaminar nuclei carrying slow pain signals project to wide areas of the cortex *(Bonica et al.,1990).*

Pain Modulating Mechanisms

Pain modulating mechanisms act to modify the peripheral input either by sensitization of receptors and neural circuits or by inhibiting pain transmission at different points in the pain pathway These mechanisms operate at different levels in the pain pathway including peripheral sensitization of nociceptors ,action of the dorsal horn cells as a gate for control of pain transmission and central sensitization at the level of the spinal cord.Lastly,these pain modulating mechanisms are controlled by the brain inhibitory -or analgesia-system *(Woolf , 1989) .*

1.Peripheral sensitization (Figure 2-3)

After peripheral tissue injury the threshold for iliciting pain is decreased both within the area of tissue injury -Primary Hyperalgesia- and in the surrounding uninjured tissue-Secondary Hyperalgesia-*(Lewis, 1942).*

In the zone of injury , the increased responsiveness is to thermal and mechanical stimuli,whereas in the surounding zone the sensitivity is exclusively to mechanical stimuli *(Campell et al., 1979).*The neuronal changes contributing to primary hyperalgesia according to Raja et al. in1984 are:

- A decrease in the threshold.
- An augmented response to suprathreshold stimuli.
- Spontaneous activity.