Relief of Pain in Trauma Patients

Essay
Submitted for Partial Fulfillment of Master Degree
in Intensive Care

By
Mona Soliman Mahmoud Metawea
M.B., B.Ch. El-Mansoura University

Under Supervision of
Prof. Dr. Amir Ebrahim Mohamed Salah
Professor of Anesthesiology, Intensive Care & Pain Management
Faculty of Medicine – Ain Shams University

Dr. Hadil Magdi Abd Elhamied
Assistant Professor of Anesthesiology, Intensive Care & Pain Management
Faculty of Medicine – Ain shams University

Dr. Simon Halim Aramanios
Lecturer of Anesthesiology, Intensive Care & Pain Management
Faculty of Medicine – Ain shams University

Faculty of medicine
Ain Shams University
2015

Acknowledgement

First, I wish to express my deep thanks, sincere gratitude to **ALLAH**, who always help me, care for me and grunted me the ability to accomplish this work.

I would like to express my deepest gratitude, thanks and gratefulness to **Prof. Dr. Amir Ebrahim Mohamed Salah**, professor of Anesthesiology and Intensive Care, Faculty of Medicine _ Ain Shams University, for his enthusiastic support, continuous encouragement, valuable scientific advices and great help throughout the accomplishment of this work.

My sincere thanks to **Ass. Prof. Dr. Hadil Magdy Abdelhamid**, Assistant professor of Anesthesiology and Intensive Care, Faculty of Medicine _ Ain Shams University, for her meticulous supervision, support, help all through this work.

I acknowledge with much gratitude to **Dr. Simon Halim Armanios**, Lecture of Anesthesiology and Intensive Care Medicine, Faculty of Medicine _ Ain Shams University for his great supervision and unlimited help to provide all facilities to accomplish this work.

Words can never express my sincere thanks to my mother, my husband, my son, my cousin, all members of my family, and my friends for their generous support and continuous encouragement.

Contents

Page No.

List of Figures

List of Tables

INTRODUCTION

Trauma is a Tissue damage caused by an extrinsic force. The stress response compromises an activation of neurohumoral and physiologic process that would lead to improved survivability of untreated patient following a traumatic injury. Worldwide, trauma constitutes a massive public health problem. Approximately 5.8 million people die annually as a result of injury, representing10% of the world's deaths. This figure is 32% more than the number of deaths from the major diseases of malaria, tuberculosis and HIV/AIDS combined ***(Bilir and Gulec, 2006).***

According to the World Health Organization (WHO), motor vehicle collisions account for 1.3 million deaths annually, and this were the ninth leading cause of disability in 2004, and will rise to the third leading cause of disability worldwide by[2030]. Outside areas of armed conflict, penetrating injuries are responsible for fewer than 15% of traumatic deaths worldwide, but these rates vary by country. Approximately half of traumatic deaths result from central nervous system (CNS) injury ***(Evan et al., 2010).***

Pain is one of the main complain of trauma patients in (pre-hospital) emergency medicine and its prevalence in pre-hospital emergency medical services is 70%. Other studies in

the emergency department report pain prevalence in these patients ranging from 52% to 90%. From a humanitarian point of view, every patient is entitled to receive adequate pain management. Inadequate relief of pain leads to delay healing, reduced functional recovery and an impaired immune function. There is also increasing evidence that inadequate pain treatment may lead to chronic pain and disability, resulting in higher costs in health care ***(Rivera et al., 2010).***

The management of pain in trauma is based on a combination of local anesthetic, simple analgesics, non-steroidal anti-inflammatory drugs (NSAIDS) and opioids. The uses of multimodal analgesia reduce hospital stay, decrease medical complications and increase patient satisfaction ***(Skinner et al., 2004).***

The limited duration of analgesia after single blocks can be prolonged by use of adjuvant (clonidine, ketamine), catheter techniques or early use of systemic analgesics. Non-opioids (acetaminophen Non-steroidal anti-inflammatory drugs) are appropriate for patients with mild to moderate pain or as a component of multimodal pain therapy ***(Sumpelmann and Mute, 2003).***

In recent years, major advances have been made in the management of trauma, the end result of which has been reduced mortality associated with trauma, which has led to an in-

creased emphasis on secondary outcome measures, such as psychological well-being, functional improvement, and vocational social reintegration ***(Jenewein et al., 2009).***

Chapter (1):

PATHOPHYSIOLOGY OF PAIN

The international association for the study of pain (IASP) defines pain as "an unpleasant sensory and emotional experience which is primary associated with tissue damage or described in terms of such damage or both".

This definition acknowledges that pain in responsive human beings is a conscious experience involving interpretation of sensory input that signals anxious event and is influenced by emotion, cognition, memory, interpersonal and social context and other factors.

Pain is classified as "structural pain", pain associated with tissue damage which results in protective withdrawal reflexes to protect tissues from further damage or "functional pain", pathological pain not associated with ongoing tissue damage ***(Gold et al., 2006)***.

Also, pain is anatomically classified into 3 categories: somatic pain (physiological and pathological), visceral, and neuropathic pain ***(Dubin and Patapoution, 2010)***.

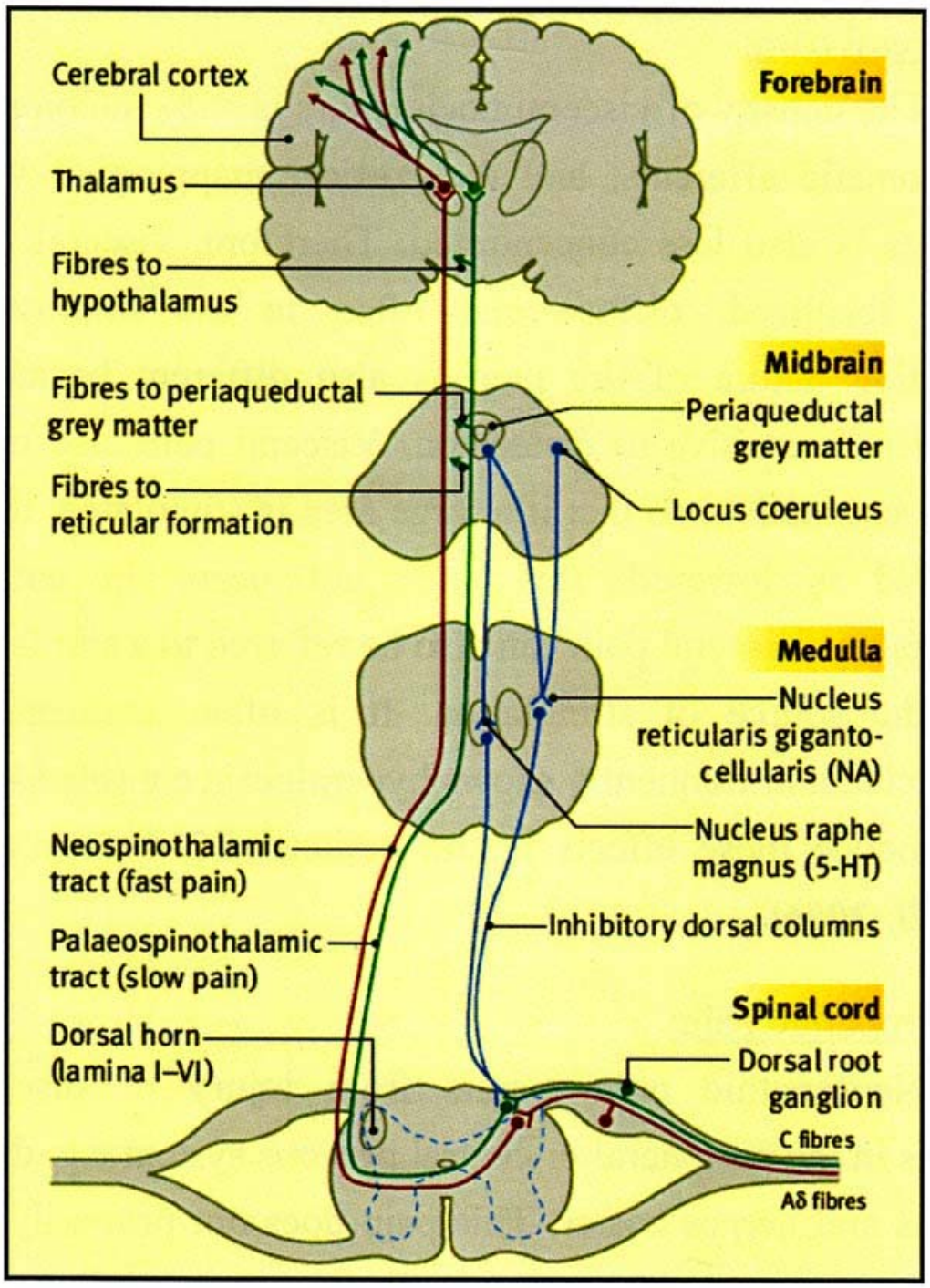

Figure (1): Spinal and Supraspinal pathways of pain ***(Serpell, 2006).***

Anatomical classification and different pathways of pain

1) Somatic pain:

a) Physiological (first or 'fast') pain: is a protective event that enables the person to localize pain rapidly, accurately, withdraw from the stimulus and to avoid or reduce further tissue damage. It is produced by stimulation of high threshold thermo/mechanical nociceptors, which transmit action potentials by fast conducting (12-30 m/s) myelinated Aδfibers that enter the dorsal horn of the spinal cord and synapse at laminae I, V and X. Conduction continues along the secondary afferent fibers via the neospinothalamic tract, which is monosynaptic, as it ascends to the posterior thalamic nuclei. From there it synapses with tertiary afferents to the somatosensory post-central gyrus at the cortex. If the stimulus is of short duration and does not cause tissue damage, the pain disappears when the stimulus stops ***(Farquhar and Smith, 2007)***.

b) Pathophysiological (second or 'slow') pain: is responsible for the delayed pain sensation that occurs after tissue injury (surgery, trauma, inflammation) and which encourages tissue healing by eliciting behavior to protect the damaged area. It originates from stimulation of the high threshold polymodal nociceptors (free endings) present in tissues. The nociceptors

respond to mechanical, chemical and thermal stimuli and are transmitted via slow conducting (0.5-2 m/s) unmyelinated C fibers that synapse at laminae II and III (substantia gelatinosa) of the dorsal horn. Secondary afferents ascend cranially via the palaeospinothalamic tract, which is polysynaptic, as it ascends to medial thalamic nuclei. It has collaterals that also project to the midbrain, pontine and medullary reticular formations, the periaqueductal grey matter, and the hypothalamus, where they synapse into neurons that in turn project to forebrain limbic structures. This system is primarily involved with the reflex responses of pain (respiratory, circulatory, endocrine) ***(Farquhar and Smith, 2007)***.

2) Visceral pain:

The density of visceral nociceptors is <1% in comparison with somatic afferents and the cortical mapping of visceral afferents is also less concentrated. Therefore, visceral pain is poorly localized, diffuse and often in the midline. The qualitative nature of the pain is also different because the viscera are sensitive to distension. Visceral pain also exhibits spatial summation, so that if a large area is stimulated, the pain threshold is lowered, this does not occur in cutaneous nociception. Visceral pain can also be referred to a site far away from the source of stimulation. It is often segmental and superficial, and frequently shows hyperalgesia e.g. (bladder

pain can produce these effects in the perianal S2-4 dermatomes) ***(Serpell, 2006)***.

3) Neuropathic pain:

Neuropathic pain results from injury or disease of neurons in the peripheral or central nervous system e.g. diabetes mellitus and herpes zoster. This pain does not primarily signal noxious tissue stimulation and, therefore, feels abnormal. It often has a burning or electrical character and can be persistent or occur in short episodes. It might be combined with hyperalgesia and allodynia ***(Schaible and Richter, 2004)***.

Pathogenesis and Mechanism of Pain at different level of pain pathway:

The periphery: the nociceptors

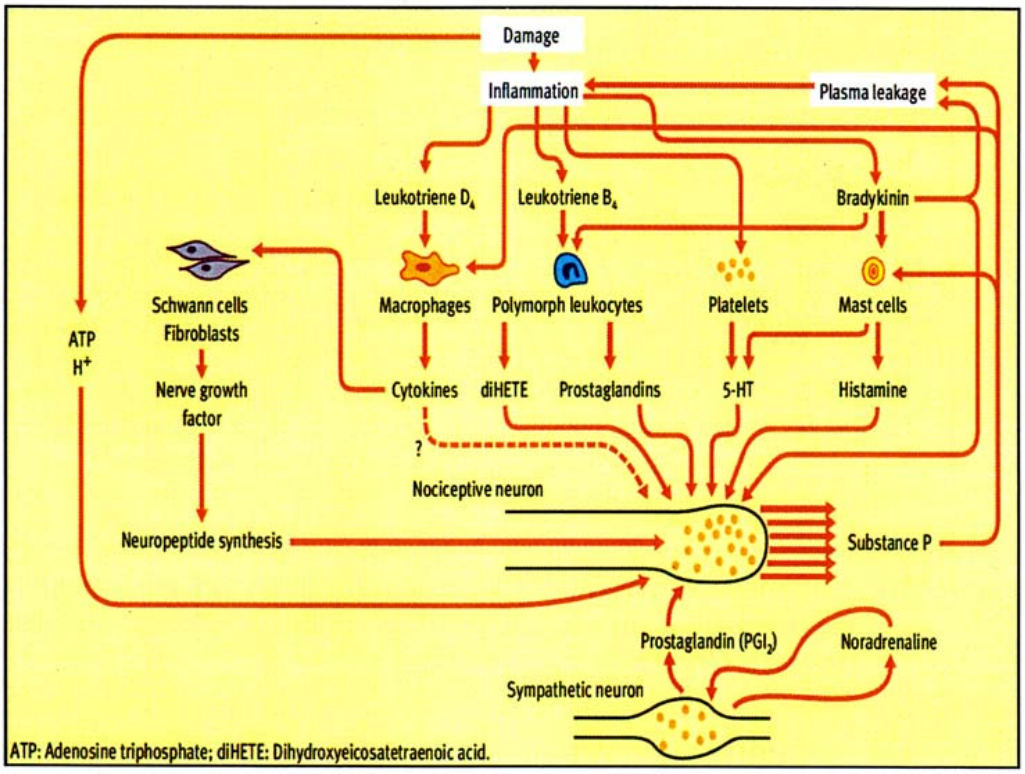

Figure (2): Peripheral inflammatory mediators ***(Serpell, 2006)***

An injury that causes a potential risk for the organism will activate free nerve endings that respond to nociceptive stimulation (Fig. 2). Most of these fibers are polymodal and will respond to different modalities, including mechanical, thermal, and chemical stimulation ***(Meyer et al., 2006)***.

A nociceptive stimulation will initiate a cascade of events. Pronociceptive inflammatory molecules will be released into the periphery and will produce peripheral hyperalgesia. These pronociceptive inflammatory molecules originate in various blood cells (mastocytes, polymorphonuclear cells, and platelets) and include bradykinins, prostaglandins, histamine, serotonin, adenosine triphosphate, and also from immune cells which produce interleukins, interferon, and tumor necrosis factors [3-6]. Substance P and calcitonin gene related protein (CGRP), which act as neurotransmitters in the CNS, are also released into the periphery and act as proinflammatory factors in the periphery, favoring neurogenic inflammation ***(Mense et al., 2008)***.

	Aβ	**Aδ**	**C**
Diameter	6 to 12 μ myelinated	1 to 5 μ myelinated	0,2 to 0,5 μ unmyelinated
Conduction	35 to 75 M/s	35 to 75 M/s	35 to 75 M/s
Role	Light touch, proprioception	Temperature, Nociception (mechanical, thermal)	Nociception (mechanical, thermal and chemical)