A COMPARISON BETWEEN MULTIMODAL ANALGESIA AND SINGLE MODE ANALGESIA IN LUMBAR VERTEBRAL SURGERY

Thesis

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مقارنة بين التسكين المتعدد الوسائط والتسكين الأحادى الوضع في جراحة العمود الفقري القطني

رسالة

توطئه للحصول على درجة الدكتوراه فى التخدير

مقدمة من

الطبيب/كيرلس عماد موريس ميساك

اقكهنيهم زيطض اهك جنح ب ه لئج زتي نطكة خبج ذ

قكي بطض ا– جئلع ب جي م سدل ز

تحت إشراف

الأستاذ الدكتور/ محمود عبد العزيز غلاب

آ زةئدطكة خبج ذ هك ذعي بطك لنقن

قكي بطض ا أ جئلع ب جي م سدل ز

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In our study, 102 patients scheduled to undergo single or double level lumbar discectomy or fixation surgery under general anesthesia were randomly assigned to one of two groups:

Group A:The multimodal group who received acetaminophen 1 gm, ketamine 0.25 mg/kg, morphine 3 mg, all given I.V before induction of general anesthesia. Then intraoperative analgesia was maintained by local infiltration of the surgical site by mixture of bupivacaine and lidocaine and I.V infusion of ketamine and morphine in a rate of 5 and 1 mg/hour respectively.

Group B: the morphine group received morphine in a dose of 0.1 mg/kg before induction of general anesthesia. Then intraoperative analgesia was maintained by morphine increments of maximum 3 mg.

The study continued for 6 hours postoperative and demonstrated that using multimodal analgesia produce more favorable pain relief and maintain stable hemodynamics in the intraoperative and early postoperative periods with less stress response, narcotic consumption and narcotic induced respiratory depression.

Regarding systolic and diastolic blood pressures and heart rate, there was a significant decrease in intraoperative and early postoperative values in the multimodal group than morphine group.

Regarding narcotic consumption there was significant lower consumption in the multimodal group and also significant longer interval time for requesting analgesia.

Regarding respiratory depression, there was no respiratory depression in both groups, but higher PaO2, and lower PaCO2 were noticed in the multimodal group.

Regarding stress response to surgery, it was much lower in the multimodal group, however intraoperative cortisol levels between the two groups were not statistically significant.

We concluded that the use of multimodal analgesia prolonged the duration of postoperative analgesia and decrease the intensity of pain, with stable hemodynamics and without any respiratory burden, furthermore it decreases the consumption of narcotics by decreasing the frequent requesting of analgesia. Moreover, cortisol levels didn’t decrease significantly with the use of multimodal analgesia.

First thanks to ***ALLAH*** to whom I relate any success in achieving any work in my life.

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INTRODUCTION

Multimodal analgesia is achieved by using combinations of analgesics (e.g., opioids, non-steroidal anti-inflammatory drugs, and local anesthetics), which act simultaneously by different mechanisms, resulting in additive or synergistic analgesia and fewer adverse effects ***(Dorr et al, 2008).***

Multimodal analgesia is a multidisciplinary approach to pain management with a goal to maximize the analgesic effect and minimize the side effects of the medications. It takes advantage of the additive or synergistic effects of various analgesics, permitting the use of smaller doses with a concomitant reduction in side effects. Because many of the negative effects of analgesic therapy are related to parenteral opioids, limiting its use is the major principle of multimodal analgesia ***(Wheeler et al, 2002).***

Postoperative pain remains poorly controlled despite recent advances in the development of new drugs and techniques. One approach to improving postoperative pain control is to use a multi-modal analgesic regimen which capitalizes on the combined actions of multiple agents in reducing perioperative nociceptive input and central sensitization ***(Joel et al, 2006).***

The concept of multimodal (or balanced) analgesia suggests the use of combinations of several analgesics of different classes that are administered by different techniques. Analgesic options for the multimodal approach to postoperative pain control in shoulder surgery include opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), and local analgesic techniques, such as, nerve blocks, wound infiltration, and the intraarticular or subacromial administration of local anesthetics ***(Tang et al, 2009).***

Pain involves multiple mechanisms that ideally require treatment using a multimodal (or 'balanced') analgesic technique with the aim of improving analgesia by combining analgesics with additive or synergistic effects ***(White et al, 2010).***

Other non-opioid analgesics including ketamine, dexmedetomidine, dextromethorphan, alpha- 2 agonists, beta-blockers, local anesthetics, and acetaminophen can help to improve the pain management and perioperative outcome ***(Suzuki et al, 2009).***

AIM OF THE WORK

This study aims to compare between the effect of multimodal analgesia and single mode analgesia in lumbar spine surgery on intraoperative and early postoperative analgesia, intraoperative cardiovascular stability and stress response, plus the consumption of narcotics in both types.

PHYSIOLOGY OF PAIN

Definition of pain:

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment. Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus ***(Mersky&Bogduk, 1994).***

Pain threshold is the least experience of pain that a subject can recognize as suggested in 1994, whereas a new definition is now being discussed by IASP that pain threshold is the minimal intensity of a stimulus that is perceived as painful ***(Loeser&Treede, 2008).***

Pain tolerance level is the greatest level of pain that a subject can recognize as suggested in 1994, whereas a new definition is now being discussed by IASP that pain tolerance level is the maximum intensity of a stimulus that evokes pain and that a subject is willing to tolerate in a given situation ***(Loeser&Treede, 2008).***

Classification of pain:

Acute pain*(Figure 1)*

Acute pain can be defined as pain that is caused by noxious stimulation due to injury, a disease process or the abnormal function of muscle or viscera. It is usually nociceptive. Nociceptive pain serves to detect, localize and limit tissue damage. This type of pain is typically associated with a neuroendocrine stress proportional to its intensity, its most common forms include posttraumatic, postoperative, and obstetric pain as well as pain associated with acute medical illnesses, such as myocardial infarction, pancreatitis, and renal calculi. Most forms of acute pain are self-limited or resolve with treatment in a few days or weeks. When the pain fails to resolve because of either abnormal healing or inadequate treatment, the pain becomes chronic. Two types of acute (nociceptive) pain; somatic and visceral are differentiated based on origin and features ***(Morgan et al, 2006).***

Somatic Pain

Somatic pain can be further classified as superficial or deep. Superficial somatic pain is due to nociceptive input arising from skin, subcutaneous tissues, and mucous membranes. It is characteristically well localized and described as a sharp, pricking, throbbing, or burning sensation. Deep somatic pain arises from muscles, tendons, joints, or bones. In contrast to superficial somatic pain, it usually has a dull, aching quality and is less well-localized. An additional feature is that both the intensity and duration of the stimulus affect the degree of localization. For example, pain following brief minor trauma to the elbow joint is localized to the elbow, but severe or sustained trauma often causes pain in the whole arm***(Morgan et al, 2006).***

Visceral Pain

The visceral form of acute pain is due to a disease process or abnormal function of an internal organ or its covering e.g., parietal pleura, pericardium, or peritoneum.

Four subtypes are described: (1) true localized visceral pain, (2) localized parietal pain, (3) referred visceral pain, and (4) referred parietal pain. True visceral pain is dull, diffuse, and usually midline. It is frequently associated with abnormal

sympathetic or parasympathetic activity causing nausea, vomiting, sweating, and changes in blood pressure and heart rate. Parietal pain is typically sharp and often described as a stabbing sensation that is either localized to the area around the organ or referred to a distant site (The phenomenon of visceral or parietal pain referred to cutaneous areas results from patterns of embryological development and migration of tissues, and the convergence of visceral and somatic afferent input into the central nervous system).***(Morgan et al, 2006).***

Chronic Pain*(Figure 1)*

Chronic pain is defined as pain that persists beyond the usual course of an acute disease or after a reasonable time for healing to occur; this period can vary from 1 to 6 months. Chronic pain may be nociceptive, neuropathic, or mixed. A distinguishing feature is that psychological mechanisms or environmental factors frequently play a major role. Patients with chronic pain often have an attenuated or absent neuroendocrine stress response and have prominent sleep and affective (mood) disturbances***(Morgan et al, 2006).***

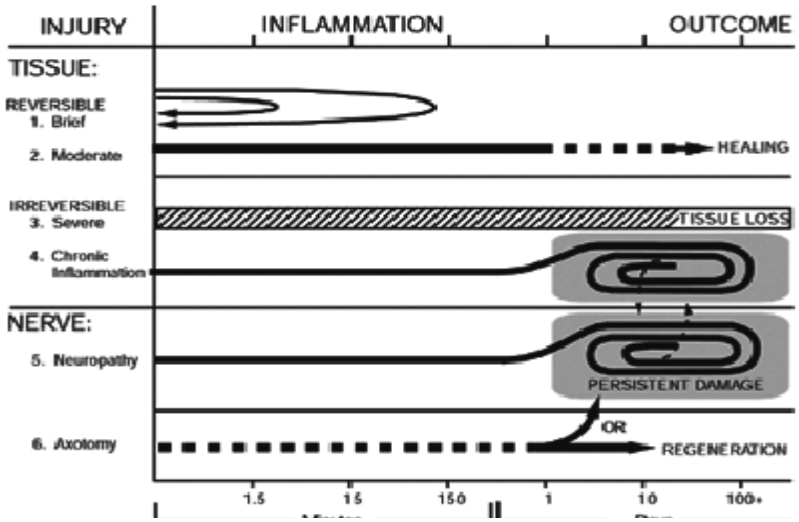

Figure 1 Six different outcomes of tissue or nerve injury are diagrammed in relation to a log time scale from minutes to more than 100 days ***(Byers &Bonica, 2001).***

How is pain transmitted?

The sequence of events by which a stimulus is transmitted & perceived involves four processes: 1.Transduction. 2.Transmission. 3. Modulation. 4. Perception.

Transduction occurs in the peripheral terminals of primary afferent neurons where different forms of energy, e.g. mechanical, heat or cold are converted to electrical activity (action potentials).

Transmission is the process by which electrical activity induced by a stimulus is conducted through the nervous system. There are three major components of the transmission system.

The peripheral sensory cells in the dorsal root ganglia (the first order neuron) transmit impulses from the site of transduction to the spinal cord where the central terminals synapse with the second order neurons. The spinal neurons are the second component in the transmission network. These cells send projections to various brainstem and diencephalic structures. Finally, neurons of the brainstem and diencephalon form the third component of the transmission network (the third order neuron) as they project to various cortical sites.

Modulation is the process whereby neural activity may be altered along the pain pathway. The dorsal horn of the spinal cord is one major site where modulation occurs involving a multitude of neurotransmitter systems.

Perception is the final stage of pain signaling process by which neural activity in the somatosensory transmission pathway results in a subjective sensation of pain. It is presumed that this process results from the concerted activation of the primary and secondary somatosensory and limbic cortices ***(Raja et al, 2006).***

Pain pathways

Nociceptors*(Figure 2)*

Nociceptoris a receptor preferentially sensitive to a noxious stimulus or to a stimulus that would become noxious if prolonged as suggested in 1994, whereas a new definition is now

being discussed by IASP that nociceptor is a sensory receptor that is capable of transducing and encoding noxious stimuli. Non-nociceptive receptors (e.g. tactile receptors, warm receptors) may respond to noxious stimuli (mechanical or thermal, respectively), when these stimuli are above their respective thresholds. But only nociceptors are capable of encoding the relevant properties of those stimuli (e.g. sharpness, heat intensity in the painful range). This is to clarify that a nociceptor is a peripheral nerve ending acting as a sensory receptor, where transduction into generator potentials and encoding into trains of action potentials take place ***(Loeser&Treede, 2008).***

All nociceptors are free nerve endings that have their cell bodies outside the spinal column in the dorsal root ganglion.

Nociceptors may have either:

1- Aδ fiber axons which are thin, myelinated axons with a rapid conduction velocity (2 to 30 m/s). These nerve fibers are associated with acute (sharp) pain and therefore constitute the afferent portion of the reflex arc that results in "pulling away" from noxious stimuli ***(Raja et al, 2006).***

2- Or more slowly conducting C fiber which are unmyelinated axons and as a result, have a slowerconduction velocity than other nerve fibers (2 m/s). These fibers are