Regional Analgesia for Postoperative Pain Control

Essay

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Mariam Momtaz Ameen Fawaz

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List of Abbreviations

ACLS	:	Advanced Cardiac Life Support
ASIS	:	Anterior superior iliac spine
ASRA	:	American society for regional Anesthesia
CNS	:	Central nervous system
cPNB	:	Continous peripheral nerve block
CSF	:	Cerebrospinal fluid
CVS	:	Cardiovascular system
FNB	:	Femoral nerve block
GA	:	General anesthesia
HTEA	:	High throracic epidural analgesia
II/IH	:	Ilioinguinal/iliohypogastric block
IPLA	:	Intraperitoneal local anesthetic
IT	:	Intrathecal
LA	:	Local anesthetic
LAST	:	Local Anesthetic Systemic Toxicity
LFCN	:	Lateral femoral cutaneous nerve
LMWHs	:	Low-molecular-weight heparins
PABA	:	Para-aminobenzoic acid
PCA	:	Patient controlled anesthesia
PCEA	:	Patient-controlled epidural analgesia
PNB	:	Peripheral nerve blocks
PNS	:	Peripheral nervous system
PT	:	Pubic tubercle
PVB	:	Paravertebral block
PVS	:	Paravertebral space.
sPNB	:	Single shot peripheral nerve block
TAP	:	Transversus abdominis plane

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Introduction

Although the medical world cannot cure every disease, the control of pain to ensure patient comfort should be a goal. One step toward improving pain management is through increased knowledge of pain physiology. Within the nervous system, there are several pathways that transmit information about pain from the periphery to the brain. There is also a network of pathways that carry modulatory signals from the brain and brainstem that alter the incoming flow of pain information ***(Renn and Dorsey, 2005).***

Acute pain is typically associated with neuroendocrinal stress response that is proportional to pain intensity. It has been hypothesized that a reduction in surgical stress response (endocrinal, metabolic and inflammatory) will lead to a reduced incidence of postoperative organ dysfunction and there by to an improved outcome ***(Richardson and Mustard 2009).***

Local anesthetic agents have a wide variety of applications. They are used as the backbone ingredients for local and regional anesthetic techniques and for analgesia in the operative and postoperative period. They are also used in the management of chronic pain where local anesthetic injections may have a prolonged effect. Modern local

anesthetics are safer then their predecessors, but risks persist. The cornerstone of safe practice is a thorough understanding of the pharmacology and toxicity of the agents used, in particular, dose and concentration required, speed of onset and duration of action. Clinicians administering local anesthetic agents must be capable of recognizing impending toxicity, and have access to the equipment, current knowledge and skills to manage these events ***(McLure and Rubin, 2005).***

Regional anesthesia is obtained by administering local anesthetics near the spinal cord and nerve roots (spinal, epidural), spinal nerves (paravertebral), or close to peripheral nerves. The same techniques are used for regional analgesia, but this is obtained by using more dilute solutions of local anesthetics, and other analgesic drugs are often added. Pain impulses are inhibited, but sensation of touch and muscle functions are intact. Regional analgesia gives superior relief of pain provoked by movement. This facilitates early postoperative mobilization of patients, even after major surgery in weak patients. For these patients optimally performed regional analgesia may reduce postoperative morbidity and mortality better than general anesthesia and opioid and non-opioid analgesics administered postoperatively ***(Breivik and Norum, 2010).***

Chapter 1
Pain physiology &Postoperative Complications

Introduction

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."***(Benzon et al., 2011)***

Thus, an understanding of the anatomic substrates and physiologic mechanisms by which noxious and non-noxious stimuli are perceived provides the essential background to apprehend the mechanisms of acute and chronic pain, and the sites of action of pharmacologic therapies for pain. ***(Brennan et al., 2007).***

Nociception is the physiologic process of activation of neural pathways by stimuli that are potentially or currently damaging to tissue. ***(Benzon et al., 2011)***

Pain in contrast to nociception, is a conscious experience. While the stimulus-induced activation of afferent neural pathways plays an important role, other factors such as alterations in somatosensory processing following injury to tissues and/ or nerves and psychosocial factors may influence the overall perception of pain. ***(Benzon et al., 2011)***

Pain pathway

The ascending pain pathways transmit nociceptive information from peripheral tissues to the cerebral cortex for interpretation as pain. The ascending pathways are complex structures, involving both the peripheral (PNS) and central nervous systems (CNS). ***(Brennan et al., 2007)***

Nociceptors

Specialized peripheral sensory neurons known as nociceptors alert us to potentially damaging stimuli at the skin by detecting extremes in temperature and pressure and injury-related chemicals, and transducing these stimuli into long-ranging electrical signals that are relayed to higher brain centers. The term noxious is applied to nociceptive stimuli because nociceptors are activated in response to strong stimuli that fall in the tissue-damaging range, whereas non-nociceptive mechanoreceptors, thermoreceptors, and chemoreceptors respond to milder stimuli that fall in a range below the tissue-damaging level. In addition to exogenous chemicals that stimulate nociceptors, a number of endogenous chemicals have been identified that can activate nociceptors, including

potassium, bradykinin, serotonin, histamine, prostaglandins, and others. ***(Adrienne and Adrem, 2010)***

First order neuron

When a noxious stimulus is transduced by a nociceptor, a signal is generated that is transmitted as an electrical action potential along small diameter A-delta (myelinated, fast transmission, sharp or pricking first pain) and C (unmyelinated, slow transmission, dull or burning second pain) primary afferent nerve fibers to the gray matter of the spinal cord. ***(Benzon et al., 2011)***.

On crosssection, the spinal gray matter forms a butterfly shape and can be divided into 10 laminae, or layers, which are numbered I through IX, from dorsal to ventral, with X surrounding the central canal. Pain processing occurs predominantly in laminae I, II, and V. The primary afferent fibers enter the spinal cord in the dorsolateral aspect of the gray matter (the dorsal horn) through the dorsal root. Upon entering the dorsal horn, the primary afferents bifurcate in a "T" pattern and travel 2 to 3 spinal segments within Lissauer'stract in both the cephalic and caudal directions. As the primary afferents travel in Lissauer's tract, they send collateral projections to the gray matter along the entire 4 to 6 segment length thus transmitting the pain signal over a broad

area of the spinal cord rather than to a discrete location. ***(Khasar et al., 2008)***

Second order neuron

The dorsal horn of the spinal cord is the site where the primary afferent fibres synapse with second-order neurons and convey the nociceptive message through the release of a variety of neurotransmitters, such as the excitatory amino acid glutamate or the peptide substance P. It is also where complex interactions occur between excitatory and inhibitory interneurons and where descending inhibitory tracts from higher centres exert their effect. After the nociceptive signal has been received in the dorsal horn, the information is transmitted to higher centers in the CNS by projection neurons. ***(Steed, 2013)***

Third order neuron

The projection neurons transmit the nociceptive signal rostrally along the ascending pathways in the spinal cord to various supraspinal structures in the brainstem and diencephalon, including the medullary reticular formation, periaqueductal gray, parabrachial region, hypothalamus, thalamus, and various limbic structures. The function of the ascending pathways is simply the transmission of the

nociceptive information. Within the supraspinal target structures of the ascending pathways, third order neurons further process the nociceptive signal and transmit it to cortical and limbic structures, where the signal is interpreted as pain. ***(Benzon et al., 2011)***

Cerebral Cortex

Ultimately, the nociceptive signal reaches the cerebral cortex where it is integrated and undergoes cognitive and emotional interpretation as stemming from a painful stimulus. The nociceptive signal is transmitted from the thalamus to a variety of cortical sites: the somatosensory S1 area and S2 area, the insular cortex, the anterior cingulate cortex, and the medial prefrontal cortex. Within these cortical regions, there is a complex network of interconnections that include the thalamus and limbic structures. This network of cortical structures is responsible for the sensory-discriminative (perception of the intensity, location, duration, temporal pattern, and quality of noxious stimuli) and motivational-affective (relationship between pain and mood, attention, coping, tolerance, and rationalization) components of the pain experience. ***(Khasar et al., 2008)***

The sequence of events by which a stimulus is perceived involves four processes: (1) transduction, (2) transmission, (3) modulation, and (4) perception.

1) ***Transduction***is the process by which afferent nerve endings participate in translating noxious stimuli (e.g., a pinprick) into nociceptive impulses. Their receptors detect mechanical, thermal, proprioceptive, and chemical stimuli. ***(Zacharoff, 2010)***.

2) ***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 transmit impulses from the site of transduction at their peripheral terminal to the spinal cord where the central terminals synapse with second order neurons. The spinal neurons are the second component in the transmission network. These cells send projections to the thalamus and various brainstem and diencephalic structures. Finally, neurons of the brainstem and diencephalon form the third component of the transmission network as they project to various cortical sites. ***(Pasternak, 2010)***.