Recent Advances in post-operative Pediatric Pain Management

Essay

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بسم الله الرحمن الرحيم

قَالُواْ سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ

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Yasser Sayed Sobhy El-Sayed

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List of Abbreviations

CEA	: Continuous epidural anesthesia
CGRP	: Calcitonin gene-related peptide
CI	: Cognitively impaired
CPR	: Cardiopulmonary resuscitation
D10W	: Dextrose 10 % in water
DRG	: Dorsal root ganglion
EO	: External oblique
FA	: Femoral artery
FDA	: Food and Drug Administration
FN	: Femoral nerve
FV	: Femoral vein
GABA	: Gamma-aminobutyric acid
GSH	: Glutathione
HSP	: Hockey Stick Probe
MCN	: Musculocutaneous nerve
NCA	: Nurse controlled analgesia
NMDA	: N-methyl-D-aspartate
NSAIDs	: Non-steroidal anti-inflammatory drugs
PCA	: Patient controlled analgesia
PCEA	: Patient controlled epidural anesthesia
pKa	: Negative base-10 logarithm of the acid dissociation constant of a solution
PNB	: Peripheral nerve block
PONV	: Postoperative nausea and vomiting
RS	: Rectus sheath
TAP	: Transversus abdominis plane
US	: Ultrasound
USG	: Ultrasound guidance
5-HT3	: Serotonin

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Introduction

Children suffer postoperative pain in the same way as adults. Frequently, factors such as fear, anxiety and lack of social support can exaggerate the physical pain in children further. The need for analgesics following surgery depends upon the nature of the procedure and the pain threshold of the patient. It does not depend on the age, or on whether the child is an inpatient or an outpatient ***(Verghese and Hannallah, 2005).***

Pain assessment is a critical component of pain management. Assessing pain in young children, however, can be challenging. Self-reporting is only possible in older children or those with considerable cognitive and communicative abilities. Therefore, several tools and pain assessment scales should be available to provide qualitative and quantitative information and documentation of pain. The measurement of pain in infants, young children, and children with developmental disabilities who are unable to self-report is particularly challenging and needs increased attention ***(O'Rourke, 2004).***

The management of postoperative pain in children is based on a combination of local anaesthetic agents, simple analgesics, non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. The use of multimodal analgesia reduces hospital stay, decreases medical complications, and increases patient satisfaction***(Llewellyn et al., 2000; Skinner, 2004).***

The limited duration of analgesia after single blocks can be prolonged by use of adjuvants (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. Patient-controlled analgesia are convenient ways for opioid administration in infants and children***(Sumpelmann and Munte, 2003).***

Adequate pediatric pain management may improve both short- and long-term health outcomes for many years following surgical intervention. Currently, there are many reliable agents and techniques available to provide a safe and effective postoperative analgesia even in neonates and small infants ***(Gold et al., 2006)***.

Aim of the Work

The objective of this Essay is to discuss current practice and recent advances in pediatric post-operative pain management in order to achieve safe and adequate analgesia for all patients.

Chapter (1):
Pathophysiology 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 American Pain Society (APS) in its Policy Statement on pain in children defines pain as “The result of a dynamic integration of biological processes, psychological factors and social/cultural factors considered within a developmental trajectory” ***(Dubin and Patapoutian, 2010).***

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 Patapoutian, 2010).***

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 Aδ fibres that enter the dorsal horn of the spinal cord and synapse at laminae I, V and X. Conduction continues along the secondary afferent fibres 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).***