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ULTRASOUND GUIDED CONTINUOUS PERIPHERAL NERVE BLOCKS IN ACUTE PAIN MANAGEMENT FOR TRAUMA PATIENT

Essay

Submitted For Partial Fulfillment of Master Degree in Anesthesia

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Hany Attia Al-Meligy

Introduction

Continuous nerve blocks represent an important therapeutic tool in managing perioperative pain and trauma pain. The indications for continuous nerve blocks for the perioperative pain management in hospitalized and ambulatory patients have extended well beyond orthopaedics. These techniques are not only used to control pain in patients undergoing major upper and lower extremity surgery, but also to provide perioperative analgesia in patients undergoing abdominal, plastic, urological, gynaecological, thoracic, and trauma surgeries. Infusion regimens of local anaesthetics and supplements must take into consideration the condition of the patient before and after surgery, the nature and intensity of the surgical stress associated with the surgery, and the possible need for immediate functional recovery (*Ludot et al.*, 2008).

Continuous nerve blocks have proved safe and effective in reducing opioid consumption and related side-effects, accelerating recovery, and in many patients reducing the length of hospital stay. They provide a safer alternative to epidural analgesia in patients receiving thromboprophylaxis, especially with low molecular-weight heparin (*Ivani and Mossetti*, 2010).

Recently, the use of ultrasound-guided techniques, alone or in combination with a neurostimulation approach, has been advocated (e.g. for femoral, gluteal, popliteal, upper extremity, and thoracic paravertebral blocks). Comparison of these

1

techniques supports the concept that the use of an ultrasound-guided technique reduces the time necessary for the placement of the perineural catheter and reduces vascular punctures, need for opioids, and the volume of local anaesthetics (*Aveline et al.*, 2010).

AIM OF THE WORK

This work presents highlights new insights into advantages, indications and guidance during procedures for continuous peripheral nerve blocks in acute pain management for trauma patient.

Chapter (1)

ANATOMY AND PHYSIOLOGY OF PAIN

Pain is defined by the International Association for the Study of Pain (IASP) as an unpleasant sensory and emotional experience that is primarily associated with tissue damage. It is a protective mechanism that occurs whenever tissues are being damaged, and it causes the individual to react to remove the pain stimulus (*Tenti and Hauri*, 2004).

Pain Pathway

Specificity theory states that there is a specific pain system that transfers information about potential or actual tissue damage to the place of perception (the brain). Nociceptive energy is transduced into electrophysiological signals that are transmitted to perceptive apparatus. However, the pain pathway is not 'hard wired', but undergoes profound functional changes and modulation under certain conditions, such as tissue damage and inflammation (e.g. postoperative pain). This plasticity is mediated by many mechanisms, including peripheral/primary and central/secondary sensitization. The substrate for these changes is a plethora of chemical mediators peripherally and spinally, comparable in complexity to neurotransmitters in the brain (Smith, 2007).

I- Primary afferent neurons:

There are three classes of primary afferent fibers in skin that may be activated by a given cutaneous stimulus. The fibers that are largest and have the fastest conduction velocity are the large-diameter myelinated (A β) fibers. These fibers, when activated, do not normally result in a sensation of pain, but rather of light touch, pressure, or hair movement. The axons of the nociceptive neurons are generally unmyelinated (C fibers) or thinly myelinated (A δ fibers) (*Dubner*, 1994).

Unmyelinated C polymodal nociceptors are activated by many potentially tissue-damaging modalities, are associated with prolonged 'burning' pain, and are slowly conducting (0.5–2.0 m/s). Some may have a differential sensitivity to heat or mechanical stimuli (*Smith*, 2007).

The Aδ is thinly myelinated, mechano-heat receptors that thought to mediate a briefer 'sharp' pain. These larger fibers are more rapidly conducting (5–20 m/s). Aδ fibers are also delineated into two types, depending on their differential responsiveness to intense heat. A final group of nociceptors do not appear to exhibit sensitivity to noxious stimuli. These 'silent' nociceptors develop novel sensitivity usually after tissue injury or inflammation. Silent nociceptors have been well characterized in the visceral domain, although there is some evidence to support the existence of somatic counterparts (*Smith*, 2007).

II- Spinal cord to brain:

Secondary afferents decussate and pass up the spinal cord to the midbrain via the spinothalamic, spinoreticular and spinomesencephalic tracts to the thalamus and to sensory cortex, but also have many other links, such as to reticular formations, limbic and hippocampus areas, figure (1). The different pathways may have functional correlates involving memory, cognition and emotion, which contribute to the neural network of overall pain perception. Moreover, neurons that project from these areas of the brain provide descending modulation of spinal cord processing (*Brooks and Tracey*, 2005).

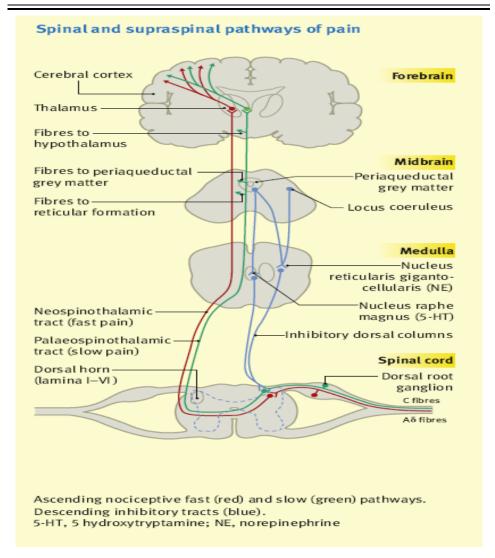


Fig. (1): Spinal and supraspinal pathways of pain (Smith, 2007)

The first synapse in somatosensory signaling occurs either at the spinal dorsal horn or in the dorsal column nuclei at the spinal cord-brain stem junction. Evidence has accumulated to indicate that both nociceptive and non nociceptive fibers provide input to both of these initial targets. However, under normal circumstances, the dorsal column nuclei can be considered to selectively process inputs from the large myelinated fiber classes