Pain control after orthopaedic surgery with nerve block: Current trends

Essay

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INTRODUCTION

Postoperative pain is a common reason for the delayed discharge and unanticipated hospital readmission of outpatients, the pain associated with most orthopaedic surgery is typically intense, making its control is essential. In addition to its humanitarian aspects, effective pain control improves surgical outcome ***(Carli, et al. In 2011)***, shortens hospital stay and rehabilitation, decreases the development of new chronic pain conditions ***(Lenart, et al. In 2012)***, and may [by decreasing the surgical stress response and reducing the negative influence of opioids and anaesthetic agents on the natural killer (NK) cells] contribute to slower progression of certain cancers ***(Biki, et al. in 2008).***

The trend in the last 10 years towards outpatient surgery and faster rehabilitation has created more interest in methods of postoperative pain control that will decrease the dependency on narcotics and their adverse effects such as drowsiness, nausea, constipation and malaise ***(Carli, et al. In 2011).***

The superiority of regional anaesthesia to all other modalities of acute pain management cannot be doubted. However, one of the major problems with regional anaesthesia is that the acute pain

usually outlasts the nerve block. To counter this problem, various manoeuvres and techniques to increase the duration of single injection nerve blocks have been developed and tried over the years. Local anaesthetics with prolonged duration of action appear imminent. Trials of an extended-release liposome, encapsulated form of bupivacaine, are appearing in the literature ***(Onel, et al.in 2011).***

Another method is the continuous peripheral nerve blockade, also called "perineural local anaesthetic infusion". It involves the percutaneous insertion of a catheter directly adjacent to the peripheral nerve supplying a surgical site. Local anaesthetic is then infused via the catheter providing site-specific analgesia ***(Boezaart, et al. in 1999).***

Other agents have been employed as adjuvants in peripheral nerve block with variable or modest success. Among those discussed in recent publications are dexamethasone, magnesium sulfate, midazolam, ketamine, buprenorphine, tramadol, and clonidine ***(Johr, Berger. In 2012).***

Multimodal analgesia has become increasingly relevant to the practice of perioperative pain medicine in the past decade. It is well established that ketamine, NSAIDs, acetaminophen, steroids, gabapentin, and other agents can improve perioperative pain relief

and compensate for the shortcomings of single-injection or continuous nerve blocks ***(Young, Buvanendran. in 2011).***

Dexamethasone deserves special mention as a multimodal agent. Not only is it a proven Antiemetics, it is also efficacious as a systemically administered analgesic ***(De Oliviera, in 2011).***

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List of Abbreviations

ACL	anterior cruciate ligament.
ASA	American Society of Anesthesiologists
ASICs	Acid-Sensing Ion Channels
BAB	Butyl amino-benzoate.
BDNF	brain-derived neurotrophic factor.
BP	brachial plexus.
CCK	cholecystokinin.
CNS	centeral nervas system.
COX-3	cyclo-oxygenases.
coxibs	Cyclo-oxygenase-2 selective inhibitors.
CPNB	continuous peripheral nerve block.
CVS	Cardiovascular system.
D.H.S	dynamic hip screw.
FDA	the Food and Drug Administration.
GABA	gamma-amino butyric acid.
HCl	hydrochloric acid.
IASP	The International Association for the Study of Pain.
IL-1	Interleukins 1.
IV	intra venous.
IVRA	intra venous regional anaesthesia.
LA	local anaesthetic.
LE	lipid emulsion.
MHz	megahertz.
NGF	neurotrophic groth factors.
NK	The natural killer cells.
NMDA	N-methyl-D-aspartate.
NRM	nucleus raphe magnus.
NRS	the numeric rating scale.
NSAID	Non-Steroidal Anti-Inflammatory drugs.
nsNSAIDs	Non-selective Non-Steroidal Anti-Inflammatory drugs.
ORL1	opioidreceptor- like.
PABA	para-aminobenzoic acid.
PCA	patient controlled analgesia.
PG	prostaglandin.
RCTs	randomized controlled trials.
SCM	sternocleidomastoid.
sP	substance P.

List of Abbreviations

TKA	Total Knee Arthroplasty.
TNF-α	Tumour necrosis factor α.
trkA, B	tyrosine kinase receptors A & B.
TRPV1	**T**ransient **R**eceptor **P**otential **V**anilloid
US	ultrasound.
VIP	vasoactive intestinal peptide.
WDR	wide dynamic range.

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Aim of the work

The purpose of this review is to outline methods of increasing the duration of postoperative (specially post orthopaedic surgery) pain control, which are: the recent advances in the use of perineural catheters for the performance of continuous nerve blocks, the use of adjuvants to extend the duration of single dose blocks, systemic multimodal analgesia, and novel or experimental agents.

PATHOPHYSIOLOGY OF PAIN AND PAIN PATHWAY

Pain

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience which is primarily 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 a noxious event and is influenced by emotion, cognition, memory, interpersonal and social context and other factors. One influential conceptual model describes pain in terms of three hierarchical levels: a sensory-discriminative component (e.g., location, intensity, quality), a motivational–affective component (e.g., depression, anxiety), and a cognitive-evaluative component (e.g., thoughts concerning the cause and significance of the pain) ***(Rolf, 2006).***

Nociception

Nociception is the sensory modality by which noxious stimuli are detected peripherally, and transmitted centrally to the central nervous system. Noxious stimuli may or may not be associated with tissue damage ***(Willis, 2007).***

▪ Nociception and inflammatory mediators

When a noxious stimulus is applied, chemical mediators such as prostaglandins, hydrogen ions and kinins are released by the nociceptor, or as a result of tissue damage. These substances not only initiate nociception but also produce hyperalgesia. In addition to these effects in the pain response, these agents mediate the inflammatory process by inducing the following changes:

- Increases in local blood flow and vascular permeability.
- Activation and migration of immune cells.
- Release of growth and trophic factors from surrounding tissues ***(Stein & Clark, 2009).***

There are different mechanisms by which inflammatory mediators influence nociceptor function:

- Activation of membrane ion channels, e.g. lipid metabolite activation of TRPV1 channels, proton activation of ASICs, ATP activation of purinergic (P2X) channels.
- Activation of G-protein coupled receptors which increase the activities of second messengers such as cAMP or inositol triphosphate (IP3). These in turn may lead to structural changes in membrane ion channels, or changes in the activity of intracellular enzymes affecting membrane excitability.
- Activation of cytokine receptors by interleukins (IL-1), or tumour necrosis factor α (TNF-α).
- Activation of tyrosine kinase receptors trkA, trkB, by specific neurotrophic factors such as NGF (which activates trkA) and

brain-derived neurotrophic factor, BDNF (which activates trkB) ***(Stein C & Clark JD,2009).***

▪ Pain pathways

Pain is conducted along three-neuron pathways that transmit noxious stimuli from the periphery to the cerebral cortex (Fig. 1). Primary afferent neurons are located in the dorsal root ganglia, which lie in the inter-vertebral foramina at each spinal cord level. Each neuron has a single axon that bifurcates, sending one end to the peripheral tissues it innervates and the other into the dorsal horn of the spinal cord. In the dorsal horn, the primary afferent neuron synapses with a second-order neuron whose axons cross the midline and ascend in the contralateral spinothalamic tract to reach the thalamus. Second-order neurons synapse in thalamic nuclei with third-order neurons, which in turn send projections through the internal capsule and corona radiata to the postcentral gyrus of the cerebral cortex ***(Guyton and Hall, 2000).***

- ***First-order neurons***

The majority of first-order neurons send the proximal end of their axons into the spinal cord via the dorsal spinal root at each cervical, thoracic, lumbar, and sacral level. Once in the dorsal horn, in addition to synapsing with second-order neurons, the axons of first-order neurons may synapse with interneurons, sympathetic neurons, and ventral horn motor neurons ***(Dubner, 1994).***

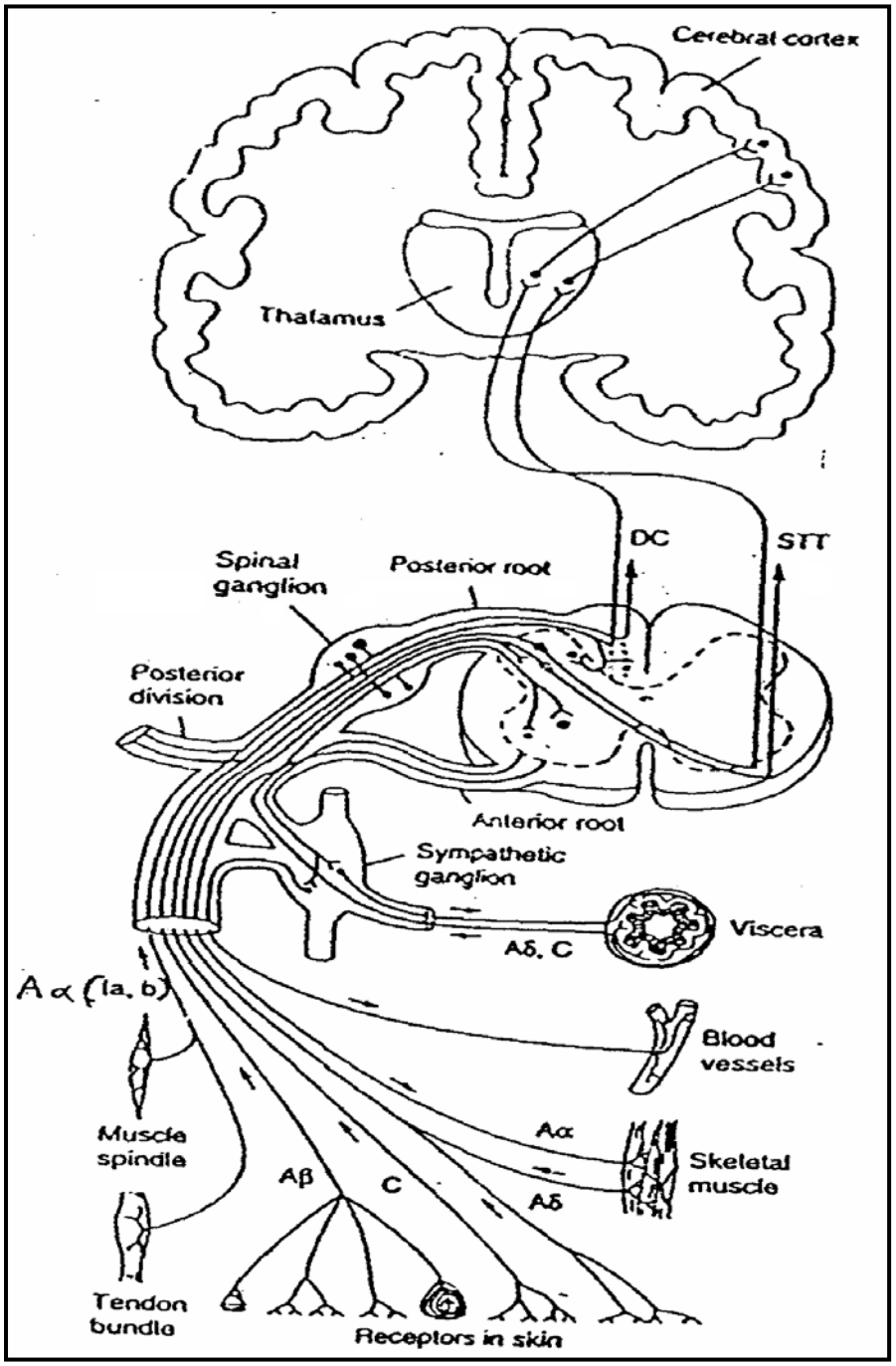

Fig. (1): Pain pathways ***(Adams and Victor, 1993).***