Efficacy and safety of thoracic paravertebral block in breast surgery: Blind-landmarks- technique vs Ultrasound guided technique

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ABSTRACT

Key words: PVB, US, breast, LA, Perioperative pain

This study demonstrates that, ultrasound guided thoracic paravertebral block (TPVB) is an effective intraoperative and postoperative technique for surgical anaesthesia and analgesia for breast surgery . It offers a long-lasting effective analgesia with a significant decrease in anaesthetic and analgesic consumption, and with a high degree of patient satisfaction, decrease the incidence of PONV and shorten length of hospital stay. Ultrasound guidance helps identifying the paravertebral space (PVS), needle placement, and to real time monitor the spread of the local anesthetic around nerves which increase the efficacy of the block and minimize the risk of complications.

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LIST OF ABBREVIATIONS

5-HT3	:	5-Hydroxytryptamine3
ASA	:	American Society of Anesthesiologists
CNS	:	Central Nervous System
COX 2	:	Cyclooxygenase 2
DBP	:	Diastolic blood pressure
DVT	:	Deep venous thrombosis
ECG	:	Electrocardiogram
ETCO2	:	End tidal carbon dioxide
HR	:	Heart rate
HS	:	Highly significant
IL	:	Interleukins
MBP	:	Mean blood pressure
MHz	:	Megahertz
NRM	:	Nucleus raphe magnus
P	:	Pleura
PACU	:	Post anaethesia care unite
PCA	:	Patient Controlled Analgesia
PCEA	**:**	patient controlled epidural analgesia
PGE 2	:	Prostaglandin E2
PONV	:	Postoperative nausea and vomiting.
RAS	:	Reticular activating system
SBP	:	Systolic blood pressure
SCTL	:	Superior costotransverse ligament.
SD	:	Standard deviation
SSR	:	Surgical stress response
TP	:	Transverse process
TPVB	:	Thoracic paravertebral block
TPVS	:	Thoracic paravertebral space.
US	:	Ultrasound
VAS	:	Visual Analog Scale

INTRODUCTION

Thoracic paravertebral block is a century old technique used for intra-operative and peri-operative pain control as well as acute and chronic non-operative pain[1].It is technique of injecting local anaethetic in the vicinity of the thoracic spinal nerve emerging from the intervertebral foramen result in sympathetic ,sensory block and motor blockade to less extent [2].

Clinically, thoracic paravertebral blockade can be unilateral or bilateral according to the analgesic need [3].

Many practitioners, however, remained hesitant to perform thoracic paravertebral block for fear of pneumothorax, it is reported that the risk of it doesn't exceed 0.5%-1%. the risk of Dural puncture is also reported but it is more common with median approach.[4]

Recently with the growth of ultrasound technology, our ability to visualize the pleura and other structures in and around the paravertebral space has fueled a tremendous increased interest in performing thoracic paravertebral block[5].

TPVB performed safely for the patients undergoing breast surgery, this technique provides an adequate anesthesia for the patients undergoing breast surgery and in addition provides stable hemodynamic status with somatic and sympathetic blockade, near-perfect control of postoperative pain, minimal nausea and vomiting rate, early discharge and low cost . For this reason, thoracic paravertebral block which is a standard method in breast surgeries for some centers should be known by all anesthesiologists[6].

We believe that, thoracic paravertebral block is a method can be applied instead of general anesthesia.

AIM OF THE WORK

The purpose of this study is to determine efficacy and safety of multiple injections TPVB in breast surgery using (blind technique) versus (ultrasound guided technique) .

Chapter1

Pathophysiology of acute pain

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage. This definition emphasizes that pain is not a predicted physiological response to stimulus nor based on observation, but is always subjective with each individual learning about pain through experiences relating to tissue injury in early life when pain is reported in terms of intensity, location and sometimes quality**.** This is referred to as the sensory- discriminative component of pain. Many people report pain in the absence of tissue injury and this pain may be amplified by psychological, emotional, cognitive and social factors as well as learned behavior**.**[8]

Surgical pain is due to inflammation from tissue trauma (i.e., surgical incision, dissection, burns) or direct nerve injury (i.e., nerve transaction, stretching, or compression) **.**Tissue injury leads to release of inflammatory mediators with subsequent nociceptor stimulation. Pain impulses are then transmitted to the dorsal horn of the spinal cord, where they make contact with second-order neurons that cross to the opposite side of the cord and ascend via the spinothalamic tract to the reticular activating system (RAS) and thalamus. The localization and meaning of pain occurs at the level of the somatosensory cortex (Fig.1).[9]

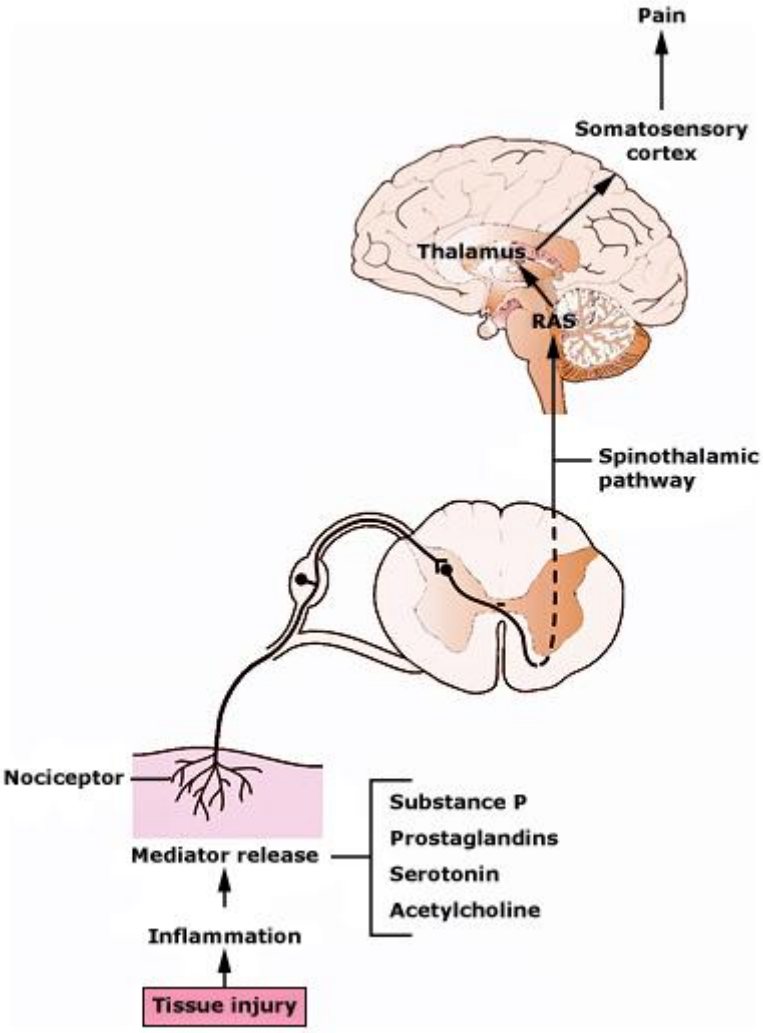

Figure 1: mechanism of acute pain.[10]

Tissue trauma releases local inflammatory mediators (e.g. histamine, bradykinin, and substance P) (Fig.2) that can produce augmented sensitivity to stimuli in the area surrounding an injury (hyperalgesia) or misperception of pain to non-noxious stimuli (allodynia). Other mechanisms contributing to hyperalgesia and allodynia include sensitization of the peripheral pain receptors (primary hyperalgesia) and increased excitability of central nervous system neurons (secondary hyperalgesia)**.**[11]

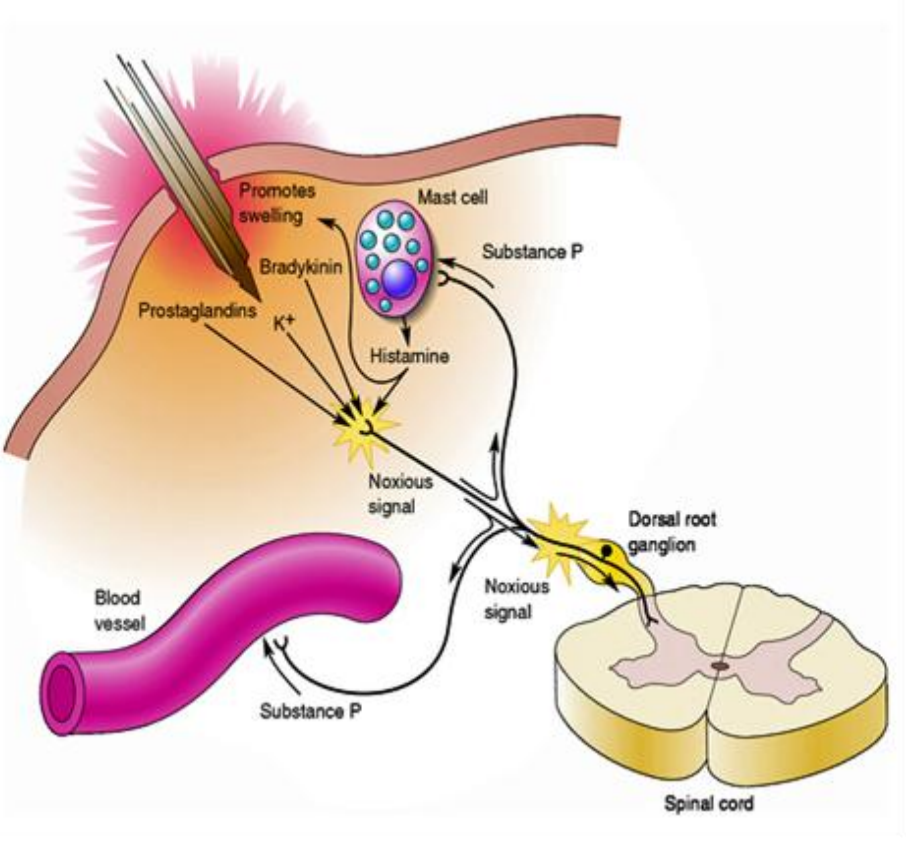

Figure 2: Peripheral chemical mediators of pain.[12]

Surgery-associated tissue injury and peripheral nerve injury sets off a cascade of related events, including nociception and inflammatory reaction ,accompanied by elevated levels of pro-inflammatory cytokines including interleukins(IL-1β and IL-6). These cytokines can induce peripheral and central nervous system sensitization leading to pain augmentation (hyperalgesia).[12]Peripheral, IL-1β induces long- lasting synthesis and release of substance P from peripheral nerve terminals of primary afferent neurons, which may contribute to neurogenic inflammation.[13]Within minutes of injury, glial cells in the central nervous system respond with increased production of immune factors, including proinflammatory cytokines.

IL-1β can induce central sensitization via IL-1 receptors on neurons or via activated glia cells, which produce pain mediators, including substance p, glutamate, and nitric oxide syntheses, all of which can alter pain processing within the central nervous system . [13]

Elevated IL-1β in the central nervous system also leads to the production of cyclooxygenase2 (COX2),which is a prostaglandin responsible of flaring inflammation and pain, by neurons in the brain and the spinal cord and further synthesis of prostaglandins E2 (PGE2) which is known to increase pain sensitivity .Similarly, IL-6 levels are also elevated following nerve injury, both peripherally and centrally, contributing to hyperalgesia by direct spinal nociceptive mechanisms or by glial activation. IL-1β and IL-6 are also involved in the mechanisms of allodynia and possibly in the development of postoperative neuropathic and chronic pain.[14]

Analgesic therapy has traditionally targeted central mechanisms involved in the perception of pain using opioids (Fig.3); however, it has become clear that a better approach uses several agents, each acting at different sites of the pain pathway. This approach lessens the dependence on a given medication and mechanism.

The transmission of incoming nociceptive impulses is modulated by dorsal horn circuitry that receives input from peripheral touch receptors and from descending pathways that involve the limbic cortical systems (orbital frontal cortex, amygdala, and hypothalamus), periaqueductal endogenous analgesic center in the midbrain, pontine noradrenergic neurons, and the nucleus raphe magnus (NRM) in the medulla.[15]

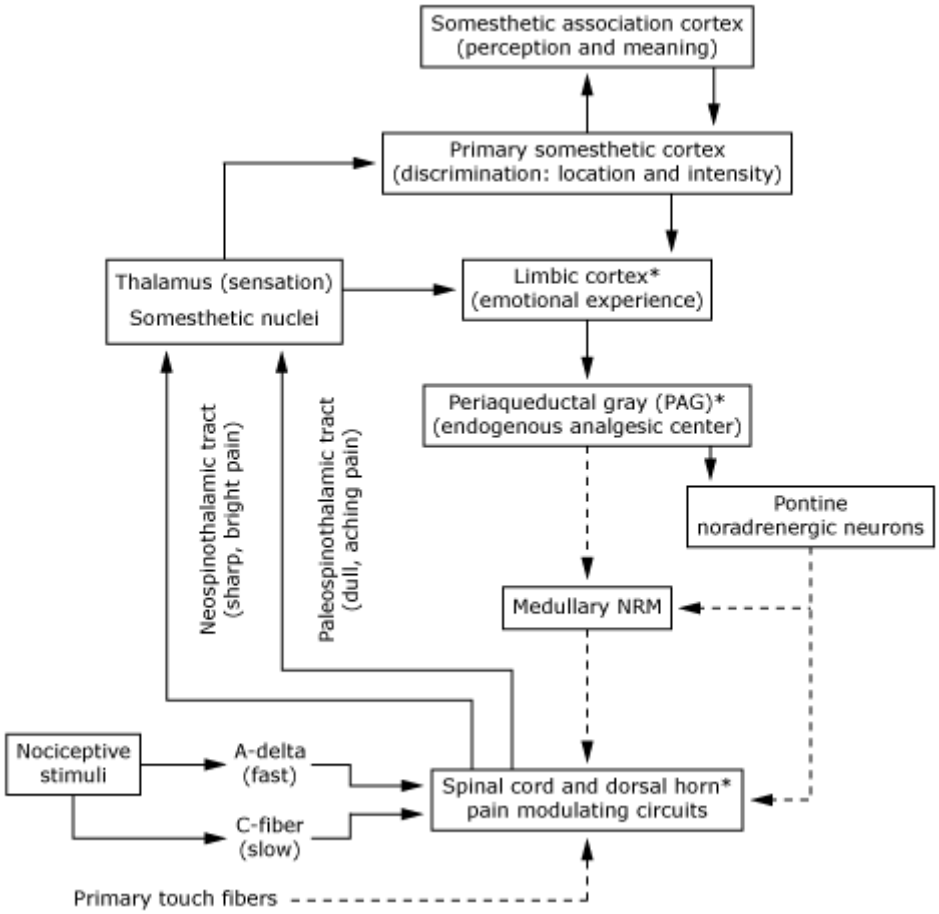

Figure 3: Central perception of pain Dashed lines indicate inhibition or modulation. (*Location of opioid receptors)**.**[12]

Axon terminals form synapses with the dendrites or somata of other neurons. When a nerve impulse arrives in the presynaptic axon terminal, neurotransmitter molecules are released from synaptic vesicles into the synaptic cleft. Neurotransmitter then binds to specific receptor proteins, causing the generation of electrical or chemical signals in the postsynaptic cell (Fig.4).[12]