

Supraclavicular brachial plexus nerve block versus patient controlled analgesia for post- operative pain management in forearm surgery.

Thesis

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List of Abbreviations

AXI	: Axillary nerve
BP	: Brachial plexus
CNS	: Central nervous system
DBP	: Diastolic blood pressure
HR	: Heart rate
LAs	: Local anesthetics
LAST	: Local Anesthetics Systemic Toxicity
MC	: Musculocutaneous nerve
MED	: Median nerve
MSM	: Middle scalene muscle
PCA	: Patient controlled analgesia
PNB	: Peripheral nerve blockade
PNS	: Peripheral nerve stimulation
RAD	: Radial nerve
SA	: Subclavian artery
SBP	: Systolic blood pressure
SD	: Standard deviation
ULN	: Ulnar nerve
US	: Ultrasound

List of Symbols

\square : Sum

N : Number of observations

% : Percentage

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Introduction

Inadequate postoperative pain management has been correlated with poor functional recovery in some patients (*Capdevila et al., 1999*), and can activate a variety of biologic cascade systems, resulting in ileus, nausea, delayed mobilization and feeding, delayed hospital discharge, and unanticipated hospital readmission (*Kehlet, 1994*). Opioids are considered the cornerstone for treatment of moderate-to-severe acute postoperative pain, and PCA is the most frequent mode of postoperative opioid administration (*Carr et al., 1998*). However, potent opioids result in potential side effects, including ventilatory depression, drowsiness, sedation, nausea, vomiting, pruritus, urinary retention, ileus, and constipation are frequently observed during opioid PCA (*White, 2002*). Because of these unwanted adverse effects, PCA is often discontinued despite insufficient pain management (*Gehling et al., 2011*). Patients consider nausea and vomiting to be the most undesirable postoperative complications (*Macario et al., 1999*).

Postoperative analgesia with fewer side effects is not only important for the patient but is also important for the surgeon. Brachial plexus block offers many advantages over general anesthesia for upper extremity surgery, including reduced surgical stress response, increased blood flow to the extremity (sympathectomy), better postoperative analgesia, earlier discharge for outpatients, and fewer side effects (*Singelyn, 2000*). The classical approaches (interscalene,

supraclavicular, infra-clavicular, and axillary) have been described for many years (*Singelyn, 2000*).

Supraclavicular Brachial plexus block is an excellent method for attaining optimal operating conditions for upper limb surgeries by producing complete muscular relaxation, maintaining haemodynamic stability and the associated sympathetic block. They also provide extended postoperative analgesia with minimal side effects. In addition, it offers a better preservation of mental functions in elderly; decreased risk of aspiration due to intact pharyngeal and laryngeal reflexes; avoids difficult intubation; decreases postoperative complications associated with intubation and provides better postoperative analgesia without undue sedation facilitating early mobilization and discharge (*Chandni et al., 2013*).

Aim of the Work

The aim of this study is to compare Supraclavicular Brachial plexus nerve block to patient controlled analgesia for postoperative pain management in forearm surgeries. Therefore, we performed a randomized study to compare the efficacy of Supraclavicular Brachial plexus nerve block with that of patient controlled analgesia.

Pathophysiology of Pain

An Overview of Pain:

The International Association for the Study of Pain (IASP) describes pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” When unexpected exposure to potentially harmful stimuli occurs, pain manifests as a reflexive withdrawal response accompanied by a motivational reaction, most frequently a feeling of unpleasantness. The sensory process of detecting the “actual or potential tissue damage” is called nociception (*Kyranou & Puntillo, 2012*).

Pain is often described as being superficial, deep, or visceral:

- **Superficial somatic pain** arises from skin, subcutaneous tissues, and mucous membranes and is often described as a sharp, pricking, throbbing, or burning sensation.
- **Deep somatic pain** is associated with muscles, tendons, joints, or bones and usually has a dull, aching quality and is less well-localized.
- **Visceral pain** arises from disease or dysfunction of internal organs or their coverings (parietal pleura, pericardium, or peritoneum). True visceral pain is dull, diffuse, and usually midline (*Kyranou and Puntillo, 2012*).

Once a nociceptor is activated, a nerve impulse travels via sensory nerves to the spinal cord where it is quickly shunted to the brain via nerve tracts in the spinal cord and brainstem. The brain processes the pain sensation and responds via the motor

pathways, providing the correct motor response to cease the action causing the pain (*IOM, 2011*).

There are two types of nerve fibers associated with the transmission of pain: small, unmyelinated C fibers and myelinated A δ fibers. C fibers conduct impulses slowly and respond to thermal, mechanical, and chemical stimuli. A δ fibers conduct impulses rapidly and respond to mechanical (pressure) stimulus (*IOM, 2011*).

One of the most important central pain pathways is the **spinothalamic tract**, which originates in the spinal cord and extends to the thalamus. It transmits sensory information related to pain, temperature, and crude touch. Another prominent pathway, the **spinoreticular tract**, is involved in nociceptive processing. The spinoreticular tract and the spinothalamic tract are excited by similar sensory fibers. Rather than ascend to the thalamus, however, spinoreticular neurons terminate within the brainstem reticular formation (*Craig, 2010*).

Peripheral, IL-1 β induces long- lasting synthesis and release of substance P from peripheral nerve terminals of primary (1ry) afferent neurons, which may contribute to neurogenic inflammation (*Ni Choileain and Redmond, 2006*).

Elevated IL-1 β in the central nervous system also leads to the production of cyclooxygenase2 (COX2), which is 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 (*Bedwell et al., 2014*).

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-1 β and IL-6 are also involved in the mechanisms of allodynia and possibly in the development of postoperative neuropathic and chronic pain (*Minett et al., 2014*).

The Endocrine Response to Surgery:

The stress response to surgery is characterized by increased secretion of pituitary hormones and activation of the sympathetic nervous system. The changes in the pituitary secretion have secondary effects on hormonal secretion from target organs (**Table1, and 2**).

The overall metabolic effect of the hormonal changes is increased catabolism to provide energy sources, and a mechanism to retain salt and water to maintain fluid volume and cardiovascular homeostasis (*Moor et al., 2017*).

Table 1: Systemic responses to surgery.

- | |
|--|
| <ul style="list-style-type: none">• Sympathetic nervous system activation• Endocrine “ stress response”• Pituitary hormone secretion• Insulin resistance• Immunological and hematological changes• Cytokines production• Acute phase reaction• Neutrophil leukocytosis• Lymphocyte proliferation |
|--|

(*Moor et al., 2017*)

Table (2): Principal hormonal responses to surgery

<i>Endocrine Gland</i>	<i>Hormone</i>	<i>Change in Secretion</i>
Anterior pituitary	ACTH Growth hormone TSH, FSH, & LH	Increase Increase May increase or decrease
Posterior pituitary	AVP	Increase
Adrenal cortex	Cortisol, Aldosterone	Increase
Pancreas	Insulin Glucagon	Often decrease Usually small increase
Thyroid	Thyroxin	Decrease

ACTH; Adreno cortico-trophic hormone, TSH; Thyroid-stimulating hormone, FSH; follicle-stimulating hormone, LH; luteinizing hormone, AVP; arginine vasopressin.

(Moor et al., 2017).

Interaction between the Immune System and the Neuro-Endocrine System:

The cytokines IL-1 and IL-6 can stimulate secretion of ACTH from isolated pituitary cells *in vitro*. In patients after surgery, cytokines may augment pituitary ACTH secretion and subsequently increase the release of cortisol. A negative feedback system exists, so that glucocorticoids inhibit cytokine production. The cortisol response to surgery is sufficient to depress IL-6 concentrations (Marik and Flemmer, 2012).