Efficacy of Saphenous nerve block in comparison to analgesics requirements for post operative pain after arthroscopic ACL repair

Thesis
Submitted for Partial Fulfillment of Master Degree
in Anesthesiology

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2017

بسم الله الرحمن الرحيم

وقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ ورَسُولُهُ والْمُؤْمِنُونَ

صدق الله العظيم

سورة التوبة آية (١٠٥)

Acknowledgement

*First of all, all gratitude is due to **Allah**almighty for blessing this work, until it has reached its end, as a part of his generous help, throughout my life.*

*Really I can hardly find the words to express my gratitude to **Prof. Dr. Amr Mohamed Abd EL Fatah Sayed,** Professor of Anesthesia, Intensive Care and Pain management, Faculty of Medicine - Ain Shams University, for his supervision, continuous help, encouragement throughout this work and tremendous effort he has done in the meticulous revision of the whole work. It is a great honor to work under his guidance and supervision.*

*I would like also to express my sincere appreciation and gratitude to **Dr. Mahmoud Hassan Mohamed Hassan**, Assistant professor of Anesthesia, Intensive Care and Pain management, Faculty of Medicine - Ain Shams University, for his continuous directions and support throughout the whole work.*

*I cannot forget the great help of **Dr. Dalia FahmyEmam Ali**, Lecturer of Anesthesia, Intensive Care and Pain management, Faculty of Medicine - Ain Shams University, for his invaluable efforts , tireless guidance and for his patience and support to get this work into light.*

*Words fail to express my love, respect and appreciation to **my wife**for her unlimited help and support.*

*Last but not least, I dedicate this work to **my family**, whom without their sincere emotional support, pushing me forward this work would not have ever been completed.*

Ahmed Gamal

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List of Abbreviations

ACL	:	Anterior Cruciate Ligament
ADHD	:	Attention Deficit Hyperactivity Disorder
ASA	:	American Society of Anesthesiologists
BP	:	Blood Pressure
CNS	:	Central nervous system
COX	:	Cyclooxygenase
CPB	:	Cardiopulmonary bypass
ECG	:	Electrocardiogram
FDA	:	Food and Drug Administration
HR	:	Heart Rate
IV	:	Intravenous
L4,L5	:	Lumbar vertebrae
LAST	:	Local Anesthetic Systemic Toxicity
NAC	:	N-acetylcysteine
NIBP	:	Non-invasive blood pressure
NMDA	:	N-methyl-D- aspartate
NSAIDs	:	Nonsteroidal anti-inflammatory drugs
PABA	:	Para-aminobenzoic acid
SD	:	Standard Deviation
SPO2	:	Pulse-oximetry
SPSS	:	Statistical Program for Social Science
VAS	:	Visual analog scale

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Introduction

Arthroscopic knee surgery is a minimally invasive technique associated with moderate pain during the first 24 hours post-surgery. Effective pain control after reconstruction of the anterior cruciate ligament (ACL) requires a multimodal approach including opiates, peripheral nerve blocks, and a local anesthetic injection into the hamstring donor site or knee ***(Baverel et al., 2016).***

A recent systematic review identified numerous relevant studies yet found no consensus about the optimal analgesic strategy ***(Secrist et al., 2016).***

Opiates are associated with side effects (i.e., nausea, vomiting, dizziness, and vertigo) that may prevent early mobilization. Local anesthesia techniques therefore hold appeal as a means of decreasing opiate requirements ***(Lefevre, 2016).***

Good pain management is achieved with different peripheral nerve block techniques. The saphenous nerve is a branch of the femoral nerve, which provides sensory innervations to the peripatellar region and the medial side of the leg, below the knee. It provides cutaneous innervations to the anterior, anteromedial and postero-medial sides of the leg from the knee to the ankle. Saphenous nerve block was presented for the first time by Van der Wal using a transsartorial approach ***(Van der Wal, 1993).***

Other approaches have been described, with a success rate of 33---100 %***(Benzon et al., 2005).***

The introduction of ultrasound technology gives anesthetist a clear view of the nerve, which greatly facilitates blockage techniques guided by the adductor canal and femoral artery ***(Tsui and Özelsel, 2009).***

Saphenous nerve block using both neurostimulation and ultrasound techniques has been shown to be an effective adjunct to postoperative analgesia in arthroscopic meniscectomy***(Hanson et al., 2013).***

Aim of the work

The aim of this study is to evaluate the efficacy of the ultrasound guided saphenous nerve block compared to intravenous analgesics for post-operative pain management tin arthroscopic ACL repair under spinal anesthesia.

Pharmacology of Local Anesthetics

Local anesthetics are defined as drugs which when applied in sufficient concentration to the site of action prevent conduction of electrical impulses by the membrane of nerve and muscles ***(Strichartz and Berde, 2005).***

Chemistry and structure of local anesthetics:

Local anesthetics have a three part structure: aromatic (benzene ring), intermediate chain, and amine group. As the intermediate chain contains an ester or an amide linkage, they may easily be divided into esters and amides **(Fig. 1)*(Columb and Lennan, 2007).***

Ester linkage (-COO):

An ester linkage is relatively unstable and broken down by hydrolysis, both in solution and following injection into the plasma by pseudo-cholinesterase enzyme. Thus, its solution has a relatively short half-life and is usually difficult to sterilize as heat cannot be used ***(Columb and Lennan, 2007).***

Amide linkage (– NHCO):

An amide linkage is much more stable than an ester linkage and the drugs in solution withstand heat sterilization and changes in ph. Also they are not broken down in the plasma and must be metabolized by the liver ***(Barash et al., 2006).***

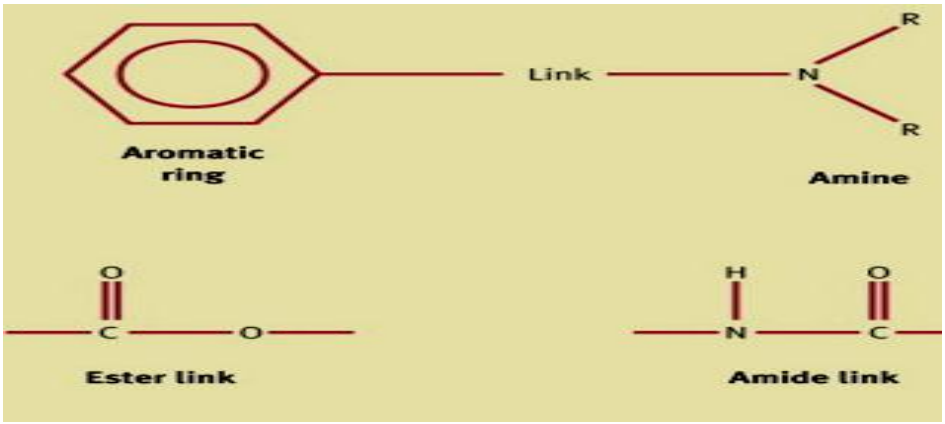

Fig.(1): Basic local anesthetic structure ***(Columb and Lennan, 2007)*.**

Mechanism of action of local anesthetics:

Solutions of local anesthetics are injected near the nerve. Diffusion of the drug molecules away from the locus is a function of tissue binding, elimination by the circulation and local hydrolysis of amino ester anesthetics. The result is penetration of the nerve sheath by the remaining drug molecules. Local anesthetic molecules then permeate the nerves axon membranes and reside there and in the axoplasm. The speed and extent of these processes depend on a particular drug's pKa, the lipophilicity of its base and cation species. Binding of local anesthetics to sites on voltage gated sodium channels prevent opening of the channels by inhibiting the conformational changes that trigger channel activation ***(Strichartz and Berde, 2005).***

All local anesthetics are membrane stabilizing drugs; they reversibly decrease the rate of depolarization and repolarization of excitable membranes by inhibiting sodium influx through sodium specific ion channels in the neuronal

cell membrane, in particular the so-called voltage-gated sodium channels ***(Rosenblatt et al., 2006)***

The receptor site is thought to be located at the cytoplasmic (inner) portion of the sodium channel. Local anesthetic drugs bind more readily to sodium channels in inactivated state, thus onset of neuronal blockade is faster in neurons that are rapidly firing. This is referred to as state dependent blockade ***(Stoelting and Miller, 2000)***.

Local anesthetics are 'weak bases' and are usually formulated as the hydrochloride salt to render them water-soluble. At the chemical's pKa, the protonated (ionized) and unprotonated (unionized) forms of the molecule exist in equilibrium but only the unprotonated molecule diffuses readily across cell membranes. Once inside the cell the local anesthetic will be in equilibrium, with the formation of the protonated (ionized form), which does not readily pass back out of the cell. This is referred to as "iontrapping" ***(Morgan et al., 2002)***.

In the protonated form, the molecule binds to the local anesthetic binding site on the inside of the ion channel near the cytoplasmic end. Acidosis such as caused by inflammation at a wound partly reduces the action of local anesthetics. This is partly because most of the anesthetic is ionized and therefore unable to cross the cell membrane to reach its cytoplasmic-facing site of action on the sodium channel ***(Morgan et al., 2002).***

All nerve fibers are sensitive to local anesthetics, but generally, those with a smaller diameter tend to be more

sensitive than larger fibers. Local anesthetics block conduction in the following order: small myelinated axons (*e.g. those carrying nociceptive impulses*), non-myelinated axons then large myelinated axons. Thus, a differential block can be achieved (*i.e.pain sensation is blocked more readily than other sensory modalities*) ***(Stoelting and Miller, 2000)***.

Since pain transmitting nerve fibers tend to be thinner and either non myelinated or lightly myelinated, the agent can diffuse more readily into them than into thicker and more heavily myelinated nerve fibers like touch, proprioception ***(Fanelli et al., 2007)***.

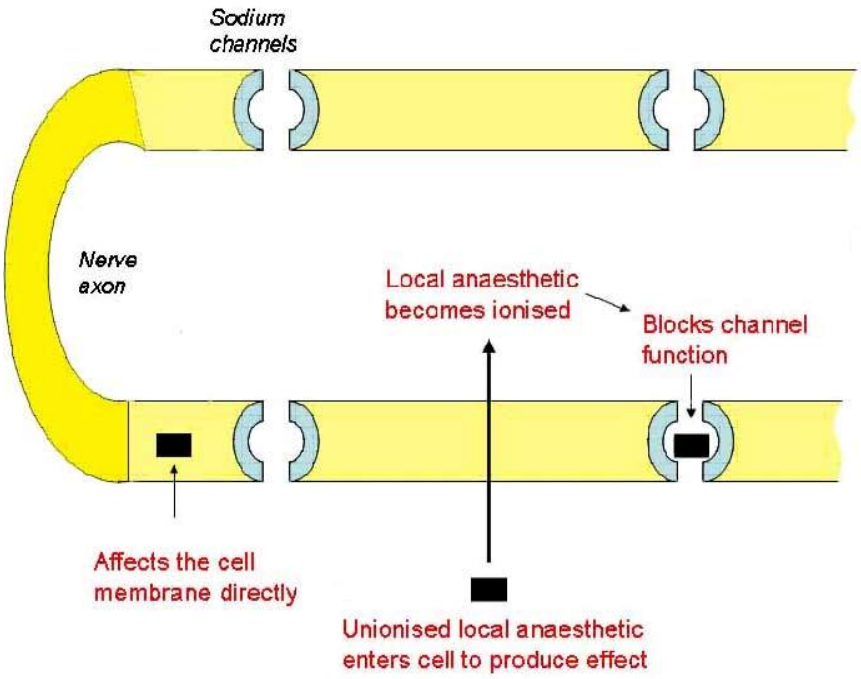

Fig. (2): Mechanism of action of local anesthetic drugs
(Lagan and McClure, 2004)