THE EFFECT OF SEVOFLUVONE OR PROPOFOL ON THE QTc INTERVAL IN PATIENTS ON Ca-CHANNEL BLOCKERS

Thesis

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By

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بسم الله الرحمن الرحيم

"ويسئلونك عن الروح قل الروح من أمر ربي
وما أوتيتم من العلم إلا قليلا"

صدق الله العظيم
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Abstract

The practical consideration of anesthesia for patients with LQTS therefore include management of torsade de pointes and avoidance of factors that increase the risk of precipitating torsade de pointes.

Key word:
SEVOFLUVONE QTc PATIENTS

TO MY PARENTS

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ABBREVIATIONS

Ach	:	Acetylcholine
AV	:	Atrioventricular node
CD	:	Changed depolarization
EAD	:	Early after depolarization
ECG	:	Electrocardiography
ICD	:	Implantable cardioverter defibrillators
ISR	:	Increased sinus rate
LAD	:	Left atrial dilatation
LAH	:	Left atrial hypertrophy
LQTs	:	Long QT syndrome
Prop	:	Propranolol
PVC	:	Premature ventricular contraction
QT	:	Correlated for heart rate
QTD	:	QT dispersion
RAD	:	Right atrial dilatation
RAH	:	Right atrial hypertrophy
RBBB	:	Right Bundle branch block
SA node	:	Sinoatrial node
Sevo	:	Sevoflurane
SLS	:	"Short-long-short" sequence
TdP	:	Torsade de points
Prop	:	Propofol

INTRODUCTION

Long QT syndrome is a disorder affecting the myocardial repolarization and result in a predisposition to the ventricular tachydysrrhythmia (Torsade de pointes). Torsade de pointes may degenerate into ventricular fibrillation and sudden death. LQTS can be divided into congenital or acquired forms.

Drugs, electrolytes abnormalities or metabolic disorders can cause acquired LQTS.

Particular aspects of the anesthetic management of patients with a prolonged QT interval include avoiding events leading to an increased sympathetic drive, increases in circulatory catecholamines and pharmacological agents may increase sympathetic discharge or prolong the QT interval.

Many drugs have been shown to be associated with a prolonged QT interval. Among these drugs sevoflurane is known to prolong the QT interval.

It has been reported that induction of anesthesia using propofol tends to shorten the QTc interval.

Calcium channel blockers are known to have major depressant effect on the atrioventricular node, a negative chronotropic effect on the SAN (sino-atrial node) and a negative inotropic effect on the cardiac muscle.

In the present study the effect of combination of calcium channel blockers with sevoflurane or propofol on the QTc interval is to be studied. Our aim is to point any interaction between these drugs on the QTc interval.

Aim of the Work:

The present study aims to assess the effect of induction of anesthesia with inhalational sevoflurane on patients receiving calcium channel blockers. Furthermore the effect of induction of anesthesia with propofol on patients receiving Calcium channel blockers on the QT interval. and finally comparing the effects of sevoflurane induction versus propofol induction on the QTc interval in these patients.

Chapter I

BASIC CARDIAC ELECTROPHYSIOLOGY

Cardiac Electrophysiology:

ECG (electrocardiogram) is a reflection of the electrical events occurring in the heart during each contraction. Its interpretation requires a good understanding of generation and conduction of cardiac electrical impulses[(1)].

Physiological anatomy of cardiac myocytes:

The atrial and ventricular muscle cells have cross striations and are branched. Each cell is bounded by a complex cell membrane, the sarcolemma and is filled with rod like bundles of myofibrils. The latter are the contractile elements. The sarcolemma of the myocyte invaginates to form an extensive tubular network (the T tubules) that extends the extracellular space into the interior of the cell. The nucleus, which contains almost all of the cell's genetic information, is often centrally located. Some myocytes have several nuclei. Interspersed between the myofibrils and immediately beneath the sarcolemma are many mitochondria, the main function of which is to generate the energy in the form of adenosine triphosphate (ATP) needed to maintain the heart's contractile function and the associated ion gradients. Of the other organelles, the sarcoplasmic reticulum (SR) is most important. From the SR is discharged, in response to the wave of electrical excitation, the calcium that triggers contraction; and when the calcium is once again taken up into the SR, relaxation ensues[(2)].

Myofibrils are formed of several fundamental contractile units called the sarcomeres. The sarcomere is limited on either side by the Z line to which the actin filaments are attached. Conversely, the myosin filaments extend from the center of the sarcomere in either direction toward but not actually reaching the Z lines. Titin is a large elastic molecule that supports myosin. During contraction, the filaments slide over each other without the individual molecules of actin or myosin actually shortening[(2)].

Cardiac myocytes are separated from one another by cell membranes called the intercalated discs. The latter have minimal electrical resistance. Thus, they form permeable communicating junctions or gap junctions that allow relatively free diffusion of ions, so that action potentials travel from one cardiac myocyte to another with only slight hindrance. This criterion creates from the cardiac muscle a functional syncytium[(3)].

From the above mentioned discussion cardiac muscle is striated in structure as skel*et al.* muscle and syncytial in function as smooth muscle[(3)] **(Fig. 1)**.

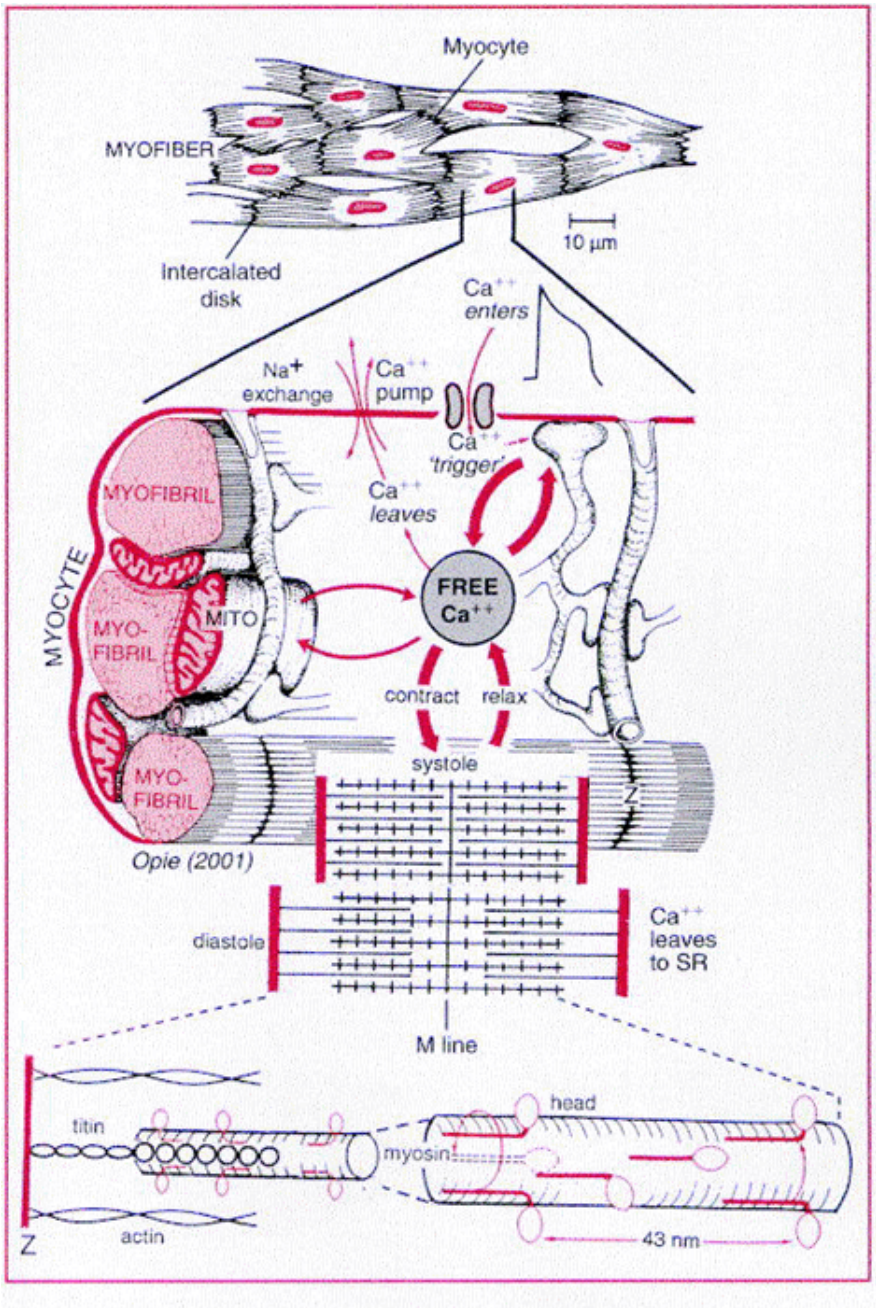

Fig. (1): Physiological anatomy of cardiac myocyte[3]