

Anesthetic Management of Patients with Cardiovascular Implantable Electronic Devices

An Essay

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

(... رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ

الَّتِي أَنْعَمْتَ عَلَيَّ وَعَلَى وَالِدَيَّ

وَأَنْ أَعْمَلَ صَالِحاً تَرْضَاهُ وَأُدْخِلْنِي

بِرَحْمَتِكَ فِي عِبَادِكَ الصَّالِحِينَ]

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✍ **Ahmed Abdo Mohamed Abd-Allah**

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List of Abbreviations

ACE	: Angiotensin converting enzyme
ACT	: Activated clotting time
AF	: Atrial Fibrillation
AICD	: Automatic implantable cardioverter-defibrillators
ANP	: Atrial natriuretic peptide
ASA	: American Society of Anesthesiologists
AVN	: Atrioventricular node
BPEG	: British pacing and electrophysiology group
BPM	: Beat per minute
CAD	: Coronary artery disease
CCB	: Calcium channel blocker
CIED	: Cardiac implantable electrical device
COP	: Cardiac output
CRT	: Cardiac resynchronization device
CS	: Coronary sinus
ECT	: Electroconvulsive therapy
EDP	: End diastolic pressure
EDV	: End diastolic volume
EF	: Ejection fraction
EMI	: Electromagnetic interference
EP	: Electrophysiological
ESC	: European Society of Cardiology
ESP	: End systolic pressure

List of Abbreviations (Cont.)

ESU	: Electrosurgical unit
ESV	: End systolic volume
ESWL	: Extracorporeal shock wave lithotripsy
HF	: Heart failure
HFA	: Heart Failure Association
ICD	: Implantable cardioverter defibrillator
LBBB	: Left bundle branch block
LV	: Left ventricle
MI	: Myocardial infarction
MRI	: Magnetic resonance imaging
MVO2	: Mixed venous oxygen saturation
NASPE	: North American society of pacing and electrophysiology
NBD	: NAPSE\BPEG Defibrillator
NYHA	: New York Heart Association
PM	: Pacemaker
PMT	: Pacemaker mediated tachycardia
PTCA	: Percutaneous transluminal angioplasty
RAA	: Renin angiotensin aldosterone
RBBB	: Right bundle branch block
RF	: Radiofrequency
RMP	: Resting membrane potential

List of Abbreviations *(Cont.)*

RV	: Right ventricle
TENS	: Transcutaneous electrical nerve stimulation
SA	: Sino atrial node
SR	: Slew rate
SV	: Stroke volume
VA	: Ventriculoatrial
VF	: Ventricular fibrillation
VT	: Ventricular tachycardia

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Introduction

Dysrhythmia is an abnormal heart rhythm. In which the heart beats may be too slow, too rapid, too irregular, or too early. Rapid arrhythmias (greater than 100 beats per minute) are called tachycardias. Slow arrhythmias (slower than 60 beats per minute) are called bradycardias. Irregular heart rhythms are called fibrillations (as in atrial fibrillation and ventricular fibrillation). When a single heart beat occurs earlier than normal, it is called a premature contraction. When dysrhythmias are severe or last long enough to affect how well the heart works, the heart may not be able to pump enough blood to the body. In most of cases sudden cardiac arrest (SCA) is fatal leading to sudden death (*Pell, 2008*).

The most common indications for pacing currently include: symptomatic bradycardia (resulting from sinus node dysfunction), atrioventricular conduction block after catheter ablation of the AV node or junction, Neurocardiogenic syncope and selected patients with Hypertrophic cardiomyopathy or the long QT syndrome (*Gregoratos et al., 2002*).

Cardiovascular implantable electronic device were first introduced in 1958, then implantable cardioverter-defibrillator followed in 1980. Since then, more than 2000 models of pacemakers. There has been a significant change

not only in pulse generators and leads but also in the indications for pacing, pacing modalities, implantation techniques and follow up of patients with implanted pacing devices (*Bryce et al., 2001*).

CIEDs include PM, ICD, cardiac resynchronization device, implantable loop recorder and implantable cardiovascular monitor (*Nacarelli et al., 2008*).

The number of patients with (CIEDs; previously termed cardiac rhythm management devices) continues to grow at an astonishing rate worldwide, but the level of comfort most anesthesiologists have in managing such patients in the perioperative period has not kept pace with that growth (*Stone and Apinis, 2009*).

Aim of the work

The main intent of this essay is to provide recommendations that promote safe management of patients with cardiovascular implantable electronic devices (CIEDs) throughout the perioperative period.

Chapter (1): **Cardiovascular Physiology**

Cardiac Muscle

The cardiac muscle is the collection of individual cells (cardiomyocytes) that are linked as a syncytium by gap junctional communication. Cardiac muscle cells also undergo excitation contraction coupling. Pacemaker cells in the heart can initiate propagated action potentials. Cardiac muscle cells also have a striated, actomyosin system that underlies contraction (**Scott B. 2010**).

Cardiac Action Potential

The myocardial cell membrane is normally permeable to K^+ but is relatively impermeable to Na^+ . A membrane-bound Na^+-K^+ -adenosine triphosphatase (ATPase) concentrates K^+ intracellularly in exchange for extrusion of Na^+ out of the cells. Intracellular Na^+ concentration is kept low, whereas intracellular K^+ concentration is kept high relative to the extracellular space. The relative impermeability of the membrane to calcium also maintains a high extracellular to cytoplasmic calcium gradient. Movement of K^+ out of the cell and down its concentration gradient results in a net loss of positive charges from inside the cell. An electrical potential is established across the cell membrane, with the inside of the cell negative with respect to

the extracellular environment, because anions do not accompany K^+ . Thus, the resting membrane potential represents the balance between two opposing forces: the movement of K^+ down its concentration gradient and the electrical attraction of the negatively charged intracellular space for the positively charged potassium ions (**Richard W, 2013**).

The differences between the membrane properties of cardiac and skeletal muscle account for the prolonged action potential in cardiac muscle. This is because the action potential in cardiac muscle is caused by opening of two types of channels: (1) the same fast sodium channels as those in skeletal muscle and (2) another entirely different population of slow calcium channels, which are also called calcium-sodium channels. This second population of channels differs from the fast sodium channels in that they are slower to open and, even more important, remain open for several tenths of a second. During this time, a large quantity of both calcium and sodium ions flows through these channels to the interior of the cardiac muscle fiber, and this maintains a prolonged period of depolarization, causing the plateau in the action potential (**Korzick DH, 2003**).