Junctional Ectopic Tachycardia after surgery for congenital heart disease

Thesis submitted for partial fulfillment of Master degree in Pediatrics

By

Hala Mohsen Abdul-Salam
M.B. B.Ch.

Supervisors

Prof. Dr. Salwa Helmy Omran
Professor of Pediatrics
Faculty of Medicine
Cairo University

Prof. Dr. Zeinab Salah El Din Selim
Professor of Pediatrics
Faculty of Medicine
Cairo University

Dr. Osama Mohamed Abdel-Aziz
Lecturer of Pediatrics
Faculty of Medicine
Cairo University

Cairo University
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"First and Foremost, Thanks are due to God,
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Abstract

Postoperative arrhythmias are a recognized complication of cardiac operations as it affects the hemodynamic stability. As Junctional Ectopic Tachycardia is the most common tachyarrhythmia in the postoperative course, we studied the incidence of JET, risk factors, inotrope usage, efficacy of treatment and outcome in 63 patients undergoing cardiac surgery on cardiopulmonary bypass during 6 months period. Twenty six patients (41.2%) developed JET. Ten patients had done fallot repair, four VSD closure, three CAVC repair & three senning operation. Correction of risk factors, hypothermia and sedation were effective in treatment of JET. Amiodarone was a safe effective antiarrhythmic drug.

Key words: Junctional ectopic tachycardia- risk factors- cardiopulmonary bypass- Amiodarone.

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List of abbreviations

ACC	Aortic cross clamp time
AEG	Atrial electrogram
AET	Atrial ectopic tachycardia
ARP	Atrial refractory period
ASO	Arterial switch operation
AV	Atrioventricular
AVN	Atrioventricular node
AVN	Atrioventricular node
AVNRT	AVN reentrant tachycardia
AVRT	Atrioventricular reentrant tachycardia
AVSD	Atrioventricular septal defect
CAVC	Common atrioventricular canal
CFB	Central fibrous body
CHB	Complete heart block
CHD	Congenital heart disease
CPB	Cardiopulmonary bypass
CS	Coronary sinus
DILV	Double inlet left ventricle
DORV	Double outlet right ventricle
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
EPDCs	Epicardium derived cells
HOCM	Hypertrophic obstructive cardiomyopathy
ICU	Intensive care unit
IVC	Inferior vena cava
JET	Junctional ectopic tachycardia

LA	Left atrium
L-TGA	Levo transposition of great arteries
LV	Left ventricle
mA	Milliampere
Mg	Magnesium
PVARP	Post ventricular atrial refractory period
PVCs	Premature ventricular contractions
QTc	Corrected QT interval
RA	Right atrium
RAA	Right atrial appendage
RAVT	Reciprocating atrioventricular tachycardia
RBBB	Right bundle branch block
RV	Right ventricle
SAN	Sinoatrial node
SVC	Superior vena cava
SVT	Supraventricular tachycardia
TAPVR	Total anomalous pulmonary venous return
TGA	Transposition of great arteries
TOF	Tetralogy of Fallot
VA	Ventriculoatrial
VRP	Ventricular refractory period
VSD	Ventricular septal defect
VT	Ventricular tachycardia
WPW	Wolf Parkinson white syndrome

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Introduction

Arrhythmias are common in the early postoperative period after cardiac surgery for congenital heart disease. Although transient and treatable in most cases, they are the cause of substantial morbidity and mortality during a vulnerable phase of hemodynamic instability **(Delaney et al., 2006).**The arrhythmia itself or the related treatment has significant clinical impact on the post-operative course and intensive care stay **(Brown et al., 2003).**

The outcome after pediatric cardiac surgery is generally excellent, and therefore a detailed evaluation of major morbidities, including arrhythmias, is vital, since this renders them more accessible to quality control **(Andreasen et al., 2008).**

The most frequent arrhythmia observed in the postoperative course of congenital heart surgery in children was frequent PVCs followed by JET then nonsustained VT, supraventricular tachycardia, and other miscellaneous arrhythmias as **Batra et al.**, **2006** stated.

JET can be resistant and life threatening despite aggressive treatment. It is certainly an important source of postoperative morbidity, and therefore a better understanding of the risk factors associated with JET is required to improve preventive management strategies, provide a guide as to how aggressive management should be to expedite arrhythmic conversion, and reduce associated in-hospital morbidity **(Batra et al., 2006).**

In general, JET is a self-limiting disorder that usually resolves within one week. On the other hand, JET can be also considered as a potential life-threatening postoperative arrhythmia due to the rapid heart rate that impairs the adequate filling of the ventricle and the loss of AV synchrony, thus leading to an acute impairment of cardiac output.

Additionally JET usually occurs at a time when the ventricles show an impairment of their diastolic function. As this tachycardia often occurs within the first 24 to 48 hours after corrective surgery, the patients may be on some form of catecholamine support. The presence of endogenous or exogenous catecholamines itself increases the heart rate thus deteriorating the clinical condition **(Dodge-Khatami et al., 2002).**

JET is a narrow QRS complex tachycardia, usually with atrioventricular dissociation and slower atrial than ventricular rate. It can also present with 1: 1 retrograde ventriculoatrial conduction **(Mildh et al., 2011).**

The precise mechanism of JET is not known, but it is thought to result from mechanical trauma to the proximal conduction tissue related to suture placement or indirect stretch injury. Several risk factors have been associated, both surgical and non surgical. The surgical risk factors include resection of muscle bundles and correction of right ventricular outflow tract. The non surgical risk factors previously reported are young patient age, longer cardiopulmonary bypass (CPB) time , aortic cross-clamp (ACC) time, low level of plasma magnesium , use of catecholamines and hyperthermia **(Rekawek et al., 2007).**

Treatment success is usually defined as a stable decrease in the ventricular rate below 140–150/min, the possibility of atrial pacing and thereby the improvement of cardiac output. Optimal success is the reinstitution of sinus rhythm. A variety of different therapeutic strategies have been tested in postoperative and congenital JET and many of these are based on a specific staged treatment protocol. These include conventional supportive treatment, specific medical antiarrhythmic therapy, surgical intervention, catheter ablation of the HIS-bundle, and specific forms of pacing and surface cooling **(Cabrera et al., 2002).**

Aim of the work

The purpose of this study is to evaluate risk factors associated with JET by prospectively evaluating all patients undergoing congenital heart surgery during a 6 months period, taking into account a wider spectrum of surgical repairs, the presence or absence of electrolyte abnormalities, and patterns of use of inotropes utilizing a graded inotropic score. In addition, the study will evaluate the efficacy of management of JET and its impact on the postoperative course and intensive care stay.

Chapter I

Cardiac Conduction System

Embryological development of the cardiac conduction system

The cardiac conduction system is a complex and highly specialized network that is fundamental to cardiac electrophysiology. Understanding the embryology underpinning the development of the mature heart not only offers insight into the critical spatial relationships of the conduction system but enables conceptualization of the relevant structures and their variants **(Mirzoyev et al., 2010).**

Myocyte development and fiber orientation

During cardiogenesis, myocytes develop into either contractile or conduction cells **(Christoffel et al., 2009).** Three models have been postulated by which cardiac cells develop and differentiate:

1. The first model is based on a multiple ring theory. It hypothesizes that during heart chamber development and growth, cells in certain regions of the heart tube do not proliferate as rapidly as cells in genetically predetermined atrial and ventricular regions. As the tubular heart grows, the slower-proliferating myocytes form constrictions or rings around which the heart will fold.
2. The second model is based on the idea that the conduction system framework is present in early development and enables recruitment of adjacent myocytes to form further elements of the conduction system.
3. The third model, the early specification model **(figure 1)**, postulates that myocytes begin expressing either conduction genes or working (contractile) genes early in the development. Cells expressing conduction system markers slowly proliferate and form components of the

conduction system, whereas cells lacking the markers proliferate faster and develop into contractile tissue **(Mirzoyev et al., 2010).**

It is important to note that concurrent with the basic chamber development, separate transcription factors simultaneously coordinate the development of the cardiac conduction system. The first indication of cardiac contraction occurs around 23 days after conception in the human and peristalsis of the tubular heart. Primitive coordinated and sequential chamber contraction occurs soon after looping of the heart and is subsequently initiated by the primordial atrium which acts as the interim pacemaker of the heart. Following that, the sinus venosus adopts this function; finally to be usurped by the sinoatrial node develops during the fifth week **(Hatcher and Basson, 2009).**

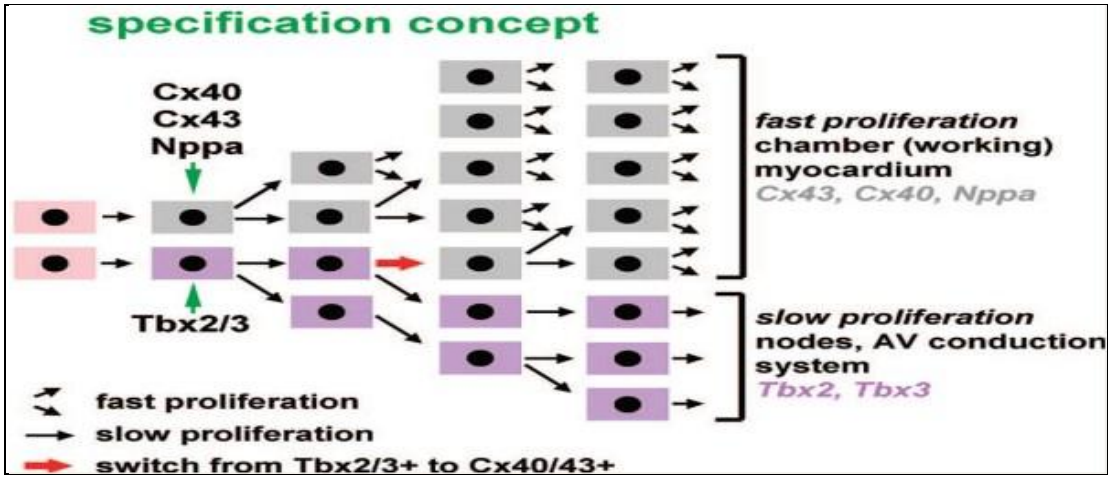

Figure (1): The specification concept is in some ways a hybrid of the ring and recruitment concept. Figure courtesy of Antoon F.M. Moorman, Academic Medical Center, and Amsterdam.

Depolarization of the cardiac muscle occurs through electrochemical and metabolic coupling which is facilitated by the connexins present within the gap junctions that act as micro-channels and allow the passage of ions between cells. Four major connexins have been identified. Based on their conductive