

CARDIAC ARRHYTHMIAS IN RELATION TO ELECTROLYTE IMBALANCE

**Essay Submitted for the partial fulfillment of
Master Degree in Intensive care**

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Abbreviations

ACE	Angiotensin Converting Enzyme
AF	Atrial fibrillation
ANP	Atrial natriuretic peptide
APD	Action potential duration
AV	Atrioventricular
CA	Carbonic anhydrate
CD	Collecting duct
Cl	Chloride
CO ₂	Carbon dioxide
DAD	Delayed afterdepolarizations
DCT	Distal convoluted tubule
EADs	Early afterdepolarizations
ECF	Extracellular fluid
ECG	Electrocardiographic
EP	Electrophysiological
ERP	Effective refractory period
F waves	Fibrillatory waves
GI	Gastrointestinal
H	Hydrogen
H ₂ CO ₃	Carbonic acid

HCO _r	Bicarbonate
HERG K ⁺	Human ether-a-go-go-related gene
I.V	Intravenous
ICF	Intracellular fluid
ICU	Intensive care unit
I _f	Funny currents
IK ₁	Inward rectifier current
I _{kr}	Delayed rectifier current
K	Potassium
LAFB	Left Anterior Fascicular Block
LBBB	Left bundle branch block
LOH	Loop of Henle
LPFB	Left Posterior Fascicular Block
L-type Ca ⁺⁺	Long-lasting Ca ⁺⁺
MAT	Multifocal Atrial Tachycardia
Mg	Magnesium
MgSO ₄	Magnesium Sulfate
Na	Sodium
NSAIDs	Non steroidal anti-inflammatory drugs
OH	Hydroxyl ion
PACs	Premature atrial complexes
PJCs	Premature junctional complexes

PO ₄	Phosphorus
PSVT	Paroxysmal supraventricular tachycardia
PTH	Parathyroid hormone
PVCs	Premature ventricular complexes
RAAS	Renin angiotensin aldosterone system
RBBB	Right bundle branch block
RMP	Resting membrane potential
RRP	Relative refractory period
SA	Sinoatrial node
TP	Threshold potential
T-type Ca ⁺⁺	Transient Ca ⁺⁺
VT	Ventricular tachycardia
WPW	Wolf-Parkinson White syndrome

Introduction

Cardiac arrhythmias are known to be caused by many factors. Among them electrolyte imbalance is the most important because of electrical activity of the heart is composed of transmembrane fluxes of Na^+ , Ca^{2+} and K^+ , so high or low concentrations of these electrolytes can affect active and passive electrical properties of the membrane in the cardiac tissues (*Nishimura et al, 1997*).

Electrolyte disorders are an important cause of ventricular and supraventricular arrhythmias in the intensive care unit. Electrolytes such as potassium, magnesium, calcium and phosphate play important roles in cellular metabolism and energy transformation, and in the regulation of cellular membrane potentials, Depletion of these electrolytes can induce a wide range of clinical disorders as severe arrhythmias.

The risk for arrhythmias increases significantly when more than one electrolyte is deficient especially in the presence of ischemic heart disease. It is well known that hypokalemia can induce cardiac arrhythmias so the importance of regulating potassium levels is well recognized in most intensive care units (ICUs) and potassium levels are measured frequently. In contrast, electrolytes such as magnesium, calcium and phosphate are measured far less frequently (*Kees and Armand, 1998*).

Electrolyte disorders exert their actions by modulating the conduction of ions across specific cardiac membrane channels and this in turn can result in antiarrhythmic or proarrhythmic sequelae.

The electrical activity of the heart depends on transmembrane ionic gradients and the time and voltage dependent. Electrolyte abnormalities may generate or facilitate clinical arrhythmias, even in the setting of normal cardiac tissue. Furthermore, electrolyte aberrations are more likely to interact with abnormal myocardial tissue to generate their own cadre of cardiac arrhythmias (*Nabil and Gioia, ~ ~ ~*).

Aim of work

The aim of this work is to highlight the electrolyte imbalance and its relation to cardiac arrhythmias.

CHAPTER (1)

Electrolyte homeostasis

Electrolytes play an essential role in numerous physiologic Functions in the body. Many metabolic processes and normal organ functions are dependent on precise intracellular as well as extracellular electrolyte concentrations (*Michael et al, ~ ~ ~*).

This balance is maintained through a complex system of multiple mechanisms, involving many different hormones and organs that influence electrolyte distribution. Consequently, several factors can equally affect electrolyte homeostasis, including acid base imbalance, fluid status, organ dysfunction, neurohormonal disorders, and disease state (*Besunder and Smith, ~ d d*).

Electrolytes abnormalities are common in the intensive care unit (ICU). Multiple mechanisms may be involved in electrolyte abnormalities in adult patients in the ICU, including altered absorption and distribution; excessive or inadequate administration, alterations in hormonal, neurologic and homeostatic mechanisms; or altered excretion via gastrointestinal (GI) and renal losses, as well as changes in fluid status and fluid shifts (*Michael et al, ~ ~ ~*).

Potassium

Potassium is the second most abundant cation in the body and the major cation in the intracellular fluid (ICF) compartment. Approximately 98% of body potassium is contained within body cells and this is maintained by the Na–K-ATPase pump. Therefore a significant shift in potassium to or from ICF can markedly affect the serum potassium concentration and exert profound effects on the resting membrane potential. The intracellular concentration of potassium ranges from 140 to 150 mEq/L. While the potassium content of the ECF (3.0 to 5.0 mEq/L) is considerably less (*Gennari, 1997*).

Potassium homeostasis

Homeostatic mechanisms maintain plasma K^+ concentration between 3.0 and 5.0 mEq/L despite marked variation in dietary K^+ intake. Potassium intake is normally derived from dietary sources. In healthy persons, potassium balance usually can be maintained by a daily dietary intake of 50 to 100 mEq (*Shoemaker, 1997*).

The kidneys are the main source of potassium loss. Approximately 80% to 90% of potassium losses occur in the urine, with the remainder being lost in stools or sweat (*Shoemaker, 1997*).

Mechanisms of Regulation

Plasma potassium is largely regulated through two mechanisms:

- (1) Renal mechanisms that conserve or eliminate potassium.
- (2) A transcellular shift between the intracellular and extracellular compartments (*Kumar and Clark, 2001*).