

CARDIAC ARRYTHMIA IN RESPIRATORY ICU

An Essay

Submitted for Partial Fulfillment in Master Degree
of Critical Medicine

By

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2012**

Acknowledgment

Thanks to ***Allah*** from start to end, that this work has been completed.

First foremost, I am extremely grateful to ***Prof. Dr. Samir Abd El Rahman El Sabaay***, Prof. of Anaesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his kind supervision, valuable guidance and continuous encouragement and I wish him a good health.

Also, I wish to express my great pleasure and deep gratitude to ***Prof. Dr. Sherif Farouk Ibrahim***, Prof. of Anaesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his support, great help, and continuous and valuable directions.

I would also like to thank ***Dr. Heba Abd El Azim Labib***, Lecturer of Anaesthesia and Intensive Care, for her very helpful suggestions and encouragement. She spent a lot of her time for completing this work.

I would like to express my special thanks to all members of Anesthesiology and Intensive Care department, faculty of medicine, Ain Shams university for their valuable support. I would like to thank my family for every thing.

AMAL GAMAL SAYED SHALABY
2012

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List of Abbreviations

<i>Abb.</i>	<i>Meaning</i>
ACPO	Acute cardiogenic pulmonary oedema
ALI	Acute lung injury
ADP	Adenosine triphosphate
AF	Atrial fibrillation
AN	Atrionodal region
AP	Action potential
ARDS	Acute respiratory distress syndrome
ATP	Adenosine triphosphate
AV	Atrioventricular node
AV	Arteriovenous
AVNRT	Atrioventricular nodal reentrant tachycardia
BiPAP	Bilevel positive airway pressure
cAMP	Cyclic adenosine 5' - monophosphate
CAP	Community acquired pneumonia
CFTR	Cystic fibrosis transmembrane conductance regulator channel.
CLCA1	Calcium activated chloride channels.
CT	High-resolution computed tomography.
CSF	Cerebrospinal fluid.
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CURB	Confusion, urea concentration (<7 mmol/L-1), respiratory rate (>30/min-1), blood pressure (systolic pressure >90 mmHg) and age (65 yrs).

DI	Diffusing capacity
DPG	2, 3-diphosphoglycerate
DVT	Deep venous thrombosis.
ECG	An electrocardiogram
E_m	Resting membrane potential
ERP	Effective refractory period
f	Respiratory rate
Fc	Cardiac frequency
GI	Gastrointestinal
GDP	Guanosine diphosphate
GTP	Guanosine triphosphate
HPS	His-Purkinje system
ICU	Intensive care unit.
IV	Intravenous
K_{2P}	two-pore Potassium channel
LAP	left atrial pressure
OSA	Obstructive sleep apnea
MAT	Multifocal atrial tachycardias
MDS	Megadose
MgSO₄	Magnesium sulfate
N	Nodal region
NH	Nodal His bundle region
NIPPV	Non-invasive positive pressure ventilation
PAC	Premature atrial contraction
Paco₂	Pco ₂ in arterial blood
PAOP	pulmonary artery occlusion pressure

PCP	Pneumocystis jiroveci pneumonia
PCV	Pressure controlled ventilation
PC-IRV	Pressure-controlled, inverse-ratio ventilation
PE	Pulmonary embolism
Peco2	Expired gas
PEEP	positive end-expiratory pressure
Pplat	Inspiratory plateau pressure
PIFR	peak inspiratory flow rate
PSP	Primary spontaneous pneumothorax
QTc	<i>Corrected QT interval</i>
RICU	<i>Respiratory intensive care unit</i>
RSA	<i>Respiratory sinus arrhythmia</i>
TRP	Transient refractory period
TDP	<i>Torsade de pointes</i>
SAN	The sinoatrial node
SSP	Secondary spontoneus pneumothorax
SSS	Sick sinus syndrome
VCV	Volume control ventilation
VGSC	Voltage-gated sodium channels
VI	Inspiratory flow rate
V/Q	The ventilation: perfusion ratio
VT	Tidal volume
VTE	Venous thromboembolism

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INTRODUCTION

The heart beat is normally initiated by an electrical discharge from the sinoatrial node. The atria and ventricles then depolarize sequentially as electricity passes through the specialized conducting tissue (*Boon et al., 2002*).

Most cardiac arrhythmias can be described as abnormalities of impulse formation, impulse conduction, or a combination of both. For example, the ventricular premature complex (VPC) which initiates monomorphic ventricular tachycardia in a patient with a prior anterior myocardial infarction represents an abnormality of impulse formation whereas the scar-mediated reentrant ventricular tachycardia is a manifestation of abnormal impulse conduction. Triggered activity and abnormal automaticity, two common mechanisms of arrhythmia, are categorized as disorders of impulse formation (*Zimetbaum et al., 2009*).

The principle function of the lungs is gas exchange. Although disease may substantially affect any of the functional elements of the respiratory system, the term acute respiratory failure (ARF) is used only when gas exchange is so severely impaired that arterial hypoxemia or hypercapnia occurs (*Smith, 2005*).

Respiratory failure may be classified as *hypercapnic respiratory failure* is defined as an arterial Pco₂ (Paco₂) greater than 45 mmHg. *Hypoxemic respiratory failure* is defined as an arterial Po₂ (Pao₂) less than 55 mmHg when the fraction of oxygen in inspired air (Fio₂) is 0.60 or greater. In many cases, hypercapnic and hypoxemic respiratory failure coexist (**Grippi, 2008**).

It is well established that progressive hypoxia may be causally associated with a rise in heart rate, the effects of hypercapnia are more unpredictable, being associated with either a rise or a fall in the heart rate (**Sanders et al., 1989**).

Patients with hypercapnic respiratory failure are subject to complications associated with both respiratory failure and its treatment. Several common complications have been identified: Cardiac arrhythmias of all types are common, relating to diverse factors including hypoxemia, wide swings in pH, electrolyte disturbances, and drugs that may be employed such as β -adrenergic agents, theophylline, and *digoxin* (**Smith, 2005**).

General principles of treatment are: 1. Treatment may be avoided if the patient is not suffering hemodynamic compromise or if this is not incipient 2. Reduce an unnecessarily high cvp to reduce risk of recurrent atrial fibrillation. 3. Correct hypokalemia. 4. Correct hypomagnesemia. 5. Avoid hypoxia or

hypercapnia. 6. Ensure central venous access is correctly positioned. 7. If ischemic consider revascularization. 8. Dc cardioversion or pacing is generally safer than pharmacotherapy. 9. Use familiar drugs where possible. 10. Avoid polypharmacy (**Montgomery et al., 2010**).

AIM OF THE WORK

To study the effect of respiratory emergencies on heart rate and rhythm.

ELECTROPHYSIOLOGY OF THE HEART AND NORMAL ECG

The heart is the life-beating, always thumping muscle in your chest. From inside the womb until death, the thump goes on. The heart for the average human will contract about 3 billion times; never resting, never stopping to take a break except for a fraction of a second between beats. If a person lives to be 80 years of age. His or her heart will continue to beat 100, 000 times a day. Many believe that the heart is the first organ to be functional. Within weeks of conception the heart starts its mission of supplying the body with nutrients (*Provophys et al., 2007*).

The heart wall, which encases the heart, is made up of three layers: epicardium, myocardium, and endocardium. The epicardium, the outermost layer, consists of squamous epithelial cells overlying connective tissue. The myocardium, the middle and thickest layer, makes up the largest portion of the heart's wall. This layer of muscle tissue contracts with each heartbeat. The endocardium, the heart wall's innermost layer, consists of a thin layer of endothelial tissue that lines the heart valves and chambers (*Follin et al., 2006*).

The heart consists of four chambers: right and left atria, and right and left ventricles. The atria are smaller, thinner

walled, low-pressure chambers. Approximately 30% of blood flow to the ventricles is the result of atrial contraction, also known as *atrial kick*. The remaining 70% of blood that reaches the ventricles is the result of pressure differences between the atria and the ventricles. The ventricles are larger, higher-pressure chambers with thicker walls than the atria. The walls of the left ventricle are thicker than the right ventricle because the left ventricle must generate a large amount of force to eject blood into the aorta (*Morton et al., 2005*).

Cardiac cells:

There are five functionally and anatomically separate types:

1. Sinoatrial node.
2. Atrioventricular node.
3. His-purkinje system.
4. Atrial muscle.
5. Ventricular muscle (*Banerjee (a), 2005*).

Properties of cardiac muscle:

Automaticity (chronotropy): the ability to initiate cardiac impulse.

Conductivity (dromotropy): the ability to conduct an electrical impulse.