Cardiac arrhythmias in critically ill patients

Essay

Submitted for partial fulfillment for master degree in Intensive care medicine

By

Mahmoud El-shahat Awad Makhluf
M.B.B.Ch.

Supervisors

Prof. Dr. Ibrahim Abd El-Ghany Ibrahim

Professor of anesthesia and ICU
Faculty of Medicine - Ain Shams University

Dr. Ahmed Mohamed Shafik

Assistant Professor of Anesthesia and ICU
Faculty of Medicine - Ain Shams University

Dr. Mayar Hassan El-Sersi

Lecturer of Anesthesia and ICU
Faculty of Medicine - Ain Shams University

Faculty of Medicine
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بسم الله الرحمن الرحيم

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A Protocol of An Essay

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Introduction

Although most hearts beat with remarkable fidelity and resilience, under certain circumstances the rhythm of the heart can fail. This is known as a cardiac arrhythmia. Cardiac arrhythmias are a leading cause of morbidity and mortality***(Keating MT and Sanguinetti MC,2001)***.

Critically ill patients often have underlying heart disease, are frequently submitted to metabolic, ischemic, or neurohormonal stressors,and are therefore particularly at risk for cardiac arrhythmias***(Trohman RG and Parrillo JE,2000)***.

Arrhythmias are common in the intensive care unit and represent a major source of morbidity and increased length of stay ***(Hollenberg SM,2007)***. The recent reported incidence of cardiac arrhythmias in a large, prospective, multicenter study in a general ICU populations is about 12%. Atrial fibrillation was the most common form of arrhythmia***(Annane D et al.,2008)***. An arrhythmia incidence of up to 78% has been reported in a single center retrospective study with atrial fibrillation being the prominent clinical problem. Arrhythmias were more likely to occur in patients admitted in the ICU for cardiovascular disorders and were associated with an increased risk of death***(Artucio H and Pereira M,1990)***. However, this retrospective study was too inclusive, including even patients with ventricular or supraventricular premature beats or bundle branch block which do not represent the commom perception of a significant sustained arrhythmia. Arrhythmias are most likely to occur in patients with structural heart disease. The inciting factor for an arrhythmia in a given patient may be a transient imbalance, often related to hypoxia, infection, cardiac ischemia, catecholamine excess (endogenous or exogenous), or an electrolyte abnormality ***(Hollenberg SM,2007)***.

Normal heart rhythm requires the finely orchestrated activity of a number of ion channels and transporters and the orderly propagation of electrical impulses throughout the myocardium; disruption of either can have severe consequences, resulting in potentially lethal heart rhythm disturbances. The generation of

cardiac arrhythmias requires the presence of a susceptible myocardial substrate and an appropriate trigger. Although any heart may serve as a substrate for the development of a potentially serious cardiac arrhythmia, in structurally normal hearts this is rare and requires highly potent triggers. Understanding the basic biology of arrhythmogenesis holds the promise of identifying novel targets for the treatment of arrhythmias, including prevention of the development of structural changes in the heart that form the substrate for cardiac rhythm disturbances***(Shah M et al., 2005)***. Cardiac arrhythmias are generally produced by one of three mechanisms: enhanced automaticity, triggered activity, or reentry***(Podrid PJ,2008)***.

Patients with cardiac arrhythmias may come to the attention of their physicians in a variety of ways. An asymptomatic arrhythmia may be fortuitously discovered on a routine electrocardiogram or physical exam. Alternatively, a patient may present with a complaint suggestive of arrhythmia, usually palpitations, syncope, or presyncope. Finally, patients who have no history of arrhythmia, but who may be prone to ventricular tachyarrhythmias because of underlying heart disease, may undergo routine testing to evaluate their level of risk. Patients in each category may be evaluated by invasive or noninvasive means***(Richardson AW and Zimetbaum PJ,2002)***.

In the management of clinical arrhythmias, the physician must evaluate and treat the whole patient, not just the rhythm disturbance. Evaluation of the patient begins with a careful history and physical examination and should usually progress from the simplest to the most complex test, from the least invasive and safest to the most invasive and risky, and from the least expensive out-of-hospital evaluations to those that require hospitalization and sophisticated, costly procedures***(Zipes DP and Miles WM,2004)***. In the great majority of cases the correct diagnosis can be determined at the initial consultation after careful analysis of the history, findings at clinical examination, and 12-lead ECG***(Garratt C,2001)***.

When present or expected, dysrhythmia should be managed seriously. Hemodynamic status may be compromised by bradycardia, tachycardia, loss of atrial transport function,or

ineffective ventricular contractions***(Thompson A and Balser JR,2004)***. The physiologic impact of a dysrhythmia depends on ventricular response rate, duration of dysrhythmia, and underlying cardiac function***(Heintz KM and Hollenberg SM,2005)***.

The presence of a dysrhythmia or cardiac conduction disturbance should provoke a careful evaluation for the underlying cardiopulmonary disease, drug toxicity, or metabolic abnormality. Therapy should be initiated for symptomatic or hemodynamically significant dysrhythmias, first to reverse an underlying cause and second to treat the dysrhythmia***(Fleisher LA et al., 2007)***.

In the past, pharmacologic therapy was the cornerstone of treatment for symptomatic and life-threatening ventricular arrhythmias. However, alternative nonpharmacologic therapy has received increasing attention because of the high failure rate, common proarrhythmic actions, and frequent toxicity of the antiarrhythmic drugs. Included in this group are the implantable cardioverter-defibrillator (ICD), catheter ablation, and, at times, cardiac surgery. Electrophysiologic cardiac mapping techniques play an integral role during catheter ablation of ventricular arrhythmias. Mapping permits precise localization of the myocardial sites of origin of focal rhythm disturbances***(Ganz LI and Arnsdorf MF,2008)***. Catheter ablation has revolutionized the management of patients with tachyarrhythmias***(Morady F,1999)***. More recently, cryoablation and other energy sources have been used in some specific clinical settings**(Ganz LI,2007)**. Antidysrhythmic pharmacology is focused primarily on the cardiac ion channels and adrenergic receptors as drug targets. The number of drug targets for antidysrhythmic therapy is expanding exponentially***(Priori SG et al.,1999)***. Currently available antidysrhythmic drugs are limited by modest efficacy and significant toxicity***(Page RL.2007)***. An ideal antidysrhythmic agent does not exist, and drug selection should be highly individualized***(Koufakia M et al., 2006)***. Thus, cardiologic intervention for dysrhythmias has evolved from pharmacologic therapy to surgically based elimination of dysrhythmogenic foci and circuits to transvenous catheter-based ablation procedures. It has dramatically changed the management of dysrhythmias***(Satti SD and Epstein LM,2003)***. Also, Invasive

cardiac electrophysiology has evolved rapidly from a research tool to an established clinical technique for the investigation and treatment of cardiac rhythm disorders***(Rahimtoola SH et al.,1987)***. Pacemaker and defibrillator management is increasingly complex and is a subject of comprehensive reviews***(Spotnitz HM 2003)***.

Arrhythmias in critically ill patients should be probably understood and managed by every intensivist in order to improve intensive care outcome. We will try to cover some aspects about this issue in our work.

Aim of the Work

Our study aims at discussing the most important causes of arrhythmias in critically ill patients, explaining how to diagnose and monitor them and presenting the recent methods of management, depending on clear understanding of the basic physiolgy, pathophysiology and molecular biology of arrhythmia.

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Mahmoud El-shahat Awad Makhluf
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