Treatment of cardiac arrhythmias in the emergency department

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Treatment of Cardiac Arrhythmias in the Emergency Department

Graduate Thesis

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This graduate thesis was written at the Emergency Department, Sisters of Charity University Hospital Center, Zagreb, mentored by Professor Vesna Degoricija, MD, PhD, and was submitted for evaluation in the academic year 2014/2015.

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List of abbreviations

AADs - Anti-arrhythmic drugs

ACE - Angiotensin converting enzyme

AED - Automated external defibrillator

AF - Atrial fibrillation

AV - Atrioventricular

Ca - Calcium

CABG - Coronary artery bypass graft

CDC - Center of Disease Control

CHD - Coronary heart disease

CHF - Congestive heart failure

CI - Confidence interval

COPD - Chronic obstructive pulmonary disease

CPR - Cardiopulmonary resuscitation

ECG - Electrocardiogram

ICD - Implantable cardioverter defibrillator

K - Potassium

IV - Intravenous

LV - Left ventricular

MI - Myocardial infarction

Na – Sodium

NSAIDs - Non-steroidal anti-inflammatory drugs

NYHA - New York Heart Association

PEA - Pulseless electrical activity

RCT - Randomized controlled trial

RFA - Radiofrequency Abrasion

SCD - Sudden cardiac death

STEMI - ST elevation myocardial infarction

SVT - Supraventricular Tachycardia

PAC - premature atrial contractions

PVC - Premature ventricular contractions

TTE - Transthoracic echocardiogram

VF - Ventricular fibrillation

VT - Ventricular tachycardia

Summary

Title: Treatment of cardiac arrhythmias in the Emergency Department
Name: Haider Sabhan

Cardiac arrhythmias, some of the most frequent diagnoses presenting at the emergency department, require a good and thorough understanding and quick recognition in the stressful emergency department environment.

Despite dedicating significant time in training to the mechanisms and treatment of cardiac rhythm disturbances, most physicians are uncomfortable and lack confidence when faced with such clinical situations. Therefore, a solid understanding of the different causes and quick action is essential and can be lifesaving in the emergency room setting.

Arrhythmias and conduction disorders are caused by abnormalities in the generation and/or conduction of electrical impulses in the heart. Symptoms such as dizziness, palpitations, and syncope are frequent complaints encountered by family physicians, internists, and cardiologists. In contrast to these ubiquitous complaints, which are generally benign, sudden cardiac death remains an important public health concern.

Classification of arrhythmias is essential for the quick recognition and, thus, quick treatment. Currently, arrhythmias are divided simply as tachycardias and bradycardias. Bradycardias are further classified into heart block or simple bradycardia. Tachycardia, on the other hand, can be divided into two categories based on the width of the QRS complex: narrow complex (QRS duration is less than 120 milliseconds) or broad complex (QRS duration is more than 120 milliseconds).

In 1970, Dr. Vaughn-Williams, Oxford University, introduced the first classification of anti-arrhythmic drugs. With regards to management of atrial fibrillation, Class I and III are used in rhythm control as medical cardioversion agents, while class II and IV are used as rate control agents.

Keywords: arrhythmias, treatment, recognition, cardiac abnormalities, classifications

Introduction

Not many things can raise the anxiety level in a physician more than a call to the emergency department about a patient who has a cardiac arrhythmia. Despite a significant amount of time throughout medical training being dedicated to understanding the mechanisms and treatments of cardiac rhythm disturbances, most physicians are uncomfortable knowing how to correctly approach these kinds of patients.

Cardiac disorders are the most common clinical cases presenting in the emergency room. Therefore, a solid understanding of the different causes and quick reaction to these clinical situations is lifesaving in the stressful emergency room environment.

The current annual incidence of sudden cardiac death in the United States of America is likely to be in the range of 180,000–250,000 per year. Coinciding with the decreased mortality from coronary artery disease, there is additional evidence pointing toward a significant decrease in rates of sudden cardiac death in the US during the second half of the twentieth century. However, the alarming rise in the worldwide prevalence of obesity and diabetes in the first decade of the 21st century would indicate that this favorable trend is unlikely to persist. We are likely to witness a reemergence of coronary artery disease and heart failure and, as a result, cardiac death will have to be confronted as a shared and unselective worldwide public health problem (1).

The healthy heart always beats in a regular, coordinated manner, due to the electrical impulses generated and spread by myocytes. Theses myocytes with special electrical properties trigger a sequence of organized myocardial cell contractions. Arrhythmias and conduction disorders are caused by abnormalities in the generation or conduction of these electrical impulses or both.

Any heart disorder, including congenital abnormalities of structure (eg, accessory atrioventricular connection) or function (eg, hereditary ion channelopathies), can disturb a heart's rhythm. Systemic factors that can cause or contribute to a rhythm disturbance include electrolyte abnormalities (particularly low K or Mg), hypoxia, hormonal imbalances (eg, hypothyroidism, hyperthyroidism), and drugs and toxins (eg, alcohol, caffeine).

Cardiac dysrhythmia is any of a group of conditions in which the electrical activity of the heart is irregular, faster, or slower than normal (1). The term tachycardia pertains to a heart rate above 100 beats per minute and bradycardia describes a heart rate below 60 beats per minute.
Although many arrhythmias are not life-threatening, some can cause serious impairment of the cardiac function, with the worst case scenario being cardiac arrest (1).
Arrhythmias can occur in the upper chambers of the heart (atria), in the lower chambers of the heart (ventricles), or at the level of the atrioventricular junction.

Cardiac arrhythmias are one of the most common causes of death in the Western world, and specifically, of sudden death. Some arrhythmias, such as atrial fibrillation, may be asymptomatic, but still may predispose the patient to potentially life-threatening conditions such as stroke or embolism.

Symptoms such as dizziness, palpitations, and syncope are frequent complaints encountered by family physicians, internists, and cardiologists. Although these presenting complaints are generally benign, arrhythmias may also present with sudden cardiac death, making arrhythmias an important public health concern. Statistics from the Centers for Disease Control and Prevention (CDC) have estimated that up to 50% of patients have sudden death as the first manifestation of cardiac disease.

This graduate thesis will explain the basics of cardiac anatomy and pathophysiology. The thesis will start with the presenting symptoms, classification, and further explore the treatments of different types of arrhythmias.

Anatomy and Physiology of the Heart

Anatomy of the heart

At the junction of the superior vena cava and the lateral right atrium is a cluster of cells that generate the initial electrical impulse of each normal heartbeat, called the sinoatrial (SA) or sinus node, can be found (2,3,4). The electrical discharge of these pacemaker cells stimulates adjacent cells, leading to further stimulation of successive regions of the heart in an orderly sequence. Impulses are transmitted through the atria to the atrioventricular (AV) node via preferentially conducting internodal tracts and unspecialized atrial myocytes (Figure 1). The AV node is located on the right side of the interatrial septum. It has a slow conduction velocity and thus delays impulse transmission (2,3,4). AV nodal transmission time is heart-rate–dependent and is controlled by autonomic tone and circulating catecholamines to maximize cardiac output at any given atrial rate.

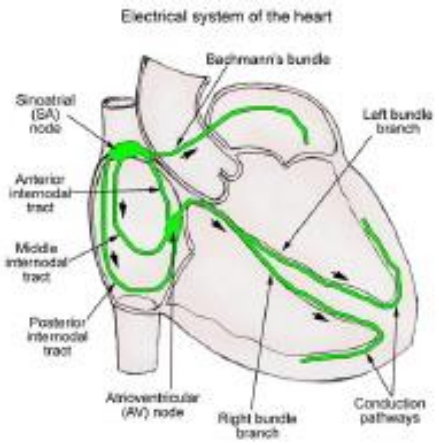

Figure 1: The normal impulse transmission through the heart.
Dr. Dale Dubin - Rapid interpretation of ECG 6th edition 2000 - page 13

The atria are electrically insulated from the ventricles by the annulus fibrosus (a thick fibrous membrane that prevent any passage of impulses) except in the anteroseptal region. There, the bundle of His, a continuation of the AV node, enters the top of the interventricular septum, where it bifurcates into the left and right bundle branches and terminates as the Purkinje fibers. The right bundle branch conducts impulses to the anterior and apical endocardial regions of the right ventricle. The left bundle branch fans out over the left side of the interventricular septum (2,3,4). Its anterior portion (left anterior hemifascicle) and its posterior portion (left posterior hemifascicle) stimulate the left side of the interventricular septum, which is the first part of the ventricles to be electrically activated. Thus, the interventricular septum depolarizes left to right, followed by near-simultaneous activation of both ventricles from the endocardial surface through the ventricular walls to the epicardial surface (2,3,4).

Physiology of the heart

Basic understanding of normal heart physiology is essential for the comprehension of the rhythm disturbances that will be explained in the following section.

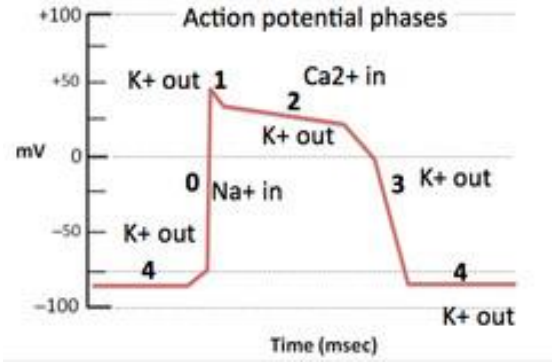

Figure 2: Standard model of a cardiomyocyte action potential Sherwood L. 2012 Human Physiology page 310

The passage of ions across the myocyte cell membrane is regulated by specific ion channels. This movement of ions causes cyclical depolarization and repolarization of the cell. This movement of ions across the myocyte cell membrane is called an action potential (Figure 2). The action potential of a working myocyte begins when the cell is depolarized from its diastolic −90 mV transmembrane potential to a potential of about −50 mV (5).

At this threshold potential, voltage-dependent fast Na channels open, causing rapid depolarization mediated by Na influx down its steep concentration gradient. The fast Na channel is rapidly inactivated and Na influx stops, but other time- and voltage-dependent ion channels open, allowing Ca to enter through slow Ca channels (a depolarizing event) and K to leave through K channels (a repolarizing event). At first, these 2 processes are balanced, maintaining a positive transmembrane potential and prolonging the plateau phase of the action potential. During this phase, Ca entering the cell is responsible for electromechanical coupling and myocyte contraction. Eventually, Ca influx ceases, and K efflux increases, causing rapid repolarization of the cell back to the −90 mV resting transmembrane potential. While depolarized, the cell is resistant (refractory) to a subsequent depolarizing event. Initially, a subsequent depolarization is not possible (absolute refractory period), and after a partial but incomplete repolarization, a subsequent slow depolarization is possible (relative refractory period) (4,5).
There are 2 general types of cardiac tissue:

- Fast-channel tissues
- Slow-channel tissues

Fast-channel tissues (working atrial and ventricular myocytes, His-Purkinje system) have a high density of fast Na channels and have action potentials characterized by little or no spontaneous diastolic depolarization (and thus very slow rates of pacemaker activity), very rapid initial depolarization rates (and thus rapid conduction velocity), and a loss of refractoriness coinciding with repolarization. As a result, these tissues have short refractory periods and the ability to conduct repetitive impulses at high frequencies (4,5).

Slow-channel tissues (SA and AV nodes) have a low density of fast Na channels and have action potentials characterized by more rapid spontaneous diastolic depolarization (and thus more rapid rates of pacemaker activity), slow initial depolarization rates (and thus slow conduction velocity), and a loss of refractoriness that is delayed after repolarization. Thus these tissues have long refractory periods and the inability to conduct repetitive impulses at high frequencies. About 1% of the cardiac muscle cells in the heart are responsible for

forming the conduction system that sets the pace for the rest of the cardiac muscle cells (4,5).

Normally, the SA node has the most rapid rate of spontaneous diastolic depolarization, so its cells produce spontaneous action potentials at a higher frequency than other tissues. Thus, the SA node is the dominant automatic tissue (pacemaker) in a normal heart (4). If the SA node does not produce impulses, tissue with the next highest automaticity rate, typically the AV node, functions as the pacemaker. Sympathetic stimulation increases the discharge frequency of pacemaker tissue, and parasympathetic stimulation decreases it.

Normal rhythm

The resting sinus heart rate in adults is usually 60 to 100 beats/min. Slower rates (sinus bradycardia) occur in young people, particularly athletes, and during sleep. Faster rates (sinus tachycardia) occur with exercise, illness, or emotion through increased sympathetic output and increased circulating catecholamines. Normally, a marked daytime variation in heart rate occurs, with lowest rates just before early morning awakening. A slight increase in rate during inspiration with a decrease in rate during expiration (respiratory sinus arrhythmia) is also normal; it is mediated by fluctuations in vagal tone and is particularly common among healthy young people. The oscillations diminish but do not entirely disappear with age. Absolute regularity of the sinus rhythm rate is pathologic and occurs in patients with autonomic denervation (eg, in advanced diabetes) or with severe heart failure (3,4).

Electrocardiogram

An electrocardiogram (ECG) is a recording of the electrical activity of the heart. Traditionally this is in the form of a transthoracic (across the thorax or chest, figure 3) interpretation of the electrical activity of the heart over a period of time, as detected by electrodes attached to the surface of the skin and recorded or displayed by a device external to the body (6).
Most cardiac electrical activity is represented on the ECG (Figure 4). The P wave represents atrial depolarization. The QRS complex represents ventricular depolarization, and the T wave represents ventricular repolarization. Atrial repolarization is not seen on the ECG because it occurs during the QRS depolarization.

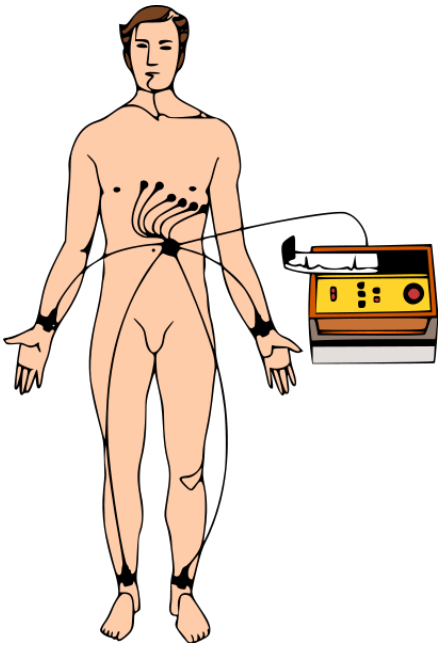

Figure 3: Figure showing a patient connected to the 10 electrodes necessary for a 12-lead ECG. Author: Madhero, March 2009

The PR interval (from the beginning of the P wave to the beginning of the QRS complex) is the time interval from the beginning of atrial activation to the beginning of ventricular activation. Much of this interval reflects the slowing of impulse transmission in the AV node. The R-R interval (time between 2 QRS complexes) represents the ventricular rate (6). This interval, as read by doctors on an ECG, is used to determine a patient's heart rate. The QT interval (from the beginning of the QRS complex to the end of the T wave) represents the duration of ventricular depolarization. Normal values for the QT interval are slightly longer in women; they are also longer with a slower heart rate.

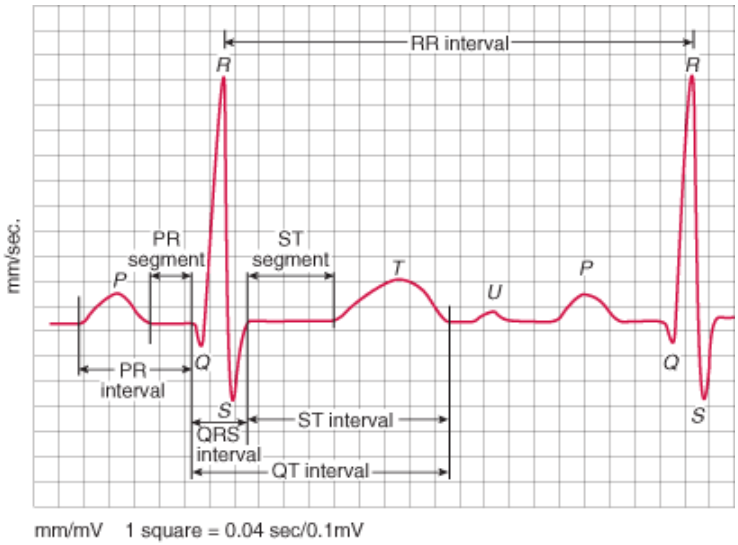

Figure 4: Different waves, segments and intervals of an Electrocardiogram.
Wagner, GS. Marriott's Practical Electrocardiography (11th edition), Lippincott Williams & Wilkins 2007. Photo by Ed Burns.

Table 1: Pathological patterns seen on ECG, followed by potential causes.
Kumar and Clark – Clinical Medicine 8th edition, chapter 14 – 2012.

Shortened QT-interval	Hypercalcemia, genetic abnormalities, hyperkalemia
Prolonged QT-interval	Hypocalcemia, genetic abnormalities, medications
Flattened or inverted T-waves	Coronary ischemia, hypokalemia, LV hypertrophy, Digoxin
Hyperacute T-waves	Possibly the first presentation of MI
Peaked T-waves, wide QRS, prolonged PR, QT short	Hyperkalemia – treat with calcium chloride, glucose and insulin.
Prominent U-waves	Hypokalemia

Classification of Cardiac Arrhythmias

The classification of arrhythmias is essential for quick recognition and prompt treatment. We classify arrhythmias simply as tachycardias (heart rate greater than 100 beats per minute) and bradycardias (heart rate less than 60 beats per minute).

Bradycardias are further classified into heart block or simple bradycardia (7). Tachycardias, on the other hand, can be divided into two categories based on the width of the QRS complex (Figure 5). If the QRS duration is less than 120 milliseconds, the tachyarrhythmia is narrow complex. If QRS duration is longer than 120 milliseconds, it is considered broad complex. When determining the QRS width, we always compare the findings on at least two different leads on the ECG, as a single lead interpretation is often insufficient (7).

If the arrhythmia has a narrow QRS complex, it is by definition a supraventricular tachycardia (SVT). These arrhythmias are usually benign, and often the patient can be treated and discharged from the emergency department to complete the evaluation as an outpatient. If the tachycardia has a wide QRS complex, it is either a ventricular tachycardia (VT), Ventricular fibrillation, or, a SVT with aberrant conduction or pre-excitation (7,8).

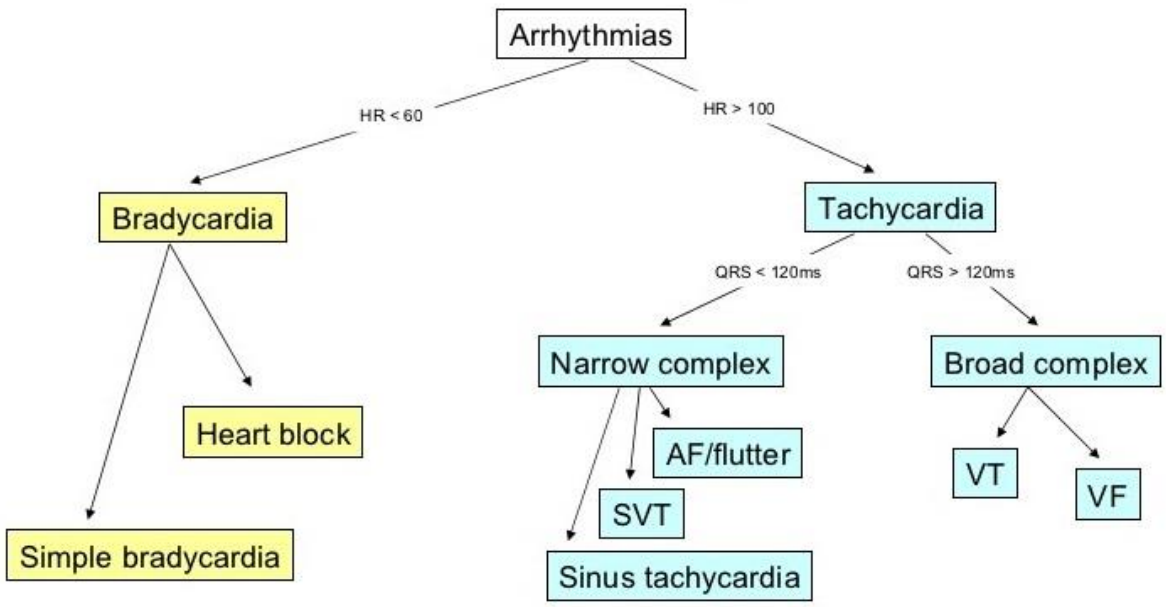

Figure 5: Classification of arrhythmias. Author: Sion Williams, March 2011, BSUH

Broad complex tachycardia

In contrast to the benign narrow complex tachycardias, these tachycardias are usually malignant and require hospitalization for further treatment.
It is extremely important to distinguish between VT and SVT as medication given for the treatment of SVT may be harmful or even fatal to a patient in VT.
The easiest way to recognize VT includes a QRS width greater than 120 milliseconds, a QRS axis in frontal plane (far left or right), concordance of QRS complexes across the precordium, and a time from onset of the R-wave to the nadir of S-wave in any precordial lead of more than 100 milliseconds (8).

Clinical data collected from the patient is also beneficial in establishing the correct diagnosis. A history of a previous myocardial infarction or structural heart disease is highly suggestive that the wide complex tachycardia is VT (8).
Usually, a proper and detailed history of illness and physical exam of the patient is enough to establish the presence or absence of structural heart disease in the patients. However, if history taking and physical examination is insufficient to give a concrete diagnosis, a transthoracic echocardiogram (TTE) will give more detailed information regarding the patient's cardiac anatomy and function.
Regarding the hemodynamic stability of the patient; Steinman and colleagues (9) demonstrated that VT is the correct diagnosis in 85% of patients who present with a wide complex tachycardia with normal hemodynamic stability.

Supraventricular tachycardias (SVT)

Supraventricular tachycardias are tachycardias that arise from foci of inappropriate electrical activity originating at or above the AV-node. As a systemic approach, every doctor should have in mind the different causes of a narrow complex tachycardia.
An easy algorithm to remember is to think anatomically, starting from where the heartbeat originates, at the SA-node, and ending at the atrioventricular AV-node. SVT can be caused by sinus tachycardias, atrial tachycardias, atrial flutter, atrial fibrillation, junctional tachycardia, AV nodal reentry, or AV reciprocating tachycardia using a bypass tract (10).
It is important to diagnose those different types of arrhythmias because treatment is specific for each one of them. For example, appropriate sinus tachycardia is treated by identifying the cause of the arrhythmia, such as fever, hypovolemia, or hyperadrenergic states rather than simply slowing the heart rhythm with beta-blockers. Patients, usually young adults, who have SVT usually present with palpitations (10). They notice a rapid heart rate and seek medical attention if it persists. They usually give a history of paroxysmal symptoms that have been present for years. Initial evaluation in the emergency department includes an ECG, history, and complete physical examination. If symptoms persist, a Valsalva maneuver or carotid sinus massage, might be useful in either terminating the arrhythmia or causing the loss of ventricular capture for a single beat to allow diagnosis of the arrhythmia mechanism (18). If the arrhythmia is not terminated by these bedside maneuvers, adenosine (6 or 12 mg intravenously) will usually cause AV nodal block, either terminating the arrhythmia or allowing the visualization of the underlying mechanism.

Adenosine has a rapid onset and short duration of action with a half-life of less than 10 seconds. It is important to administer the adenosine rapidly and to follow the infusion with a flush of saline for a better effect. The usual side effects of flushing, dyspnea, and chest discomfort are usually short lived (11). Other drugs that could be used as AV-blocking agents include beta-blockers and calcium-channel blockers. These drugs have a longer half-life and their own potential side effects, leading to the preferential usage of adenosine by emergency physicians due to its quick onset of action and short half-life.

Most SVTs are benign and can be treated in the emergency department, with the patient being discharged soon after (12,14). However, if a patient presents with new-onset atrial fibrillation, they cannot be discharged so quickly. This arrhythmia presents as an irregularly irregular ventricular rate with QRS complexes of variable duration. This arrhythmia may present with hemodynamic compromise if the ventricular rate is sufficiently fast. When treating this arrhythmia, calcium-channel blocking drugs and adenosine should be avoided. These drugs often increase conduction through the bypass tracts, while decreasing conduction through the AV node. The general effect causes an increase in the ventricular response rate and can cause hemodynamic compromise (13,14).

Atrial flutter and atrial fibrillation

Atrial fibrillation (AF) has its own special weight, as it's the most common abnormal heart rhythm. In the majority of patients, AF will be an accidental finding as it can be asymptomatic (15). However, it patients may also present with complaints of palpitations or chest pain, or episodes of fainting (15). AF can result in a reduction of cardiac output or formation of blood thrombi in the heart chambers. These thrombi may break apart, causing emboli to be released into circulation. The risk of stroke is increased 5 times in individuals with AF.
Atrial flutter and atrial fibrillation (Figure 6) represent a special challenge to the emergency department physician. Both arrhythmias may present as incidental findings in a patient who presents to the emergency department for other complaints. These arrhythmias are common in the general population, with the incidence increasing with age. For both, hypertension is considered the most common alterable risk factor.

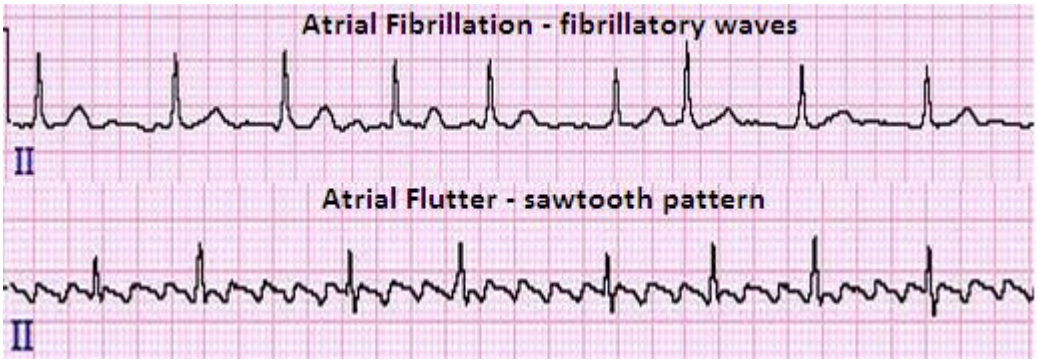

Figure 6: Pay attention to the missing p-waves and irregular beat in atrial fibrillation and the sawtooth pattern of the atrial flutter. John R. Hampton – 150 ECG problems 3rd edition 2008 page 47.

Ventricular tachycardia

This is a potentially life-threatening arrhythmia that emergency department physicians dread, due to its potential of evolving into VF, asystole, or sudden cardiac death (16).
Most patients who present with these arrhythmias have underlying heart disease. If the patient is hemodynamically unstable, perform an electrical cardioversion as quickly as possible. The rate of survival depends on the speed of cardioversion (16).
The most common cause of VT is myocardial scarring from a previous myocardial infarction. This scar is composed of fibrous tissue, meaning that it cannot conduct electrical activity, so there is a potential circuit around the scar that results in the tachycardia.
Patients who will be successfully treated will have to be admitted to the cardiology department for further evaluation and treatment.

Bradycardia

Bradycardias are often asymptomatic, and occur more often in older people. A rate below 50 beats per minute can cause fatigue, weakness, dizziness and rarely fainting. A heart rate below 40 beats per minute is called absolute bradycardia.
Bradycardia usually occur during sleep, and in well-trained athletes, and is considered normal.
The mechanisms that act on the heart and cause bradycardia can either be depressed automaticity of the heart, conduction block, or escape pacemakers.
For the emergency room physician, and our algorithm, we classify bradycardias as disorders of the SA-node and disorders of the AV-node. With SA-node dysfunction (Sick sinus

syndrome), there may be disordered automaticity or impaired conduction of the impulse from the sinus node into the surrounding atrial tissue (an "exit block"). Second-degree sinoatrial blocks can be detected only by use of a 12-lead EKG (17). It is difficult and sometimes impossible to assign a mechanism to any particular bradycardia, but the underlying mechanism is not clinically relevant to treatment, which is the same in both cases of sick sinus syndrome: a permanent pacemaker.

AV-node disturbances (AV-blocks) may result from impaired conduction in the AV node, or anywhere below it, such as in the Bundle of His. The clinical relevance affecting to AV blocks is greater than that of sinoatrial blocks (17).

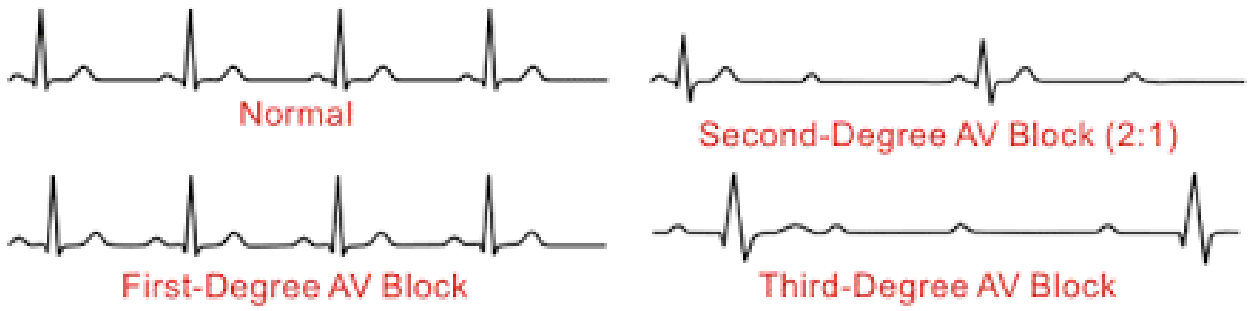

Figure 7: A comparison of the three different AV blocks to the normal ECG. John R. Hampton – 150 ECG problems 3rd edition 2008 page 17.

Diagnosis and Presentation of Cardiac Arrhythmias

The presentation of cardiac arrhythmias can range from none to syncope, loss of consciousness, or sudden cardiac death. In general, the more severe symptoms usually present in patients with underlying structural heart disease.
For example, patient with an anatomically normal heart may have sustained monomorphic VT that is completely tolerated without any symptoms of syncope. In contrast, another patient with left ventricular dysfunction with VT may be hemodynamically unstable or have marked symptoms present.
Different arrhythmias have different symptoms. Some of the common presenting symptoms in the emergency department are lightheadedness, shortness of breath, dizziness, syncope, fluttering or palpitations, pounding, chest discomfort, and painful extra beats.
For the experienced physician, some descriptions of symptoms can raise the index of suspicion and provide some differential diagnoses about the type of arrhythmia. If a young person presents with a sustained regular palpitations without any evidence of structural heart disease, the first differential in mind should be SVT caused by AV-nodal re-entry or SVT caused by an accessory pathway. An isolated premature beat, again in a normal heart, suggests premature atrial contractions (PAC) or premature ventricular contractions (PVC).

Syncope has a vast list differential diagnosis and requires a proper history taking and physical exam. But again, the presentation of syncope in the setting of noxious stimuli such as pain, particularly when preceded by vagal-type symptoms (nausea, vomiting, diaphoresis), suggests neurocardiogenic or vasovagal syncope (18).

The most common symptom of arrhythmia is palpitation - an abnormal awareness of heartbeat. This feeling could be infrequent, frequent or continuous. Some of this arrhythmias are harmless but some predispose to adverse outcomes.
The same is with silent arrhythmias. For example, AF, can be silent until the patient presents with stroke, but some are symptomatic continuously, but never increase the mortality or disability.

Bradyarrhythmias are usually well tolerated. But when the heartbeat is too weak to produce sufficient cardiac output, blood pressure drops and may cause lightheadedness, dizziness, or syncope.

Although some emergency physicians feel comfortable dealing with arrhythmias and recognize the common signs, all patients require an electrocardiogram (ECG) to establish tbhe diagnosis of an arrhythmia (19). Simple auscultation of the heart is never enough.
A 24 hour ECG, called a Holter monitor, may assist in confirming the diagnosis when the arrhythmia occurs sporadically, as is often the case.

Treatment of Arrhythmias in the emergency department

Emergency treatment of bradyarrhythmias

As previously mentioned, bradyarrhythmias are arrhythmias when the heart rate is below 60 beats per minute. This can be normal in the majority of people and require no further treatment. However, a ventricular rate less than 40 beats per minute usually requires emergency treatment (2,3,4).

The first step in the approach to a bradyarrhythmic patient is to try to find the cause of the bradyarrhythmia, e.g.: electrolyte disturbances, excessive vagal stimulation, and pericardial tamponade.

The next step will be determined by the hemodynamic stability of the patient. Severe bradycardia with loss of consciousness should be treated with resuscitation immediately, until a temporary pacemaker is available.

The following is an algorithm for the emergency treatment of bradyarrhythmias:

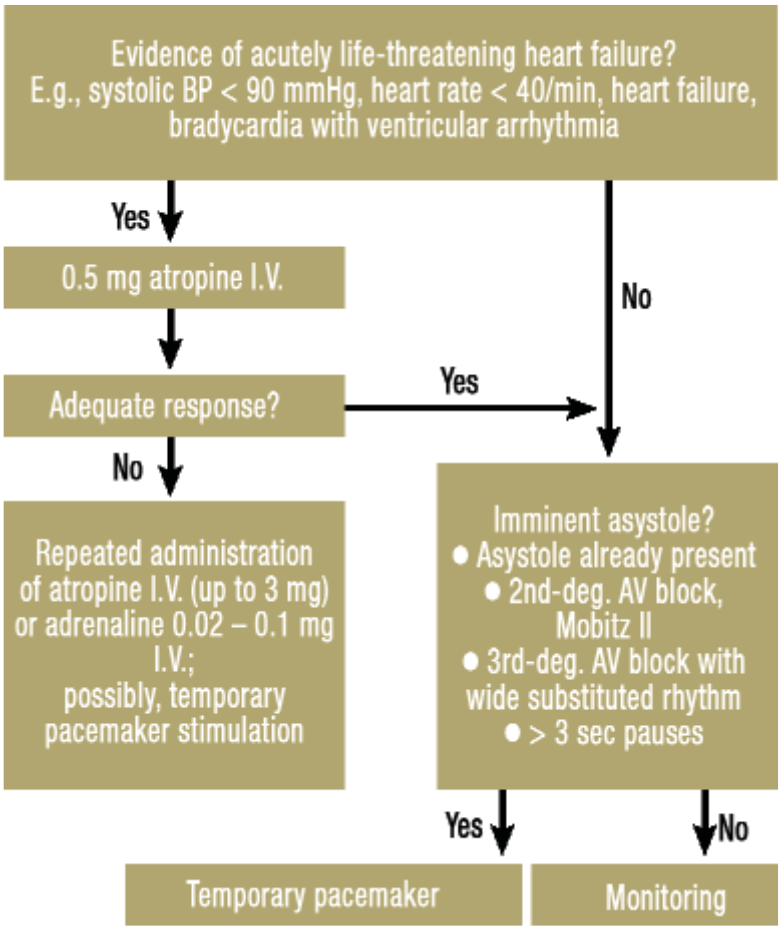

Figure 8: Algorithm for fast treatment of bradyarrhythmia. European Resuscitation Council Guidelines for Resuscitation 2005. Resuscitation 2005; 67: 39–86.

As seen in Figure 8, for a symptomatic but hemodynamically stable patient, the treatment of choice is atropine (20).

Atropine is an antimuscarinic agent that is given for bradycardia, asystole, and AV-block above the bundle of HIS. It is given at a dosage of 0.5 mg every 2-5 minutes, up to a maximum dose of 0.04mg/kg. An easier way to think of this, is 3 mg for every 70kg.

The contraindication for atropine, is an AV-block below the bundle of His, meaning Mobitz type 2 or third-degree AV-block with a substituted rhythm and a widened QRS-complex.

In those patients, atropine can cause paradoxical bradycardia. In this situation, catecholamines should be given to the patient, until a pacemaker can be implanted. The catecholamines used in clinical practice are adrenaline (0.1 mg IV) and orciprenaline (0.5 mg IV).

Emergency treatment of tachyarrythmias

Tachycardia is when the heart rate is above 100 beats per minute. A heart rate between 100-150 bpm is called a simple tachyarrhythmia, while a heart rate between 150-250 bpm is called a paroxysmal tachyarrhythmia. The arrhythmias with the fastest heart rates are atrial flutter with 250-350 bpm and atrial fibrillation with greater than 350 bpm. Patients with heart rates above 150 bpm usually present with symptoms to the emergency department.
The first step in the treatment of tachyarrhythmias will always depend on the hemodynamic stability of the patient.

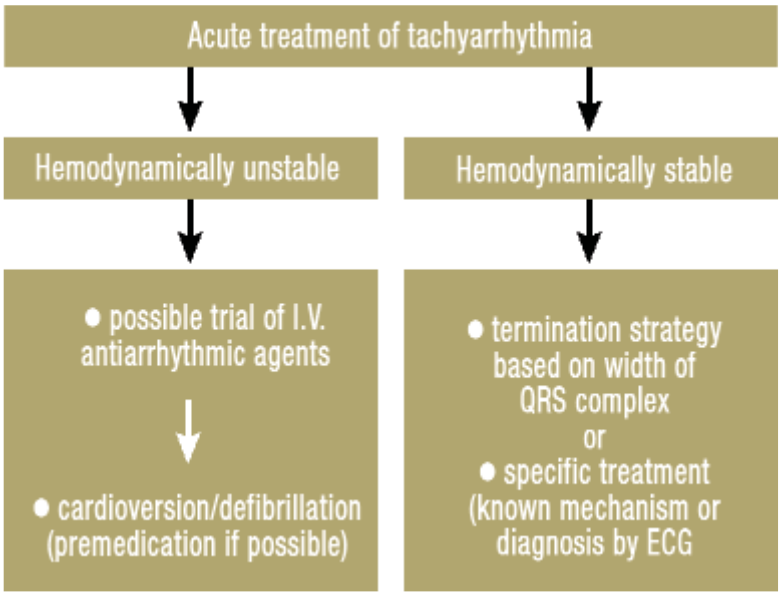

Figure 9: Algorithm for treatment of tachyarrhythmia. European Resuscitation Council Guidelines for Resuscitation 2005. Resuscitation 2005; 67: 39–86.

Hemodynamically unstable patients are those presenting with shock, alteration of consciousness, or pulmonary edema. This patient group should be treated as quickly as possible with intravenous antiarrhythmic agents or cardioversion/defibrillation (20).
Different arrhythmias will respond to different energies of defibrillation. Fibrillation and flutter usually respond to lower energies of 100 joules, while polymorphic ventricular tachycardia or VF should be treated with defibrillation energies of 200-300 joules. If the usage of lower defibrillation energies fails, which is often the case, then higher energies (360 J) should be tried.
In addition to the defibrillation, IV administration of amidarone (150-300 mg IV) should be given (recommendation from a prospective, randomized study) (21).
If a patient is conscious, hemodynamically unstable, and requires defibrillation, a sedative or analgesic should be given prior to defibrillation.

A hemodynamically stable patient is treated with medications. The emergency physician should monitor the patient closely all the time and the patient should be placed on ECG monitoring.
The ECG will help the doctor to determine which type tachycardia is present; the type of tachyarrhythmia dictates the type of pharmaceutical treatment (22).
The width of the QRS is the deciding criteria for the classification of our arrhythmias.

A narrow QRS complex is considered to reflect physiological anterograde conduction in the His-Purkinje system, so we presume that the patient will have a supraventricular tachycardia originating above the bundle of His.
On the other hand, a wide QRS complex is an indication of ventricular tachycardia and ventricular fibrillation (22).

Antiarrhythmic drugs
The Cardiac Arrhythmia Suppression Trial (CAST-study), published in 1989, changed the entire use of antiarrhythmic drugs.
The drugs that were used in the study – moricizine, flecainide, encainide – were known to have ventricular arrhythmia suppression properties. However, CAST study demonstrated an increase mortality in patients treated with the wrong antiarrhythmic drug for the wrong arrhythmia (23).
In 1970, Dr. Vaughn-Williams from Oxford University, introduced the first classification of anti-arrhythmic drugs. With regards to management of atrial fibrillation, Class I and III are used in rhythm control as medical cardioversion agents, while class II and IV are used as rate control agents. There are five main classes in the Vaughan-Williams classification.

Table 2: Discerption of the different classes of antiarrhythmic drugs.
Singh Vaughan Williams 1970 Oxford Univeristy. From Chaudhry G, Muqtada MD, Haffajee 2000;28:N158-N164.

Class	***Mechanism of Action***	***Example***	***Clinical indications***
Ia	Na-channel blockers – intermediate association/dissociation	Quinidine Procainamide Disopyramide	-Ventricular arrhythmias -Prevention of paroxysmal recurrent atrial fibrillation -Procainamide in WPW-syndrome
Ib	Na-channel blockers – fast association/dissociation	Lidocaine Tocainide	-Ventricular tachycardia
Ic	Na-channel blockers – slow association/dissociation	Flecainide	-Prevents paroxysmal atrial fibrillation -Treats recurrent tachyarrythmias
II	B-blockers	Cardeviol Propanolol Timolol Atenolol Bisoprolol	-Decrease MI mortality -Prevent recurrence of tachyarrythmias -Propanolol has some class I action
III	K-channel blockers	Amiodarone Sotalol Ibutiide	-WPW-syndrome -Sotalol is given for ventricular tachycardia and AF -Ibutilide is given for Atrial flutter
IV	Ca-channel blockers	Verpamil Diltiazem	-Prevent recurrence of Paroxysmal supraventricular tachycardia -Reduce ventricular rate in patients with AF

Class I drugs
The class I antiarrhythmic agents block sodium channels and interfere with the cardiac action potential. These agents are also called membrane-stabilizing agents, as they decrease the excitogenicity of the plasma membrane.
Class I agents are divided into 3 groups, based upon their effect on the length of the action potential (24,25).

- Ia lengthens the action potential (right shift)
- Ib shortens the action potential (left shift)
- Ic does not significantly affect the action potential (no shift)

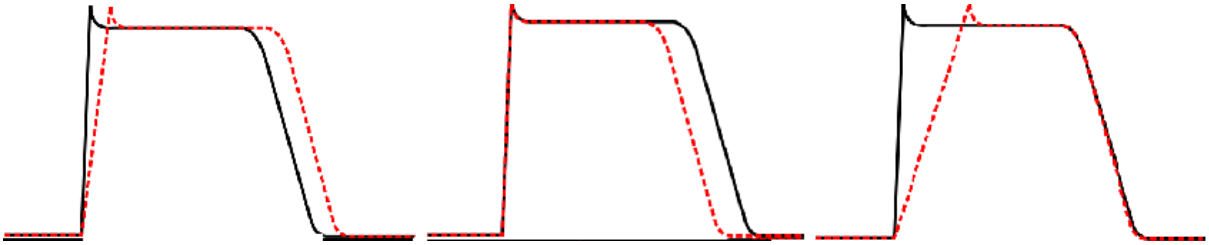

Figure 10: Class I agents and their effect on action potential.

Milne JR, Hellestrand KJ, Bexton RS, Burnett PJ, Debbas NM, Camm AJ (February 1984).

Class II drugs

These agents act by blocking the effect of catecholamines at B1-adrenergic receptors, which will lead to decreased sympathetic activity on the heart (24).
The most common indication for the use of these agents is supraventricular tachycardias.
Most SVTs are caused by a reentrant circuit involving the AV node or use the AV node to conduct to the ventricle. Therefore, long-term treatment is directed at decreasing conduction through the AV node. Beta-blockers and calcium channel blockers are predominantly valuable for treating these arrhythmias. Once patients have been stabilized in the emergency department, they can usually be discharged on either a long acting beta-blocker or calcium-channel blocker to complete their evaluation in the outpatient clinic.

Class III drugs
Class III agents act upon and block the potassium channels, which prolongs the repolarization and action potential (26). Since these agents do not affect the sodium channel, conduction velocity is not decreased. They are mostly described for the prevention of re-entry arrhythmias. The effect of slowed atrial-ventricular myocyte repolarization usually leads to a prolonged QT-interval that can be obtained by the emergency physician (26).

Class IV drugs
Class IV agents act on the AV-node, blocking its calcium channels, resulting in prolonged AV-nodal conduction time. This causes a reduction in the contractility of the heart (negative inotropic effect).
In contrast to beta-blockers, they allow the body to retain adrenergic control of heart rate and contractility (27).

Usually, type Ia and III agents are initiated in the hospital with patient monitoring. Ic agents, however, are safe when used in a normal heart, i.e.: in the absence of structural heart anomalies. Amidorone, because of its long half-life (43 days), can be given at low doses in an outpatient setting, in the absence of bradycardia or left ventricular dysfunction.

Pacemakers and Defibrillators

The implementation of a pacemaker requires specific indications according to the American College of Cardiology (ACC) and American Heart Association (AHA) guidelines.
The correlation of symptoms with underlying bradyarrhythmias or heart blocks type 2 and 3 is required (29).
The implantable cardioverter-defibrillator (ICD) is indicated for sustained VT or VF, survivors of cardiac arrest, or sustained monomorphic VT (29).
Based on the MADIT II study, ICDs will routinely be implanted in patients with LV dysfunction, EF of less than 35%, and a previous myocardial infarction. The emergency indications for ICD implantation include patients with syncope who have dilated cardiomyopathy, patients who have hypertrophic obstructive cardiomyopathy, and especially patients who are at high risk for sudden cardiac death (family history of sudden cardiac death, syncope, non-sustained VTs).

In the last 50 years, since the development of the pacemaker, there has been vast improvement in a pacemaker's functionally and longevity (28).
The pacemakers used today can last more than 10 years and pacemaker leads constantly improving. Unfortunately, in some instances leads do need to be changed due to a manufacturing defect or secondary to infection. Lead extraction, after a long time, is usually problematic due to being heavily fibrosed by endovascular tissue.
The ICDs used today have a battery life of 6-8 years, with improvements to battery life in development (30). Patients with an implanted ICD usually need testing of their pacing every 6-12 months.
An ICD can terminate VT or VF with a shock, but it can also terminate sustained VT with anti-arrhythmic pacing.

Defibrillators can be single chamber or dual chamber. Single chamber devices are recommended for patients with severe LV dysfunction, normal and intact SA- and AV-nodes, and have no indication for pacing.
Dual chamber devices are reserved for patients with an abnormal SA- or AV-node or conduction system who are prone to frequent episodes of supraventricular arrhythmias, such as atrial fibrillation (2).

Atrial Fibrillation

Atrial fibrillation (AF) is the most common sustained abnormal heart rhythm worldwide, with an estimated 9 million people (1-2% of the general population) currently affected in Europe and USA (32,36).

AF has a strong association with other cardiovascular diseases, such as coronary heart diseases, heart failure, valvular heart disease, diabetes mellitus, and most commonly, hypertension (34).

It is characterized by an irregular and rapid heartbeat caused by an unknown mechanism.

The normal impulses generated by the SA-node are overwhelmed by disorganized electrical impulses originating in the roots of the pulmonary veins, leading to irregular conduction of ventricular impulses that generate the heartbeat.

Genetics, atrial ischemia or inflammation, metabolic or hemodynamic stress are all claimed to be risk factors for the development of AF.

The primary factors determining AF treatment (31) are the duration of the fibrillation and evidence of hemodynamic instability. Cardioversion is indicated with new onset AF (onset within 48 hours) and in patients with hemodynamic instability. If rate and rhythm control cannot be maintained by medication or cardioversion, it may be necessary to perform radiofrequency ablation (RFA) of abnormal electrical pathways.

AF is associated with a 5 times increased risk for stroke (33), and one in five strokes is attributed to this arrhythmia. Strokes caused by cardiogenic emboli are associated with high mortality, and those who do survive, are usually left with great disability.

The reason for why we lack an early recognition of AF is the “silent” nature of this rhythm disturbance.

Clinically, and in the emergency room, we distinguish five different types of AF, based on the presentation and duration of the arrhythmia:

1. First diagnosed (resent onset)
2. Paroxysmal AF
3. Persistent AF
4. Long-standing persistent AF
5. Permanent AF

The first type, first diagnosed, is given for every patient with newly diagnosed AF, irrespective of the duration of the arrhythmia or presence of related symptoms.

The second type, paroxysmal AF, is usually self-terminating within 48 hours, a time which is clinically important regarding the treatment. Persistent AF, the third type, is diagnosed when an AF episode lasts longer than 7 days or requires termination by cardioversion – either by drugs or direct current cardioversion (DCC). The fourth type, longstanding persistent AF, is diagnosed when AF has been present for more than one year. The last type, permanent AF, is said to exist when the presence of the arrhythmia is accepted by the patient and physician (34).

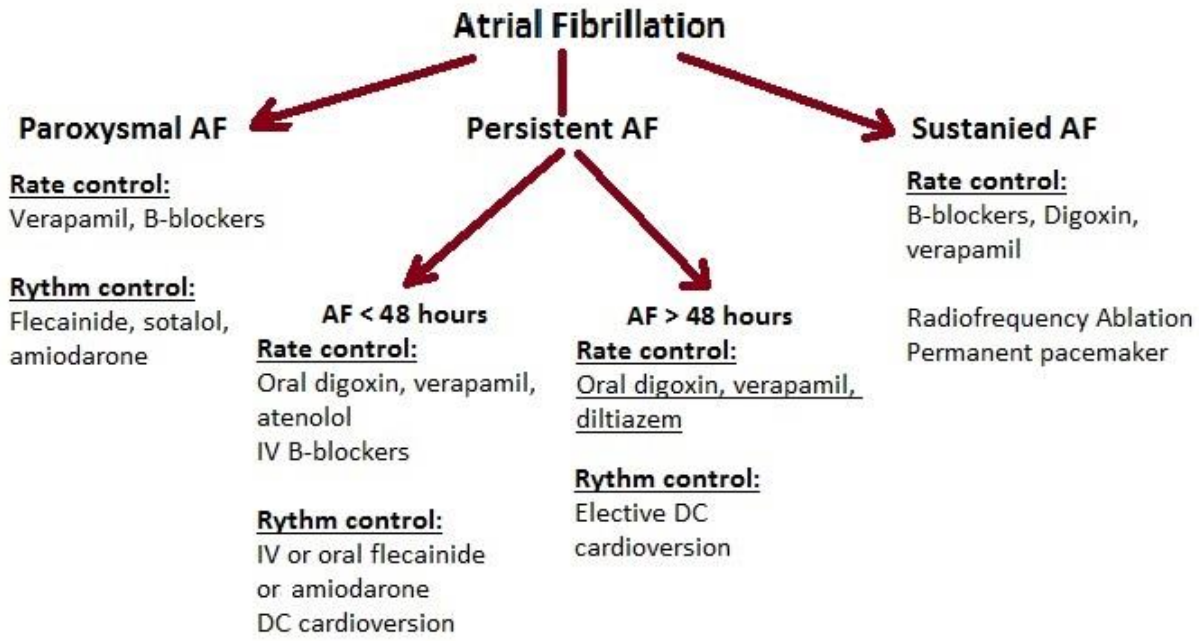

Figure 11: Management Strategies for Atrial Fibrillation. Source: Br J cardiol 2003

Acute AF (less than 48 hours of duration):

If the patient presenting to the emergency department is unwell or hemodynamically unstable, the first choice of treatment should be oxygen and emergency cardioversion for rhythm control. If cardioversion is unavailable or not possible, then pharmacological conversion using amiodarone should be attempted (35).

For rate control, the first line drugs are diltiazem (60-120 mg / 8 hours orally) or verapamil (40-120 mg / 8 hours orally). Second line treatment is digoxin or amiodarone.

The emergency physician should also start the patient on low molecular weight (LMWH), to anticoagulate the patient and prevent mural thrombus formation (37).

Drug cardioversion is often preferred by physicians, with the most commonly used agent being amiodarone, given either IV (5mg/kg over 1 hour, then 900mg over 24 hours) or orally (200mg every 8 hours for one week, then 200mg over 12 hours for 1 week, and 100mg over 24 hours maintenance).

Chronic AF (longer than 48 hours of duration)

The main goal in the treatment of these patients is rate control and anticoagulation. B-blockers or calcium channel blockers are first line treatments of choice. If the usage of one of two drugs is not successful, the physician should add digoxin, then consider amiodarone (38).

Rhythm control is the goal of treatment in younger or symptomatic patients. If the time since the onset of AF unknown and if cardioversion has been chosen as the treatment method, echocardiography should be performed and the patient have to be pre-treated for 1 week with sotalol or Amiodarone, together with anticoagulation (38). If there is a risk of cardioversion failure, then pharmacological cardioversion with flecainide should be the next best step.

Anticoagulation and AF

When a patient presents with AF, it is very important to determine the risk of an embolism and to start the patient on anticoagulation. The most accurate, and most widely used scoring system, is the CHADS2 score (39).

CHADS2 score is a clinical prediction system used for estimating the risk of developing a stroke in patients with a known AF (39,40). This scoring system determines whether or not treatment is required with anticoagulation therapy or antiplatelet therapy (41).
A higher CHADS2 score corresponds to a greater risk of stroke, while a lower score corresponds to a lower risk of stroke.

Table 3: Abbreviation for CHADS2 and the points awarded for each. VA Palo Alto Medical Center and at Stanford University: the Sports medicine Program and the Cardiomyopathy Clinic 2007-09-14.

	Conditions	points
C	Cognitive heart failure	1
H	Hypertension >140/90 mmHg	1
A	Age > 75 years	1
D	Diabetes Mellitus	1
S2	Prior Stroke, or TIA	2

Table 4: Annual stroke risk, in regard to the CHADS2 score. Karthikeyan G, Eikelboom JW. The CHADS2 score for stroke risk stratification in atrial fibrillation--friend or foe? Thromb Haemost. 2010 Jul 5;104(1):45-8.

CHADS2 Score	**Stroke Risk %**
0	1.9
1	2.8
2	4.0
3	5.9
4	8.5
5	12.5
6	18.2

A score of 0, is considered a low risk patient and require no aspirin or anticoagulants. A score of 1 is moderate risk and the patient should be started on aspirin. A score of 2 or higher is high risk patients, requiring LMWH or warfarin.

Conclusion

The understanding and quick recognition of arrhythmias and mastering the treatment is one of the most important tasks for an emergency physician (2, 3).
The diagnosis of arrhythmias can be challenging, especially in stressful conditions like in the emergency department. Ideally, the patients should be treated in areas with full access to ECG monitoring, oxygen and external defibrillator.
A good understanding of the diversity of conditions that can predispose a patient, with a normal or abnormal heart, to arrhythmias is essential so that a physician can easily choose the appropriate treatment method.
The algorithm that was presented in this thesis and the recommended therapeutic strategies should function as a rapid guide for the emergency physician.
Recognition and early treatment of arrhythmias have a very good prognosis, with a 95% success rate.

The most important decision-making criteria in the emergency treatment of cardiac arrhythmia are hemodynamic stability or instability in tachycardia and the width of the QRS complex. This information serves as a marker for a presumed ventricular or supraventricular origin of the disturbance.
Additionally, all clinical staff working in the emergency department should be trained in basic life support, intermediate life support, and ideally, advanced life support.

With the development of modern medicine, our elderly population has been increasing. Additionally, the number of obese persons is on the rise. With the increase of these two population groups, the prevalence of obesity- and age-related cardiovascular diseases also increases.
The population living with, or suffering from cardiac diseases is more than ever. It is estimated that 36% of people post myocardial infarction will present with an arrhythmia within two years after their diagnosis (42).

As a result of continuing research and better education, the number of deaths resulting from arrhythmias and the number of medical errors have been reduced much more radically than was previously possible.
Cardiology, cardiac pharmacy and cardiac models, are some of the most widely developed areas in medicine with continuous research with the goal for a better tomorrow.

Acknowledgment

This assignment could not be completed without the effort and co-operation of my mentor, Professor Vesna Degoricija MD. PhD, an inspiring teacher that followed this paper to its end. Last, but not least, I would like to express my gratitude to some special friends and for my family for their love and support.

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Biography

Haider Sabhan, was born on July 20, 1988 in Baghdad, Iraq but spent the majority of his life in Stockholm, Sweden. After completing high school in June 2008, he was enrolled to the Pre-Medical International course in Sweden as a preparation before medical studies.
In July 2009, he passed the entrance exam for Zagreb Medical University and started the program in September of the same year.

After graduation in July 2015, he plans to complete an internship and the medical board exam in Sweden in order to be licensed in Europe. After the internship, he is planning to start a medical career in Berlin, Germany.

Haider has a big interest in movies and music. He also spends most of his time doing sports or reading books.