Shendi University

Faculty of Graduate Studies & Scientific Research

Research about:

Impact of an educational program on recognition and management of Ventricular arrhythmias guideline among critical care nurses in Khartoum City, Sudan

A thesis submitted in the fulfillment of the requirements for the Degree of Doctor of Philosophy in medical surgical nursing

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﴿ بِسْمِ اللّهِ الرَّحْمَنِ الرَّحِيمِ ﴾

قال تعالى:

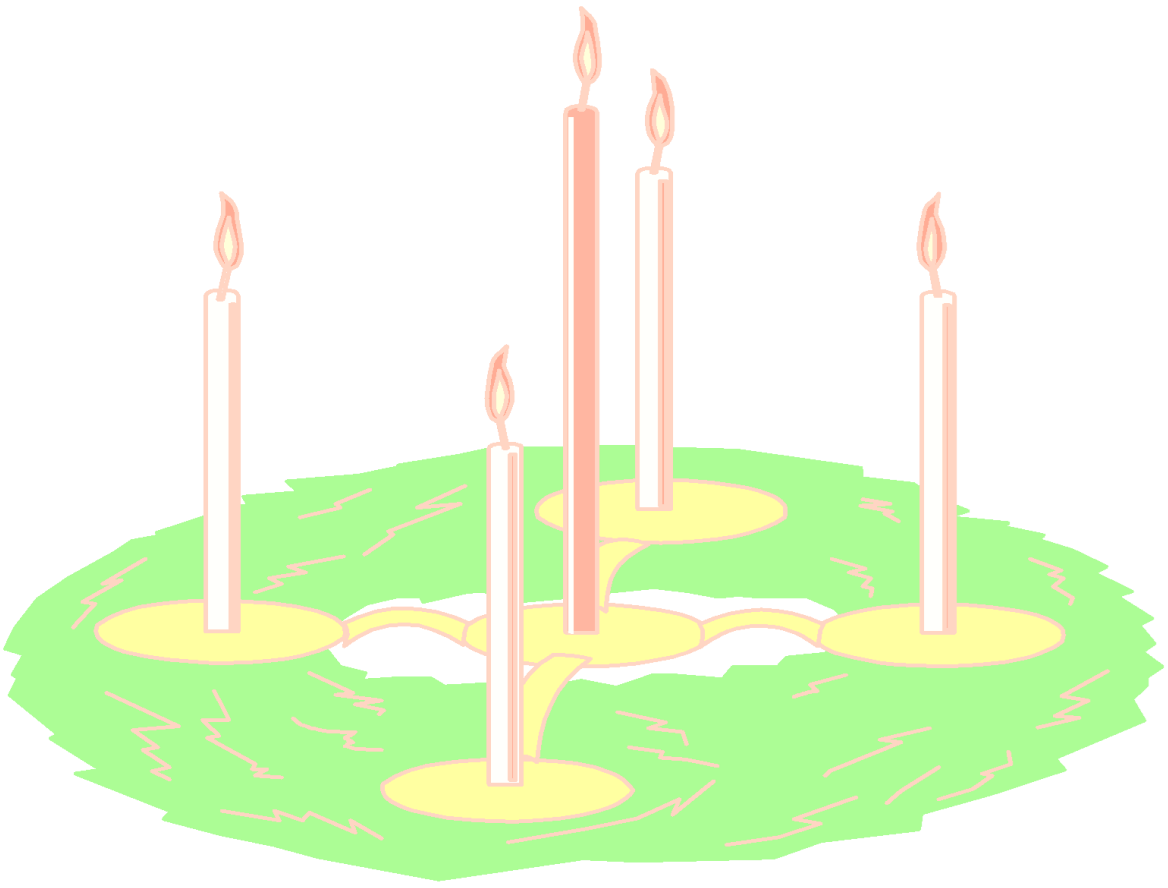
﴿٣١﴾ مِنْ أَجْلِ ذَلِكَ كَتَبْنَا عَلَى بَنِي إِسْرَائِيلَ أَنَّهُ مَن قَتَلَ نَفْسًا بِغَيْرِ نَفْسٍ أَوْ فَسَادٍ فِي الْأَرْضِ فَكَأَنَّمَا قَتَلَ النَّاسَ جَمِيعًا وَمَنْ أَحْيَاهَا فَكَأَنَّمَا أَحْيَا النَّاسَ جَمِيعًا وَلَقَدْ جَاءَتْهُمْ رُسُلُنَا بِالْبَيِّنَاتِ ثُمَّ إِنَّ كَثِيرًا مِّنْهُم بَعْدَ ذَلِكَ فِي الْأَرْضِ لَمُسْرِفُونَ﴿٣٢﴾

﴿ سورة المائدة﴾ صدق الله العظيم

DEDICATION

This work dedicated to all the candles that fired to lighten my way:

- *To my father & mother …*
- *To my brothers & sisters …*
- *To my partner …*
- *To my daughter and son …*
- *To my colleagues and friends …*



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Abstract

Introduction: Nurses' knowledge regarding the ventricular arrhythmias (ventricular fibrillation and pulseless ventricular tachycardia) causes, signs and symptoms, diagnostic method, and appropriate management based on updated standard guideline is very important, in order to provide high-quality patient-centered care. Prompt recognition and management of ventricular arrhythmias by the nurse will prevent the fatal complications as sudden cardiac death.

Aim: The study aimed to evaluate the impact of an educational program on recognition and management of ventricular arrhythmias guideline among critical care nurses.

Methodology: The nature of this study was a hospital-based quasi-experimental study, was conducted in Sudan, Khartoum city at Sudan heart center, Khartoum teaching hospital and Alshaab teaching hospital. One hundred sixty-eight nurses were included as all the entire population taken as a sample; the data was collected in three phases by using a self-administered questionnaire and observational checklist. Phase one; include an orientation session about the training program, and the pretest was obtained. Phase two; include an implementation of the training program and first posttest was obtained. Phase three (follow up phase) after one month and six month from implementation of the training program same self-administrative questionnaire and the observational checklist formats were used to make sure of the consistency .The data was analyzed by computer software program (SPPS) (version 21), and presented in form of tables and figures.

Result: The study data revealed that, among 168 critical care nurse (77 %) of study group were female while, and Half of them (49 %) their years of experience less than one year.

The nurses' knowledge regarding recognition and management of ventricular fibrillation and pulseless ventricular tachycardia was intermediate (54%) and mean was (0.54) pre intervention, and in the third post intervention the percentage reached (87%) and mean was (0.87).

Overall nurses' skills regarding management of ventricular fibrillation and pulseless ventricular tachycardia; the mean was (1.44) in pretest, increased to (2.79) in the first posttest, in the second posttest the mean was (2.92) and in the third posttest the mean was reached (3).

Conclusion: There was highly statistically significant association between the nurses' performance and implementation of this program in pre, post I, and post II posttest III.

Recommendations: Specific courses about the recognition and management of ventricular arrhythmias (ventricular fibrillation and pulseless ventricular tachycardia) must be establish for critical care nurses to enhance them to be more knowledgeably

and skillfully in recognition and management of ventricular arrhythmias. On the other hand, policies and regulations from Ministry of Public Health and Sudanese national council for medical and health professions should maintain optimum level of nurses' knowledge and skills regarding ventricular arrhythmias recognition and management, especially for critical care nurses.

مستخلص الدراسة

مقدمة: تعتبر معرفة الممرضين بشأن عدم انتظام ضربات القلب البطيني (الرجفان البطيني وعدم انتظام دقات القلب البطيني غير النبضي) الأسباب، الأعراض والعلامات، طريقة التشخيص والخطة العلاجية المستندة إلى إرشادات قياسية محدثة أمرًا مهمًا للغاية، من أجل توفير رعاية عالية الجودة تتمحور حول المريض. السرعة في التعرف وعلاج عدم انتظام ضربات القلب البطيني من قبل الممرضين سيمنع المضاعفات القاتلة مثل الموت القلبي المفاجئ.

الهدف: هدفت الدراسة إلى تقييم أثر البرنامج التعليمي في التعرف وعلاج عدم انتظام ضربات القلب البطيني من قبل ممرضي الرعاية الحرجة

المنهجية: طبيعة هذه الدراسة هي دراسة شبه تجريبية في المستشفيات، أجريت في السودان مدينة الخرطوم بمركز السودان للقلب ومستشفى الخرطوم التعليمي ومستشفى الشعب التعليمي. تم اختيار عينة مكونة من 168 ممرض عناية حرجة كتغطية شاملة، تم جمع البيانات في ثلاث مراحل باستخدام استبيان يتم تعبئته بواسطة المشاركين وقائمة تحقق. المرحلة الأولى تشمل جلسة تنويرية حول برنامج الدراسة وأهدافه وتم الحصول على الاختبار الأول القبلي. المرحلة الثانية وتشمل تنفيذ البرنامج التدريبي وبعد الانتهاء من البرنامج تم جمع البيانات مرة ثانية. المرحلة الثالثة (مرحلة المتابعة) بعد شهر وستة أشهر من تنفيذ البرنامج التدريبي تم استخدام نفس الاستبيان وقائمة التحقق مرة اخري للتأكد من ثبات المعلومات والمهارات المتعلمة. تم تحليل البيانات باستخدام الحزمة الإحصائية (الإصدار 21)، تم عرض البيانات في شكل جداول وأرقام.

النتائج: أوضحت نتائج الدراسة أن 168 من ممرضي الرعاية الحرجة (77٪) من مجموعة الدراسة كانت إناث، ونصفهم (49٪) سنوات خبرتهم اقل من سنة واحدة.

كانت حصيلة المعرفة الأساسية والمعرفة بالمبادئ التوجيهية للممرضين عن الرجفان البطيني وعدم انتظام دقات القلب البطيني غير النبضي متوسطة بنسبة (54٪) والوسط الحساب (0.54) قبل تطبيق البرنامج التدريبي، وفي الاختبار الثالث بعد تطبيق البرنامج بلغت النسبة (87٪) والوسط الحساب (0.87).

وبشكل عام زادت مهارات الممرضين المتعلقة بعلاج الرجفان البطيني وعدم انتظام دقات القلب البطيني غير النبضي وفقاً للمبادئ التوجيهية، كان الوسط الحسابي (1.44) قبل تطبيق البرنامج التدريبي، ذاد الوسط الحسابي الي (2.79) في الاختبار البعدي الاول، وفي الاختبار البعدي الثاني كان الوسط الحسابي (2.92) وفي الاختبار البعدي الثالث وصل الوسط الحسابي الي (3).

الخلاصة: كان هناك ارتباط ذو دلالة إحصائية عالية بين أداء الممرضات وتنفيذ هذا البرنامج عند مقارنة مرحلة ما قبل الاختبار مع الاختبار البعدي الأول والثاني والثالث.

التـــوصيـــات: يجب تصميم وتنفيذ دورات تدريبية عن معرفة وعلاج عدم انتظام ضربات القلب البطيني (الرجفان البطيني وعدم انتظام دقات القلب البطيني غير النبضي) لممرضي العناية الحرجة ليكونوا أكثر دراية ومهارة في التعرف وعلاج عدم انتظام ضربات القلب البطيني. ومن ناحية اخري السياسات واللوائح الصادرة من وزارة الصحة العامة و المجلس القومي للمهن الطبية والصحية يحب ان تحافظ علي المستوي الامثل لمعرفة ومهارات الممرضين في التعرف وعلاج عدم انتظام ضربات القلب البطيني، وخصوصاً ممرضي العناية الحرجة.

Abbreviations

Abbreviation	Meaning /Phrase
ACC	Arrhythmia Care Coordinator
ACS	Acute Coronary Syndromes
AED	Automated External Defibrillator
AHA	American Heart Association
BLS	Basic Life Support
AMI	Acute Myocardial Infarction
BNP	B-type Natriuretic Peptide
BUN	Blood Urea Nitrate
CABG	Coronary Artery Bypass Graft
CCU	Coronary Care Unit
CKD	Chronic Kidney Disease
CPD	Continuing Professional Development
CPR	Cardiopulmonary Resuscitation
CRT	Cardiac Resynchronization Therapy
CT	Computed Tomography
ECG	Electrocardiogram
ERC	European Resuscitation Council Guidelines
ES	Electrical Storm
HF	Heart Failure
ICD	Implantable Cardioverter-Defibrillator
ICU	Intensive Care Unit
LV	Left Ventricular
MAR	Medication Administration Record
MI	Myocardial Infarction
NCD	Non Communicable Disease
NSVT	Non-Sustained Ventricular Tachycardia
OHS ICU	Open Heart Surgery Intensive Care Unit
PCI	Percutaneous Coronary Intervention
PVC	Premature Ventricular Complex
PVT	Pulseless Ventricular Tachycardia
RV	Right Ventricular
SCA	Sudden Cardiac Arrest
SCD	Sudden Cardiac Death
SVT	Supraventricular Tachycardia
TOF	Tetralogy Of Fallot
VF	Ventricular Fibrillation
VA	Ventricular Arrhythmia
VT	Ventricular Tachycardia

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CHAPTER ONE

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Introduction

As is well known, as a muscular pump of the circulatory system, the heart is powered by electrical impulses generated by the primary and secondary pace makers in the sino-atrial node (SA node) and atrio-ventricular node (AV node) respectively. The transmission of electrical signals throughout the anatomy of the heart and in particular, the heart's four chambers to enable atrial and ventricular contraction and relaxation is aided by the electrical fibres emanating from the AV node and the Bundle of His, through to the Purkinje fibres in the substance of the heart muscles.

Given the endless nature of the cardiac cycle, an average heart beats approximately 100,000 times a day, pumping some 2000 gallons of blood through the circulation. Over a 70-year life span, that adds up to more than 2.5 billion heartbeats.

This normal flow process can however be disrupted by many forms of heart disease, causing abnormality in the heart rhythm in the form of cardiac arrhythmias. These conditions make the heart less effective, with downstream effects on the blood supply to organs and tissues and cardiac return, the consequences of which include syncopal attacks (fainting) angina pectoris (chest pain), arrythmias and even sudden death. [1] In the present study, ventricular arrhythmias are of particular interest and form the basis for the research work.

Ventricular arrhythmias generally include two main types namely; ventricular tachycardia (VT) and ventricular fibrillation (VF). These are known to be the leading causes of sudden cardiac death (SCD), which in turn represents about half of all cardiovascular mortality and accounts for over 350,000 deaths annually in the United States alone. [2] The world health organization (WHO) in 2012 estimated that in Sudan cardiovascular diseases prevalence is 12% and the probability of dying between ages 30 to 70 years from the main NCD is 17 %. According to the age-standardized death rates per 100,000 the rate in males is 240:100,000 and for females it is 220:100,000 deaths from cardiovascular diseases (WHO, 2012).

Ventricular tachycardia (VT) it is the most common form of complex tachycardia seen in association with structural heart disease. [3] And is associated with myocardial pathologic processes that promote cardiac fibrosis or inflammation, most commonly from coronary artery disease (CAD) in over 80% of patients. [4] However, myocarditis, dilated cardiomyopathy, congenital heart disease, cardiac infiltrative diseases, arrhythmogenic right ventricular cardiomyopathy and hypertrophic cardiomyopathy

are also known to contribute to the problem. Still in about 10% of patients, VT occurs in the absence of structural heart disease.[5] The appearance of VF includes rapid, irregular, undulating waveforms (usually faster than 200 ms) that are more disorganized than polymorphic VT. As VF persists, the fibrillation slows with waveforms also developing decreased amplitude preceding asystole. [6, 7]

In the pharmacological treatment for Ventricular tachycardia/fibrillation (VT/VF), intravenous Amiodarone is often considered the drug of choice in emergency care medicine. [8] The first and immediate choice for Ventricular tachycardia/fibrillation (VT/VF) management is to achieve electrical defibrillation. Electrical defibrillation should be performed for VFib and hemodynamically unstable VT and electrical cardioversion for stable sustained VT either as the first choice or for those who do not respond to antiarrhythmic medications. Generally the recommended energy levels with modern biphasic defibrillators range from 150 to 200 Joules. [9]

Nurses in the emergency department play a vital role in the diagnostic and management process for patients with VT/VF. To make the diagnosis in acute arrhythmia requires recognition and interpretation of important electrocardiogram (ECG) finding and knowledge of resuscitation guidelines by emergency health practitioners. [10] Nurses play a critical role in arrhythmia identification and management at the bedside. Based on the nurse's interpretation of the electrocardiographic (ECG) monitor recording, the nurse may simply gather more data, notify the physician who makes treatment decisions based on the rhythm interpretation of the nurse, or institute pharmacologic and counter shock therapies consistent with unit-specific protocols. Therefore understanding the nurse's perspective of arrhythmia knowledge, and ultimately, developing tools to evaluate this knowledge, and competence in the recognition of ECG rhythms, are of critical importance to emergency nursing. [11]

Justification

Nurses play a key role in the management in hospital patients with cardiac arrest. They are often the first responders on the scene of an arrest initiating cardiopulmonary resuscitation (CPR) as well as summoning assistance from the "advanced life support / cardiac arrest team". Thus it is argued that nurses should be trained and acquires the skills necessary to perform defibrillation when required. Notwithstanding this, the community has an expectation (which is not entirely realistic) that all nurses are able to approximately manage a collapse situation.[13]

Since nurses care for their patients around the clock, they need to have higher levels of knowledge and skill regarding recognition and early management of ventricular arrhythmias (pVT and VF). Therefore familiarity with guidelines to promote the quality and appropriate care for patients should be part of their portfolio and their scope of practice. In recognition of the need to equip nurses adequately to support emergency practice, this study has been designed to assess the effectiveness of introducing a structured teaching program on the knowledge and skills of nurses regarding recognition and management of ventricular arrhythmias (pVT and VF), based on the European Resuscitation Council Standard Guidelines for critical care nurses. The rationale is to enable nurses to identify and manage ventricular arrhythmias skillfully and efficiently with the view to reducing the incidence of deaths due to life threatening arrhythmias.

Problem statement

Cardiac monitoring, once a skill limited only to critical care units, is being used with increasing frequency in a variety of clinical settings. Staff in medical/surgical units, oncology units, progressive care units, labor and delivery units, outpatient surgical units, and ambulatory care areas provides patient support that incorporates cardiac rhythm recognition. Critical thinking skills represent a second aspect of practice that is related to cardiac rhythm interpretation and are important to providing effective patient care. [14]

A study was conducted to assess the level of knowledge and attitude in terms of interpretation and management of arrhythmias among nurses cardiac care unit. Descriptive research design and total of 60 trained staff nurses were selected and assessed the level of knowledge and attitude in regards to interpretation and management of arrhythmias by administering structured questionnaire and attitude rating scale. The study results showed that the nurses were having less knowledge and attitude in interpretation and management of arrhythmias. [15]

Besides all mentioned points the investigator also having Emergency Room (ER), post open-heart surgery intensive care unit (OHS ICU), coronary care unit (CCU) and medical surgical ward working experience knows the importance of accurate identification and proper management of ventricular arrhythmias (pVT& VF). However, there are several technical advancements in assessing and interpreting cardiac arrhythmia nurses play a vital role to take care necessary and immediate action to correct them at right time.

Objectives

General objective:

To study the Impact of an educational program on recognition and management of Ventricular arrhythmias (VF& pVT) guidelines among critical care nurses.

Specific objectives:

1. To assess the nurses knowledge regarding Ventricular arrhythmias (VF& pVT) definition, signs and symptoms, causes, monitoring, diagnostic methods, management, complication, and prevention
2. To assess the nurses skills regarding recognition of Ventricular arrhythmias (VF& pVT) from ECG rhythm strip and Monitor.
3. To design and implement an educational program for nurses regarding recognition and management of Ventricular arrhythmias (VF& pVT) guideline among critical care nurses.
4. To evaluate the efficiency of structured teaching program on the level of Knowledge and skills of recognition, and management of Ventricular arrhythmias (VF& pVT) among critical care nurses.

Research questions:

1. Will familiarity with the guidelines make a difference in nurses' knowledge pre and post the program?
2. Will introducing the program make a difference in nurses' skills and performance?

Hypothesis:

H_1: There will be an improvement in the level of knowledge on recognition, and management of Ventricular arrhythmias (VF& pVT) among critical care nurses post structured teaching program using the European guidelines.

H_2 There will be a significant difference in the level of skills and performance on recognition, and management of Ventricular arrhythmias (VF& pVT) among critical care nurses following introduction of the structured teaching program using the European guidelines.

CHAPTER TWO

❖ *Literature Review*

2. Literature Review

2.1. Definitions*:*

'Lethal' a Greek word means 'causes death'. Arrhythmias which cause death are called lethal cardiac arrhythmias and commonly ventricular tachycardia (VT), ventricular fibrillation (VF) and ventricular asystole fall into this category. An arrhythmia is a disorder of the formation or conduction of the electrical impulse in the heart. These disorders can cause disturbances of the heart rate, the heart rhythm or both. [16] Ventricular fibrillation (VF) is a life-threatening cardiac arrhythmia in which the coordinated contraction of the ventricular myocardium is replaced by high frequency, disorganized excitation, resulting in failure of the heart to pump blood. VF is the most commonly identified arrhythmia in cardiac arrest patients characterized by rapid grossly irregular electrical activity with marked variability in electrocardiographic waveform and a ventricular rate usually above 300 bpm (cycle length: <200 MS). [17] VT refers to any rhythm faster than 100 (or 120) beats/min, with three or more irregular beats in a row, arising distal to the bundle of His. The rhythm may arise from working ventricular myocardium, the distal conduction system, or both. Ventricular tachycardia (VT) or ventricular fibrillation (VF) causes most of the sudden cardiac deaths in the United States. VF usually ends in death within minutes unless prompt corrective measures are instituted. Sudden arrhythmic death refers to hemodynamically unstable (VT) or (VF), and accounts for the majority of all episodes of sudden cardiac death (SCD). [18, 19]

2.2. Cardiac Anatomy

The heart is an electromechanical pump, which has four chambers: two atria and two ventricles. Atria receive the blood returned back to the heart, and ventricles eject blood into arteries. As well as atria and ventricles, the heart also divided into two as pumps: right and left pumps. The muscular wall that separates those pumps is called the septum. In normal adult heart, the septum prevents mixture of the blood in the right and left of the heart. Total heart size is approximately the size of a fist and its weight is around 300 grams. Heart beats approximately 100.000 times a day and pumps around 7200 liters per day. For an adult, at rest nearly 5 liters of blood is pumped out of the heart per minute and it takes about 1 minute for blood to pump out of the heart to the body and back through the superior or inferior vena cava to the heart again. This

pumping action is enabled by muscular activity, which is necessary to generate the required pressure to push blood to whole body. [20]

2.3. Cardiac Electrophysiology

The heart is acts as a biomechanical pump. Electrical impulses are generated in specialized cells and flow through the heart myocardium by the ion changes on the cell membrane which is the beginning of both the electrical and the mechanical activity. Both the electrical and the mechanical states of the organ will directly affect the pumping activity. The cardiac conduction system is the component that initiates and coordinates the heart's electrical activities and is vital for maintenance of cardiac stability. An important property of most cardiac cells is that they are electrically excitable and linked to each other. This allows a signal to spread from cell to cell in a chain reactive manner. The normal rhythmical heartbeat is called the sinus rhythm. Cardiac contraction is normally started from the sinoatrial node (SA node), which is therefore called the primary pacemaker. [21]

The sinoatrial node is found in the upper part of the right atrium near to the junction with the superior vena cava.[12] The electrical signal generated by the SA node travels through the right atrium in a radial way that is not completely understood. It travels to the left atrium via Bachmann's bundle, such that the muscles of the left and right atria contract together.[22, 23, 24] The signal then travels to the atrioventricular node. This is found at the bottom of the right atrium in the atrioventricular septum—the boundary between the right atrium and the left ventricle. The septum is part of the cardiac skeleton, tissue within the heart that the electrical signal cannot pass through, which forces the signal to pass through the atrioventricular node only.[25] The signal then travels along the bundle of His to left and right bundle branches through to the ventricles of the heart. In the ventricles, the signal is carried by specialized tissue called the Purkinje fibers , which then transmit the electric charge to the heart muscle.[26]

2.4. Cellular Mechanisms:

The mechanisms of ventricular tachyarrhythmias which include ventricular tachycardia and ventricular fibrillation, are enhanced normal automaticity, abnormal automaticity, triggered activity induced early or late after depolarization and re-entry [27, 28, 29].

2.4.1. Automaticity:

Normal automaticity results from phase 4 spontaneous depolarization of the transmembrane action potential arising from a normal resting potential, reaching

threshold and initiating an action potential [27, 29]. An initiating current (If) is responsible for spontaneous phase 4 depolarization in the sinus node. The rate is determined by the integration of the maximum diastolic potential at the end of repolarization, the slope of phase 4 depolarization, and the threshold potential. In contrast, abnormal automaticity arises from a partially depolarized membrane potential that is usually close to the activation potential for calcium channels in the cell membrane [27, 29].

2.4.2. Triggered Activity:

Early after depolarizations occur during late phase 2 or early phase 3 of the action potential [29, 30, 31], usually in the setting of action potential prolongation due to an increase in inward currents (the late sodium current, the inward calcium current or the sodium calcium exchange current) or a decrease in repolarizing potassium currents.

Delayed after depolarizations occur after complete membrane repolarization and develop under conditions of intracellular calcium overload. Factors contributing to elevated intracellular calcium load include tachycardia, catecholamines, hypokalemia, digoxin toxicity, cardiac hypertrophy, and HF [32, 33].

2.4.3. Reentry:

Reentry is the underlying mechanism for most sustained VA in the presence of structural heart disease [27- 29, 34-36]. Reentry may occur around a fixed anatomical obstacle, such as scar after an MI or surgically repaired congenital heart disease. In this setting, an excitable gap separates the excitation wave front from its tail of refractoriness. The existence of structural reentrant substrates provides the rationale for VT ablation in scar related VTs [35, 36].

Functional reentry around areas of functional block without anatomical obstacles can also occur. Two main models of functional reentry have been proposed [28, 29]. The leading circle model has a functionally refractory core and no excitable gap. Spiral wave reentry is driven by a rotor with a curved wave front and wave tail pivoting around an excitable but unexcited core. There remains much debate about the precise mechanism(s) of VF (rotor versus multiple wavelet reentry). Both mechanisms may be operational in different phases of VF [34].

2.5. Types of ventricular tachyarrhythmia:

Ventricular tachyarrhythmia can be grossly categorized based on electrocardiogram into two main morphologies:

I. Ventricular Tachycardia (VT)

II. Ventricular Fibrillation (VF).

Each of these is due to a pathophysiologic mechanism, in which a substrate is affected by a triggering event.

2.5.1. The **ventricular tachycardia** (VT) refers to any rhythm faster than 100 (or 120) beats/min arising distal to the bundle of His [18], have many types such as below classifications:

1. According to the duration (with a generally accepted cutoff of 30 seconds), VT classified to:

- Sustained (lasting >30 s).
- Non-sustained (lasting <30 s).

2. Sustained VT is a potentially life-threatening arrhythmia, which occurs in three major forms in the electrocardiographic (ECG) morphologies[19]

- Monomorphic VT, if the QRS complex remains identical from beat to beat, as occurs when VT originates from a single focus or circuit (usually "macro re-entrant" and scar-related).
- Polymorphic VT, if the QRS morphology changes from beat to beat (usually ischemia-related).
- Bidirectional VT, which displays a beat-to-beat alternans in the QRS morphology and/or axis. [37]

In **monomorphic VT**, the ventricular activation morphology is the same on a beat-to-beat basis, and most commonly is a reentrant electrical wave front around a fixed obstacle such as myocardial scar. Specific locations within the ventricles have associated morphologies of ventricular tachyarrhythmias seen on electrocardiogram [39]. Within or at the border of these scar zones, slow conduction provides the necessary construct for VT to sustain itself [40]. Among episodes of ES, monomorphic VT comprises 77% of the cases [41].

Another form of monomorphic VT involves triggered activity, usually in structurally normal hearts [42]. These episodes of VT are usually self-limited, and uncommonly cause ES. Re-entry involving the His-Purkinje system in patients with cardiomyopathy

or conduction system disease can result in bundle-branch reentrant tachycardia, usually with left bundle branch block morphology [43]. Another less common monomorphic VT is **ventricular flutter**, which is quite rapid with a cycle length of approximately 200 ms [44].

In **Polymorphic ventricular** tachycardia on a beat-to-beat basis, polymorphic VT has varying amplitude and/or duration of the QRS complex, and this type of ventricular activation includes **torsades de pointes**. Polymorphic VT can occur in patients with normal and prolonged QT intervals during sinus rhythm [45]. Among ES cases, polymorphic VT comprises 7% of cases [41].

Polymorphic VT occurring with a normal QT interval usually involves ischemic heart disease or non-ischemic cardiomyopathy. During acute myocardial infarctions, 2 to 4% of patients develop polymorphic VT, but this arrhythmia is more common with coronary vasospasm [46]. In non-ischemic cases, hypertrophic cardiomyopathy and acute myocarditis can present with polymorphic VT [45]. In addition, catecholaminergic polymorphic VT may present with polymorphic VT or bidirectional tachycardia with alternating QRS morphologies [47].

A specific subtype of ventricular tachyarrhythmias that should be mentioned is **bidirectional VT**, which displays a beat-to-beat alternans in the QRS morphology and/or axis, most notable in the frontal plane leads. While commonly associated as one of the arrhythmia manifestations of digitalis toxicity, bidirectional VT can also be seen in catecholaminergic VT [37].

3. According to the pulse (absent or present), VT classified to:

- Pulse less VT (pVT)
- VT with Pulse.

4. Finally, the last classification can be made based on the substrate and the location of the earliest activation.

2.5.2. The appearance of **Ventricular Fibrillation** includes rapid, irregular, undulating waveforms (usually faster than 200 ms) that are more disorganized than polymorphic VT. As VF persists, the fibrillation slows with waveforms also developing decreased amplitude preceding asystole [48, 49]. After approximately 15 minutes, asystole is reached, possibly because of depletion of the heart's energy reserves.

On the electrocardiogram (ECG), VF manifests as a chaotically irregular pattern. This pattern is coarse initially but becomes finer as ventricular disorganization increases. Based on that VF classified as:

- Coarse VF
- Fine VF.

2.6. Epidemiology and incidence rate:

In 2008, 36 million (63%) of global deaths were attributed to NCDs, principally cardiovascular diseases, diabetes, cancer and chronic respiratory diseases [50]. Nearly 80% of these NCD deaths (29 million) occurred in low- and middle-income countries. NCDs are the most frequent causes of death in most countries in the Americas, the Eastern Mediterranean, Europe, South-East Asia, and the Western Pacific. About 33,380 global deaths per year are due to cardiac arrhythmias with the weighted average of 3158.1 deaths. Prevalence of arrhythmias was 53 per 1,000 in 2005 [51]. Based on WHO report for 2012 about Sudan, the probability of dying between ages 30 to 70 years from the main NCD is 17 %. Cardiovascular diseases percentage is 12%, and according to the age-standardized death rates per 100,000 the male rate is 240: 100.000 and female rate is 220:100.000 deaths due to cardiovascular diseases. [52]

The 2017 update of cardiovascular statistics from the American Heart Association (AHA) estimated the total annual burden of out-of- hospital cardiac arrest at 356,500 [53]. An additional 209,000 in-hospital cardiac arrests occur annually [54]. Among the out-of-hospital cardiac arrest group, approximately 357,000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age.

Ventricular arrhythmias, including ventricular tachycardia (VT) and ventricular fibrillation (VF), are the leading cause of sudden cardiac death (SCD), which in turn represents about half of all cardiovascular mortality and accounts for over 350,000 deaths annually in the United States [55]. Sustained VT and VF are a potentially life-threatening arrhythmia. Sudden arrhythmic death refers to hemodynamically unstable VT or ventricular fibrillation (VF), and accounts for the majority of all episodes of sudden cardiac death (SCD), a diagnosis that includes hemodynamically unstable VT, VF, asystole, and non-arrhythmic cardiac causes. Sudden cardiac death is responsible for an estimated 184,000 – 462,000 American deaths yearly, and is a major cause of mortality in the elderly. [19]

Approximately half of patients with out-of-hospital cardiac arrest with the first rhythm identified as VF and who survive to hospital admission have evidence of acute MI (AMI). Of all out-of-hospital cardiac arrests, >50% will have significant coronary artery lesions on acute coronary angiography [56]. Of patients hospitalized with AMI, 5% to 10% have VF or sustained VT prior to hospital presentation, and another 5% will have VF or sustained VT after hospital arrival, most within 48 hours of admission. A study of patients with non–ST-elevation ACS who underwent cardiac catheterization within 48 hours found VT/VF in 7.6% of patients, with 60% of those events within 48 hours of admission [57].

Sustained VA that occurs in the setting of an ACS is more often polymorphic VT or VF than monomorphic VT. Risk factors for VT/VF include prior history of hypertension, prior MI, ST-segment changes at presentation, and chronic obstructive pulmonary disease [58].

The Framingham Heart Study compared the incidence of SCD in women and men across all age groups (n=5209), aged 30 – 62 years at study entry. Women comprised just over half of the study population (n=2873, 55%), with a peak incidence of SCD which lagged that of men by > 10 years. Congestive heart failure increased the risk of SCD by 5-fold in women and 16-fold in men. The incidence of SCD progressively increased with age, especially after age 74 years, reflecting the high prevalence of structural heart disease in the elderly. [59]

Aging is associated with a myriad of changes in the cardiac conduction system, some of which manifest in association with cardiovascular disease, and others develop as part of normal aging. These changes include sinus node dysfunction, slowing of AV nodal conduction, left axis deviation, bundle branch blocks, and an increased prevalence of both supraventricular and ventricular premature beats and arrhythmias. LBBB, AF, and sustained VT are particularly predictive of future adverse cardiac events, and frequently herald the presence of underlying cardiovascular disease. The prognostic significance of any given conduction abnormality or rhythm disturbance is dependent primarily on the presence and severity of any accompanying cardiac disease. Gaining an appreciation of the epidemiology of cardiac conduction disorders and arrhythmias in the elderly will assist the practitioner in differentiating ECG findings that represent normal aging from those suggesting a disease process requiring further evaluation.[60]

Circadian rhythm may play a role. There is also a preponderance of ES during winter months (December, January, and February) and late afternoon similar to other data for myocardial infarction and sudden cardiac death.[61]

2.7. Pathophysiology:

Ventricular arrhythmias occur in a variety of clinical situations but are most often associated with coronary artery disease (CAD). VF can result from acute myocardial infarction (MI) or ischemia or from myocardial scarring from an old infarct. [62]Ventricular tachycardia (VT) may degenerate into VF. Intracellular calcium accumulation, the action of free radicals, metabolic alterations, and autonomic modulation are important influences on the development of VF during ischemia.

Initiation of ventricular arrhythmias can occur in several ways. For example, if the myocardium is stimulated by a ventricular premature complex during the ascending limb of the T wave [63] the impulse can propagate erratically through the variably refractory myocardial cells and establish reentrant patterns that result in chaotic ventricular depolarization. Consequently, coordinated myocardial contraction becomes disrupted.

The reentrant patterns break up into multiple smaller wavelets and the level of disorganization increases, with reentrant circuits producing high-frequency activation of cardiac muscle fibers. As the heart loses its ability to pump blood, myocardial ischemia worsens and a self-perpetuating vicious cycle ensues, leading to death if not corrected.

On electrocardiogram (ECG), VF manifests as a chaotically irregular pattern. This pattern is coarse initially but becomes finer as ventricular disorganization increases. As the ECG waveform flattens, the likelihood of successful defibrillation decreases. [64]

At the cellular level, VT is caused by electrical reentry or abnormal automaticity. Myocardial scarring from any process increases the likelihood of electrical reentrant circuits. These circuits generally include a zone where normal electrical propagation is slowed by scar. Ventricular scar formation from a prior myocardial infarction (MI) is the most common cause of sustained monomorphic VT.

VT in a structurally normal heart typically results from mechanisms such as triggered activity and enhanced automaticity. Torsades de pointes, seen in the long QT syndromes, is likely a combination of triggered activity and ventricular reentry.[65]

During VT, cardiac output is reduced as a consequence of decreased ventricular filling from the rapid heart rate and lack of properly timed or coordinated atrial contraction.

Ischemia and mitral insufficiency [66] may also contribute to decreased ventricular stroke output and hemodynamic intolerance.

Hemodynamic collapse is more likely when underlying left ventricular dysfunction is present or when heart rates are very rapid. Diminished cardiac output may result in diminished myocardial perfusion, worsening inotropic response, and degeneration to ventricular fibrillation (VF), resulting in sudden death.

When documentation of the antecedent rhythm is available, it often shows that rapid VT precedes VF. In patients with chronic ischemic heart disease, monomorphic VT arising from a reentrant focus is the most common precursor to VF. Other factors associated with increased risk of VF include frequent PVCs, particularly complex forms (such as multiform PVCs) and ones with short coupling intervals (R-on-T phenomenon).[67]

Idiopathic VF is triggered by ventricular premature beats that may originate in the distal Purkinje conducting system, LV septum, anterior right ventricle, or RV outflow tract (RVOT). Early repolarization, or J wave (elevation at the junction of between the QRS complex and ST-segment), has been identified in patients with idiopathic VF and has been associated with mutations in a variety of ion channel genes.[68] Catheter ablation that targets triggers of ventricular premature beats can provide long-term freedom from recurrence of idiopathic VF.[69]

2.8. Etiology:

The most common ventricular fibrillation and ventricular tachycardia etiology include the following:

2.8.1. Cardiac causes with structural heart disease include the following:

2.8.1.1. Acute and chronic ischemic heart disease:

Coronary artery disease (CAD) is the single most common etiologic factor predisposing patients to ventricular arrhythmia. In survivors of cardiac arrest, CAD with greater than 75% stenosis is observed in 40-86% of patients, depending on the age and sex of the population studied. In postmortem studies of people who have died from VF, extensive atherosclerosis is the most common pathologic finding. Cardiac arrest attributable to ventricular arrhythmias may occur with acute ischemia or in the absence of an acute disturbance of coronary flow, due to scarring from a previous MI. An infarct scar can serve as the focus for reentrant ventricular tachyarrhythmias, which may occur shortly after the infarct or years later. Many studies support the relationship of symptomatic and

asymptomatic ischemia as markers of myocardium at risk for arrhythmias.[70, 71]

When documentation of the antecedent rhythm is available, it often shows that rapid VT precedes VF. In patients with chronic ischemic heart disease, monomorphic VT arising from a reentrant focus is the most common precursor to VF. Other factors associated with increased risk of VF include frequent PVCs, particularly complex forms (such as multiform PVCs) and ones with short coupling intervals (R-on-T phenomenon). [67]

2.8.1.2.Non-atherosclerotic coronary artery abnormalities such as include:

Congenital lesions, Embolism, Arteritis, and Mechanical abnormalities, such as coronary artery aneurysms. [17, 18]

2.8.1.3. Non-ischemic cardiomyopathies

Dilated cardiomyopathy ,Hypertrophic cardiomyopathy, and Arrhythmogenic right ventricular cardiomyopathy/dysplasia.[17, 18]

2.8.1.4. Structural heart diseases include the following:

Tetralogy of Fallot, Transposition of the great arteries, Physiologic single ventricle, Marfan syndrome, Eisenmenger syndrome, Congenital heart block, Pericardial tamponade, Myocarditis, Valvular lesions (e.g. Aortic stenosis, Aortic dissection), and Ebstein anomaly .[17, 18]

2.8.1.5. Cardiac causes non-structural abnormalities

These generally are a group of abnormalities in which patients have no apparent structural heart disease but have a primary electrophysiologic abnormality that predisposes them to VT or VF.[72] Nonstructural heart disease include the following:

Idiopathic ventricular arrhythmias, Mechanical (commotio cordis) or electrical accidents, Catecholaminergic polymorphic VT (CPVT), Familial SCD of uncertain cause, Preexcitation including Wolff-Parkinson-White (WPW) syndrome, Primary VT and VF, Primary pulmonary hypertension, Heart block, Channelopathies (long& short QT syndrome, or Brugada syndrome, and Drug-induced QT prolongation with torsades de pointes.[17, 18]

2.8.2. Non-cardiac respiratory causes include the following:

Pulmonary embolism, Tension pneumothorax, Primary pulmonary hypertension, Sleep apnea, Bronchospasm, Aspiration, and Hypoxia. [17, 18]

2.8.3. Metabolic or toxic causes include the following:

Electrolyte imbalances (e.g., hypokalemia, hypocalcaemia, & hypomagnesaemia), Antiarrhythmic Medications or drug ingestion, toxicity,Acidosis, Sepsis, and Environmental poisoning. [17, 18]

2.8.4. Neurologic causes include the following:

Seizures, Cerebrovascular accident - Intracranial hemorrhage or ischemic stroke and Drowning. [17, 18]

2.9. Risk Factors:

The risk of SCA is increased 6- to 10-fold in the presence of clinically recognized heart disease, and two- to four-fold in the presence of coronary heart disease (CHD) risk factors [73, 74]. A number of clinical characteristics and other factors are associated with an increased risk of SCA among persons without prior clinically recognized heart disease [75].

Most risk factors for CHD are also risk factors for SCA and ventricular arrhythmia. Risk factors that relate to coronary artery disease (CAD) and to subsequent myocardial infarction (MI) and ischemic cardiomyopathy are also important which is including family history of premature CAD, Adults past the age of 60 years, Cigarette smoking, Dyslipidemia, Hypertension, Diabetes, Obesity, Sedentary Lifestyle, Increase level of CRP, Excess alcohol intake, Psychosocial factors, Caffeine, Fatty acids, Previous cardiac arrest, Syncope or near-syncope, Prior MI, especially within 6 months, LV ejection fraction less than 30-35%, History of frequent ventricular ectopy, Renal or liver failure. [76, 77]

2.10. Signs and symptoms:

The most common ventricular fibrillation and ventricular tachycardia signs and symptoms are include; Chest pain or discomfort, Palpitation, Syncope, Anxiety, Agitation, Lethargy, Coma, Light-headedness, Shortness of Breathing (SOB), Dyspnea, Nausea, Cold sweats, Altered mental status, Diaphoresis, Hypotension, Lightheadedness, Hemodynamic Compromise or collapse, Pulseless (No Pulse), Hypotension or non-recordable BP, Tachypnea or apnea, Signs of diminished perfusion, including a diminished level of consciousness, pallor, diaphoresis, High jugular venous pressure, Cannon a waves (if the atria are in sinus rhythm), Variation in intensity of first heart sound, caused by loss of atrioventricular (AV) synchrony. [17, 18, 75]

2.11. Diagnostic methods & investigations:

2.11.1. Noninvasive Evaluation

2.11.1.1. Electrocardiography (ECG):

2.11.1.1.1. 12-lead ECG and Exercise Testing:

A 12-lead ECG during tachycardia is the first diagnostic test that should be done in any patient found to be in a stable wide QRS complex tachycardia on a monitor. VT is the diagnosis in most adults with wide complex tachycardia and underlying structural heart disease [78]. Criteria that support a diagnosis of VT include AV dissociation, a QRS complex >0.14 s, monophasic R wave in aVR, specific QRS morphologies (e.g., positively or negatively concordant QRS complexes in the precordial leads), the absence of an RS complex in all precordial leads and an RS interval >100 ms in at least 1 precordial lead [79]. Exceptions occur, particularly in patients with advanced heart disease and with the use of certain antiarrhythmic medications [80]. For patients with preexisting bundle branch block, comparison of the QRS morphology during sinus rhythm with that during wide complex tachycardia is often relevant.

For exertion-related arrhythmic symptoms, exercise in a monitored setting may reproduce the symptoms and/or the related arrhythmia, allowing for diagnosis.

2.11.1.1.2. Exercise testing:

Is particularly important when catecholaminergic polymorphic ventricular tachycardia is a possibility. However, exertion-related symptoms and findings may not be reliably reproducible with exercise testing, and long-term electrocardiographic monitoring with external or implantable recorders may be necessary.

The presence of ventricular fibrillation (VF) can be confirmed only with an electrocardiogram (ECG). In addition, an ECG is indicated in all patients who have experienced VF, as it may provide evidence of an underlying condition that led to the episode.

Electrocardiography (ECG) is the criterion standard for the diagnosis of VT. If the clinical situation permits, a 12-lead ECG should be obtained before conversion of the rhythm. In a patient who is hemodynamically unstable or unconscious, however, the diagnosis of VT is made from the physical findings and ECG rhythm strip only.

Normal Cardiac Rhythm Parameters:

- NSR HR: 60 bpm–100 bpm
- PR interval: 0.12–0.20 sec

  (Unable to determine; consider atrial arrhythmia, junctional arrhythmia; examine QRS to determine if ventricular arrhythmia)
- P wave: Generally round (Saw toothed → Consider atrial flutter)
- QRS: 0.06–0.10 sec (Wide, bizarre; consider PVC, VT)

Ventricular Tachycardia (VT) Parameters:

Three or more PVCs together with the same shape and amplitude, unstable rhythm, and easily progresses to VF if VT sustained & untreated. Patient may or may not have a pulse, no BP.

■ Atrial rate: Unable to determine; no P waves; no PR interval

■ Ventricular rate: 100–250 bpm

■ Rhythm: Usually regular

■ QRS: Wide & bizarre, >0.10 sec

Ventricular Fibrillation (VF):

Chaotic pattern. No effective ventricular contraction. No C.O., no pulse, no BP. Brain death occurs within 4–6 min, if untreated.

■ Atrial rate: Unable to determine; no P waves; no PR interval

■ Ventricular rate: Fibrillatory waves with no pattern

■ Rhythm: Irregular

The ECG should be repeated once sinus rhythm has been restored, or when prior VT, or VF is suspected, as in a patient who experienced syncope. The ECG may also provide clues for differentiating among potential arrhythmia mechanisms or causes of VT, or VF.

Nurses should have a working knowledgeable of electrocardiography (ECG) especially those working in critical care units. This allows them to distinguish the normal ECG rhythm from abnormal patterns such as life-threating arrhythmias. A number of studies have been conducted to assess nurses' knowledge regarding ECG.

An exploratory study was conducted to assess the recognition of telemetric electrocardiogram diagnostic skills among nurses. The form of a ten-slide presentation of six second rhythm strips was administered among 25 emergency department nurses, 23 intensive care unit nurses, 34 cardiac technicians and 37 physicians to make interpretations. All the samples' interpretations were reviewed and it was found that the

ICU nurses had the best performance overall compared to the other participants. ICU nurses also had less difficulty in identifying and treating various kinds' arrhythmias. [81]

In another study, clinical skills conducted in the UK, competency in cardiac rhythm monitoring was found to be beneficial in identifying changes in cardiac status, assessment of response to treatment, diagnosis and post-surgical monitoring. The study concluded that the first line assessment assists health care practitioners to provide care to their patients. [82]

A descriptive study was conducted to assess nurses' own perception of their knowledge of arrhythmias using a qualitative research design in the form of focus groups. The subjects were critical care nurses who work in an acute care setting where they read electrocardiography data and make treatment decisions. Five focus groups were conducted over a period of twelve months. Group size ranged from 4 - 8 participants. Participants were asked to describe their perception of arrhythmia knowledge and to assign a rating score related to the level of knowledge needed to identify specific arrhythmias. This study revealed a deficit in nurses' ability to recognize and identify specific arrhythmias including heart block, aberrant conduction, and tachyarrhythmias. Understanding of lead placement concepts varied greatly among the participants. It was concluded that insight and perspective of critical care nurses relating to the level of arrhythmia knowledge are needed for the development of competency measures and evidence –based teaching strategies. [83]

In patients with VA symptoms associated with exertion, suspected ischemic heart disease, or catecholaminergic polymorphic ventricular tachycardia, exercise treadmill testing is useful to assess for exercise-induced VA [84, 85]

In patients with suspected or documented VA, a 12-lead ECG should be obtained in sinus rhythm to look for evidence of heart disease [86].

2.11.1.1.3. Ambulatory Electrocardiography:

Ambulatory electrocardiographic monitoring is often used to assess the effectiveness of treatments to suppress arrhythmias, but more robust data are needed on the clinical use of this practice. Continuous or intermittent ambulatory electrocardiographic recording with a Holter monitor or an event recorder is helpful in diagnosing suspected arrhythmias, establishing their frequency, relating them to symptoms, and assessing the response to therapy. Although the yield of these tests is

relatively low, VT is occasionally documented [87]. A 24-hour continuous Holter recording is appropriate when symptoms occur at least once a day or when quantitation of PVCs/ NSVT is desired to assess possible VA-related depressed ventricular function. For sporadic symptoms, event or "looping" monitors are more appropriate because they can be activated over extended periods of time and increase diagnostic yield [88, 89]. Adhesive patch electrocardiographic monitors can record for weeks and allow for continuous short-term 1-lead monitoring and patient activation for symptoms. Studies have shown satisfactory patient compliance, and arrhythmia detection; however, with some monitors, detected arrhythmias are not discovered until the patch is returned for analysis [87, 90].

Ambulatory electrocardiographic monitoring is useful to evaluate whether symptoms, including palpitations, presyncope, or syncope, are caused by VA [87].

2.11.1.1.4. Implanted Cardiac Monitors

Implanted cardiac monitors provide continuous rhythm monitoring and stored recordings of electrograms based on patient activation or preset parameters, allowing a prolonged monitoring period of a few years. These devices require a minor invasive procedure with local anesthesia for implantation. In patients with sporadic symptoms, including syncope, implantable recorders are useful in diagnosing serious tachyarrhythmias (including VA) and bradyarrhythmias [91, 92]. They are generally reserved for patients in whom other ambulatory monitoring is nonrevealing due to the infrequency of events. A 25% added yield in diagnosis has been described after an unrevealing external ambulatory monitor [89]. In a study of patients with syncope, the implantable monitor had a greater diagnostic yield than "conventional" testing with external monitoring, tilt table testing and electrophysiological study [93]. A systematic review in patients with syncope concluded that use of these devices provide a higher rate of diagnosis and a trend toward reduction in syncope relapse after diagnosis, as compared with conventional management [93]. A prospective study of patients after MI, with LVEF <40%, demonstrated NSVT (>16 beats long) in 13%, VT (>30 s) in 3% and VF in 3% of patients [94]. It is important to accurately correlate the symptoms with the arrhythmias detected by implanted cardiac monitors.

In patients with sporadic symptoms (including syncope) suspected to be related to VA, implanted cardiac monitors can be useful [89- 94].

2.11.1.2. Echocardiography

Assessment of global and regional myocardial function, valvular structure and function, along with assessment for adult congenital heart disease is required in patients with or at high risk for VA or SCD, including patients with cardiomyopathy, HF, prior MI, family history of cardiomyopathy or SCD, or an inherited structural heart disease associated with SCD. Echocardiography is the most readily available and commonly used imaging technique, and it recommended being use in patients with known or suspected VA that may be associated with underlying structural heart disease or a risk of SCA, echocardiography is recommended for evaluation of cardiac structure and function [95, 96].

2.11.1.3. Cardiac CT and cardiac MRI:

In patients, presenting with VA who are suspected of having structural heart disease, cardiac magnetic resonance imaging (MRI) or computed tomography (CT) can be useful to detect and characterize underlying structural heart disease. Cardiac CT and cardiac MRI allow for evaluation of structural heart disease and assessment of LV and RV function including quantification of LVEF, LV mass and volume, valvular structure and coronary anatomy including anomalous coronary origins. Cardiac MRI can be useful in the evaluation for myocardial scar and infiltrative processes evident as late gadolinium enhancement [97- 101]. Cardiac MRI also provides high quality assessment of LV and RV function, size, and degree of fibrosis and is particularly useful in arrhythmogenic right ventricular cardiomyopathy and HCM.

In SCD-HeFT (the Sudden Cardiac Death in Heart Failure Trial) [102], the benefit of the ICD was not dependent on the modality (i.e., echocardiography, radionuclide angiography, or contrast angiograms) by which the LVEF was assessed. In clinical practice, if cardiac CT [103] or cardiac MRI has been performed and provides sufficient evaluation, echocardiography may be unnecessary. This recommendation for imaging differs from that of the 2017 ACC/AHA/HRS syncope guideline [104] that applies to patients who may not have VA.

2.11.1.4. Genetic Considerations:

In patients and family members in whom genetic testing for risk stratification for SCA or SCD is recommended, genetic counseling is beneficial. In young patients (<40 years of age) without structural heart disease who have unexplained cardiac arrest, unexplained near drowning, or recurrent exertional syncope, genetic testing may be important to identify an inherited arrhythmia syndrome as a likely cause [105- 111]

The decision to proceed with genetic testing requires discussion regarding the clinical use of genetic information to be obtained for both the proband and family members, as well as consideration of the important psychological, financial, employment, disability, and life insurance implications of positive genotyping [105, 106, 108, 112]. Balancing privacy of health care information for the proband with the "right to know" for family members, and the ability to provide appropriate communication of information to all potentially affected family members can be challenging on many levels, including family dynamics, geographic proximity, and access to health care [113]. For these reasons, genetic counseling generally occurs before proceeding with genetic testing, and, from a patient's perspective, is optimally provided by genetic counselors, if available, in collaboration with physicians [114, 115]. A combined approach of genetic counseling with medical guidance may appropriately balance the decision as to whether genetic testing would be beneficial on an individual basis.

2.11.2. Invasive evaluation of patients with suspected or known ventricular arrhythmias

2.11.2.1. Coronary angiography

The Recommendations from European Society of Cardiology (ESC) regarding angiography are Coronary angiography should be considered to establish or exclude significant obstructive CAD in patients with life-threatening VAs or in survivors of SCD who have an intermediate or greater probability of having CAD by age and symptoms. [116] Coronary angiography plays an important diagnostic role in establishing or excluding the presence of significant obstructive CAD in patients with life-threatening VA or in survivors of SCD. Urgent coronary angiography followed, when indicated, by revascularization is recommended in patients with recurrent VT or VF when myocardial ischemia cannot be excluded. In addition, Urgent angiography is recommended in patients symptomatic for high-degree AV block who have not received reperfusion [117, 118].

2.11.2.2. Electrophysiological (EP) studies.

In patients with ischemic cardiomyopathy, NICM, or adult congenital heart disease who have syncope or other VA symptoms and who do not meet indications for a primary prevention ICD, an electrophysiological study can be useful for assessing the risk of sustained VT [119- 125].

A study of electrophysiological testing in patients with symptomatic NICM found inducible VT/VF in 28% of patients which was associated with a higher rate of ICD

events during follow-up [126]. In a prospective cohort of 180 patients with ischemic or NICM and syncope, induction of VT or VF at electrophysiological study correlated with cardiac mortality only in patients with ischemic heart disease. In patients with NICM, cardiac mortality correlated with LVEF but not with inducibility on electrophysiological study [127]. An electrophysiological study may be helpful, however, in selected patients suspected to have preexcitation or supraventricular arrhythmias as the cause of symptoms or wide complex tachycardias that warrant definitive diagnosis and management. SVT leading to VT/VF or aberrantly conducted SVT may also be suspected in younger patients or those with a preserved LVEF. Induction of SVT and ablation may then be curative, with no need for an ICD. In such cases, failure to induce VT/VF after elimination of the substrate for SVT would be expected.

Risk stratification for channelopathies is generally made on the basis of symptoms, the ECG [128, 129-134], exercise treadmill testing [135, 136], and the results of genetic testing [137, 138]. The electrophysiological study (i.e., programmed ventricular stimulation) does not have prognostic value for risk stratification in patients with these cardiac channelopathies [139- 142].

2.11.2.3. Appropriate laboratory studies may include:

- Serum electrolyte levels, including serum calcium, magnesium, and phosphate levels. Ionised calcium levels are preferred over total serum calcium. Hypokalaemia, hypomagnesaemia, and hypocalcaemia may predispose patients either to conventional VT or torsades de pointes.[17, 18]
- Cardiac enzymes (eg, creatine kinase, myoglobin, troponin)
- Complete blood count (CBC) to detect contributing anemia
- Arterial Blood Gases (ABGs) to assess degree of acidosis or hypoxemia
- Quantitative drug levels (eg, quinidine, procainamide, tricyclic antidepressants, and digoxin)
- Toxicology screens and levels as clinically indicated
- Thyroid-stimulating hormone
- B-type natriuretic peptide (BNP)

Elevated levels of natriuretic peptides—B-type natriuretic peptide (BNP) or N-terminal pro-BNP—are associated with increased risk of SCA and appropriate ICD therapies, even after adjustment of LVEF and other risk factors [143, 144]. These biomarkers are also predictive of nonsudden cardiovascular mortality and thus are not specific to SCD

risk alone. Natriuretic peptides have also been evaluated for predicting SCD in the general population [145- 146].In the Nurses' Health Study, an elevated N-terminal pro-BNP was an independent risk marker for SCD in presumably healthy women [145].

2.12. General Evaluation of Patients With (pVT & VF):

2.12.1. History:[147]

2.12.1.1. Personal clinical history

1. Symptoms/events related to arrhythmia: Palpitations, lightheadedness, syncope, dyspnea, chest pain, cardiac arrest
2. Symptoms related to underlying heart disease: Dyspnea at rest or on exertion, orthopnea, paroxysmal nocturnal dyspnea, chest pain, and edema.
3. Precipitating factors: Exercise, emotional stress
4. Known heart disease: Coronary, valvular (e.g., mitral valve prolapse), congenital heart disease, other
5. Risk factors for heart disease: Hypertension, diabetes mellitus, hyperlipidemia, and smoking
6. Medications:
    - Antiarrhythmic medications
    - Other medications with potential for QT prolongation and torsades de pointes
    - Medications with potential to provoke or aggravate VA
        a. Stimulants including cocaine and amphetamines
        b. Supplements including anabolic steroids
    - Medication-medication interaction that could cause QT prolongation and torsades de pointes
7. Past medical history:
    - Thyroid disease
    - Acute kidney injury, chronic kidney disease, or electrolyte abnormalities
    - Stroke or embolic events
    - Lung disease
    - Epilepsy (arrhythmic syncope can be misdiagnosed as epilepsy)
    - Alcohol or illicit drug use
    - Use of over-the-counter medications that could cause QT prolongation and torsades de pointes

- Unexplained motor vehicle crashes

2.12.1.2. Family History

1. SCD, SCA, or unexplained drowning in a first-degree relative
2. SIDS or repetitive spontaneous pregnancy losses given their potential association with cardiac channelopathies
3. Heart disease
   - IHD
   - Cardiomyopathy: Hypertrophic, dilated, ARVC
   - Congenital heart disease
   - Cardiac channelopathies: Long QT, Brugada, Short QT, CPVT
   - Arrhythmias
   - Conduction disorders, pacemakers/ICDs
4. Neuromuscular disease associated with cardiomyopathies
   - Muscular dystrophy
5. Epilepsy

2.12.2. Physical Examination

1. Heart rate and regularity, blood pressure
2. Jugular venous pressure
3. Murmurs
4. Pulses and bruits
5. Edema
6. Sternotomy scars [147]

2.13. Management of ventricular arrhythmias(pVT& VF):

2.13.1. Pre ventricular arrhythmias(pVT& VF) management:

When the patient start to have palpitation the nurse in critical care unit must follow these steps, before ventricular arrhythmias take place

Immediate Interventions:

- Place patient supine in bed. Apply O2 if available at bedside.
- Stay with patient, and provide reassurance.
- Take BP, and assess apical HR and rhythm. Compare apical rate to radial rate as one measure of perfusion.
- Check for patent IV access.
- Quickly assess perfusion by assessing mental status, peripheral pulses.

- Observe cardiac monitor if patient is being monitored. Obtain rhythm strip to document event.
- Notify physician or NP.
- Document patient's status, phone call to physician or NP, and physician or NP response.[148]

Focused Assessment:

- Assess LOC, VS, and pulse quality and rhythm.
- Assess precipitating event, pain level, anxiety, and hyperventilation.
- Assess breath sounds, O2 saturation
- Assess peripheral pulses, skin temperature and color, edema.
- Assess trends in pertinent laboratory data, e.g., Hg, Hct, electrolytes.
- Obtain and assess laboratory data such as ABG, cardiac enzymes, if appropriate.
- Document assessment thoroughly. [148]

Stabilizing and Monitoring:

- Continue to monitor rhythm; obtain and analyze rhythm strip every 4 hours and when rate or rhythm changes.
- Continue to monitor VS and O2 saturation.
- Keep IV line patent, and infuse IVF.
- Review laboratory data such as Hgb/Hct; BUN and creatinine; electrolytes, other chemistries, blood glucose, liver and cardiac enzymes.
- Check MAR for possible drug side effect or interactions.
- Chart patient status, and convey to physician or NP. [148]

Be Prepared To:

- Obtain a 12- or 15-lead ECG
- Administer antiarrhythmic medication (e.g. amiodarone).
- Obtain IV access, administer ordered IVF and medications.
- Transfer patient to a unit with cardiac monitoring.
- Assist with placement of temporary transvenous or external pacemaker or cardioversion. [148]

2.13.2. Acute management for ventricular arrhythmias(pVT& VF):

The most common electrical mechanisms for cardiac arrest are VF or VT, brad arrhythmias, asystole and electromechanical dissociation (pulseless electrical activity). Overall, survival is better for patients presenting with ventricular tachyarrhythmia compared with asystole. [149]

In hospital settings, Pulseless Ventricular Tachycardia and & Ventricular Fibrillation are treated by using Advance Life Support (ALS) protocols. For long-term management of Ventricular tachycardia/fibrillation (VT/VF) an implantable cardioverter defibrillator (ICD) prolongs the lifespan of patients when used for primary or secondary prophylaxis of sudden cardiac death. [150, 151] Surgical correction of underlying disorders (e.g., percutaneous coronary intervention, coronary artery bypass surgery) may also be indicated.

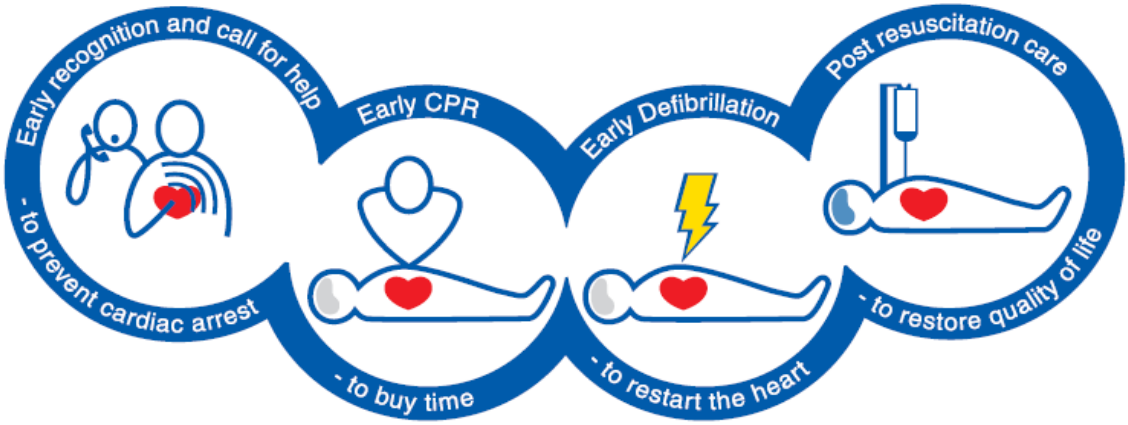
Moreover, Pulseless VT and VF treated with immediate defibrillation. High-dose unsynchronized energy should be used. The initial shock dose on a biphasic defibrillator is 150 J, followed by a higher shock dose 200 J for subsequent shocks. If a monophasic defibrillator is used, the initial and subsequent shock dose should be 360 J.

Semi-automated defibrillators provide an excellent technology to spread defibrillation capability within hospitals. In patients with an ICD, the defibrillator patches should be placed on the chest wall ideally at least 8 cm from the generator position. Intravenous amiodarone may facilitate defibrillation and/or prevent VT or VF recurrences in an acute situation. Advanced life-support activities other than those related to electrical measures for termination of ventricular tachyarrhythmias are summarized in the ILCOR document. [152]

The chain of survival

The Chain of Survival summarizes the vital links needed for successful resuscitation **(Fig. 1).** Most of these links apply to victims of both primary cardiac and asphyxial arrest.[153]

- Early recognition and call for help.
- Early bystander CPR.
- Early defibrillation.
- Early advanced life support and standardized post-resuscitation care.



(Figure No. 1): From European Resuscitation Council (ERC)

A study was conducted on endorsement of the chain of survival concept and prevalence of defibrillation and cardio version in hospital setting. The sites selected for this study were two acute care hospitals in rural Australia (RRMA Classification). Each of these hospitals was in located 'other rural areas' (RRMA Classification) in separate towns and had 25 and 30 beds. The study sample consisted of 10 females and two males. The result revealed that the two categories were 'quicker response times' (15 responses) and 'increased success with resuscitation' (8 responses). [154]

The management of VF& pVT based on the European Resuscitation Council Guidelines for Resuscitation 2015, ALS algorithm (Fig. 1.2):

1. Initial evaluation:
    - Confirmed cardiac arrest.
    - Activate emergency response system.
2. Initial intervention:
    - Start high-quality cardiopulmonary resuscitation (CPR) with a compression: ventilation (CV) ratio of 30:2.
    - Administer oxygen.
    - Attach monitor/defibrillator.
    - Monitor blood pressure and pulse-oximetry.
3. Check rhythm:
    - Shockable rhythm = VF/pVT
4. Initial treatment of VF/pVT:
    - If VF/pVT is confirmed.
    - Charge the defibrillator while another rescuer continues chest compressions.
    - Once the defibrillator is charged, pause the chest compressions.
    - Quickly ensure that all rescuers are clear of the patient
    - Then give one shock immediately.
    - Continue CPR for 2 minutes
    - Obtain intravenous (IV)/intraosseous (IO) access
    - Consider advanced airway, end-tidal carbon dioxide tension ($PETCO_2$)
5. Check pulse and rhythm (every 2 minutes):
    - Shockable rhythm = VF/pVT
    - Non-shockable rhythm = asystole/PEA
    - Rotate chest compressors
6. Continuing treatment of VF/pVT:
    - Defibrillate immediately
    - Continue CPR for 2 minutes
    - Treat reversible cause
7. Administer epinephrine after third Shock and then every 3-5min.
8. Administer amiodarone if it is available or lidocaine after third Shock.
9. Check pulse and rhythm (every 2 minutes)
    - If shockable, return to step 4

- If non-shockable, follow asystole/PEA algorithm
- Rotate chest compressors

Drug therapy

- Epinephrine 1 mg IV/IO every 3-5min.
- Amiodarone 300 mg IV/IO bolus first dose, 150 mg IV/IO second dose.
- Flush medications with 20 mL fluid after and elevate extremity for 10-20 seconds.
- Combining medications is not recommended and may cause harm
- Routine use of sodium bicarbonate is not recommended

Shock energy

- Biphasic: (150–360 J), use maximum dose if unknown waveform type
- Monophasic: 360 J

CPR quality

- Ensure high quality of Chest Compression.
- Push hard and fast (>100/min)
- Allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- Compressions-to-ventilations ratio of 30:2
- Continuous compressions if advanced airway present
- If $PETCO_2$ < 10 mm Hg, attempt to improve CPR quality
- If diastolic pressure < 20 mm Hg, attempt to improve CPR quality

Defibrillation

- Attach and use defibrillator as soon as available
- Minimize interruptions in chest compressions before and after shock
- Resume CPR beginning with compressions immediately after each shock

Advanced airway

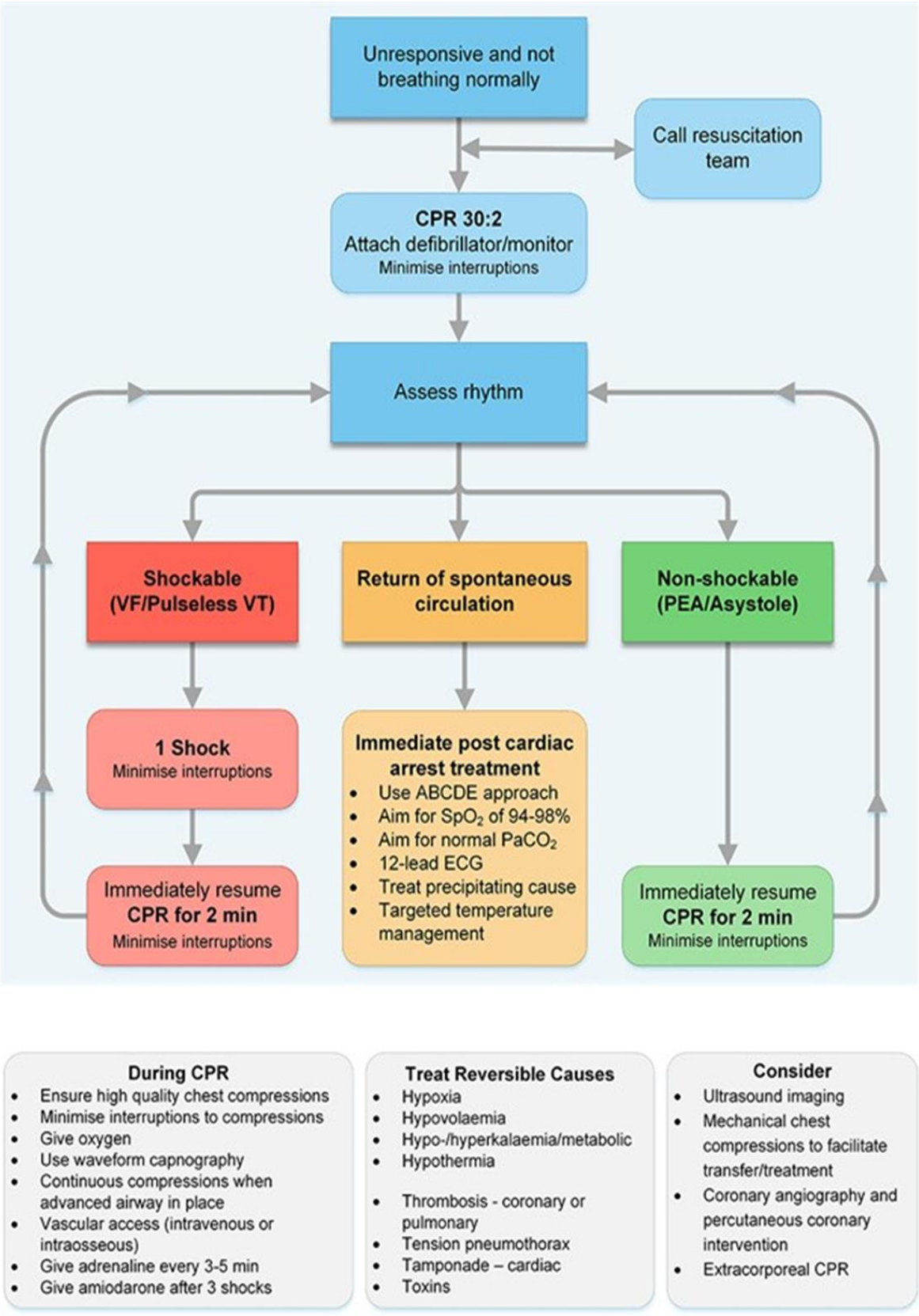
- Supraglottic advanced airway or endotracheal (ET) intubation
- Waveform capnography to confirm and monitor ET tube placement
- Ventilation every 6-8 seconds asynchronous with compressions
- Stop CPR for no longer than 10 seconds for the placement of an advanced airway

Reversible causes

- **H's:** Hypovolemia, hypoxia, H+ (acidosis), hypokalemia, hyperkalemia, hypothermia
- **T's:** Toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary, coronary)

Return of spontaneous circulation

- Pulse and blood pressure present
- Abrupt sustained increase in $PETCO_2$ (typically >40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring. [155]



(Figure No. 2): From European Resuscitation Council (ERC)

Important Recommendations for Management for pVT and VF:

1. CPR should be performed in patients in cardiac arrest, according to published basic and advanced cardiovascular life support algorithms.
2. In patients with hemodynamically unstable pVT and VF that persist or recur after a maximal energy shock, intravenous amiodarone should be administered to attempt to achieve a stable rhythm after further defibrillation.
3. Patients presenting with pVT and VF with hemodynamic instability should undergo direct current cardioversion.
4. In patients with polymorphic VT or VF with ST-elevation MI, angiography with emergency revascularization is recommended.
5. In patients with a witnessed cardiac arrest due to VF or polymorphic VT that is unresponsive to CPR, defibrillation, and vasopressor therapy, intravenous lidocaine can be beneficial.
6. In patients with a recent MI who have VT/VF that repeatedly recurs despite direct current cardioversion and antiarrhythmic medications (VT/VF storm), an intravenous beta blocker can be useful.
7. In patients in cardiac arrest, administration of epinephrine (1 mg every 3 to 5 minutes) during CPR may be reasonable.
8. In patients with cardiac arrest, administration of high-dose epinephrine (>1 mg boluses) compared with standard doses is not beneficial.
9. In patients with refractory VF not related to torsades de pointes, administration of intravenous magnesium is not beneficial.
10. In patients with suspected AMI, prophylactic administration of lidocaine or high-dose amiodarone for the prevention of VT is potentially harmful.
11. Minimize the delay between stopping chest compressions and delivery of the shock (the pre-shock pause); even a 5–10 s delay will reduce the chances of the shock being successful.
12. Without pausing to reassess the rhythm or feel for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions to limit the post-shock pause and the total peri-shock pause. Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it takes time to establish a post shock circulation and it is very rare for a pulse to be palpable immediately after defibrillation.

13. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved.

Medication used during pVT and VF Management:

Another technique that is promulgated to support patients with cardiac arrest is IV administration of medication to improve blood pressure and flow to the coronary and cerebral circulation so as to enhance the likely restoration of circulation and to reduce brain injury. Increased coronary perfusion pressure and cerebral perfusion pressure during CPR may be offset by increased myocardial work and reduced sub endocardial perfusion [156]. Multiple large observational studies from Southeast Asia have suggested that early but not late use of epinephrine is associated with improved outcomes after OHCA [157, 158].

I. Epinephrine

Epinephrine produces beneficial effects in patients during cardiac arrest, primarily because of its alpha-adrenergic (i.e., vasoconstrictor) effects. These alpha-adrenergic effects can increase coronary and cerebral perfusion pressure during CPR. The value and safety of the beta-adrenergic effects of epinephrine are controversial because they may increase myocardial work and reduce sub endocardial perfusion [156].

Based on expert consensus, for VF/pVT give adrenaline after the third shock once chest compressions have resumed, and then repeat every 3–5 min during cardiac arrest (alternate cycles). Do not interrupt CPR to give drugs. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. If ROSC is suspected during CPR, withhold adrenaline and continue CPR. Give adrenaline if cardiac arrest is confirmed at the next rhythm check. [155]

Epinephrine may increase coronary and cerebral perfusion pressure during CPR because of its vasoconstrictive effects. High doses of epinephrine (0.1 to 0.2 mg/ kg IV, as opposed to a standard dose of 1 mg) have been studied in RCTs. In out-of-hospital cardiac arrest unresponsive to defibrillation, administration of high-dose epinephrine improved survival to hospital admission, but there was no difference compared to standard dose epinephrine in survival to hospital discharge [159]. There was also no improvement in long-term survival [160]. Of note, the administration of vasopressin is

no longer recommended in the most recent advanced cardiovascular life support algorithms [156].

Timing of adrenaline dosing can cause confusion amongst ALS providers and this aspect needs to be emphasized during training [161]. Training should emphasize that giving drugs must not lead to interruptions in CPR and delay interventions such as defibrillation. Human data suggests drugs can be given without affecting the quality of CPR. [162]

II. Amiodarone:

Amiodarone possesses a wide spectrum of actions that include blockade of beta-receptors and sodium, calcium and potassium currents (i.e., a multichannel blocker). Its overall long-term effect on survival is controversial, with most studies showing no clear advantage over placebo. A few studies and a meta-analysis of several large studies have shown a reduction in SCD using amiodarone in patients with LV dysfunction due to prior MI and NICM [163- 165]. For secondary prevention of SCD, the same systematic review identified neither risk nor benefit with amiodarone [166]. Compared with beta-blocker therapy and other antiarrhythmic medications (including sotalol), amiodarone appears to reduce the risk of SCD and all-cause mortality [166]. Intravenous amiodarone has a role in reducing recurrent VF/VF during resuscitation [167- 170].

The dose is 300 mg bolus for VF/ pulseless VT arrest; 1 mg/min x 6 h, then 0.5 mg/min x 18 h.

*Although up to 800 mg every 8 h might be used, higher doses of amiodarone are associated with a higher risk of adverse events.

III. Lidocaine:

Except in specific circumstances, sodium channel blockers (Vaughn-Williams class I agents) have a limited role in the prevention of VT/SCD; this is based on a lack of survival benefit and increased mortality observed during chronic therapy in patients with ischemic heart disease. Specific circumstances where sodium channel blockers have been used to treat VT/SCA include intravenous lidocaine for patients with refractory VT/cardiac arrest [167].

The dose is 1 mg/kg bolus, 1–3 mg/min 1-1.5 mg/kg.

Repeat 0.5– 0.75 mg/kg bolus every 5– 10 min (max cumulative dose 3 mg/kg).

Maintenance infusion is 1–4 mg/min although one could start at 0.5 mg/min

IV. Electrolytes:

Administration of potassium and magnesium has been proposed as helpful adjuncts in the prevention of VA [171, 172]. Hypokalemia and hypomagnesemia are common consequences of diuretic therapy in HF, both have been associated with VA during an acute MI [172, 173], and can increase the risk of torsades de pointes in patients on medications or with conditions known to prolong the QT interval [174]. In fact, in patients with torsades de pointes, intravenous magnesium is first-line therapy [175]. In patients who are deficient in both magnesium and potassium, magnesium should be repleted to facilitate replacement of the potassium [176].

In the case of potassium, some recommend keeping the potassium level between 4.5 mmol/L and 5 mmol/L to prevent VA and SCD [177, 178]. A large observational study of patients with an acute MI found that the lowest rates of death were seen in patients with serum potassium concentrations between 3.5 mmol/L and <4.5 mmol/L [179].

Magnesium may suppress automaticity, suppress early and late after-depolarizations, and inhibit calcium flux into cardiomyocytes. It is effective in suppressing VA related to acquired long QT syndrome. However, 2 RCTs that investigated the use of intravenous magnesium in patients with cardiac arrest and refractory VF found no benefit [180, 181]. In a study of out-of-hospital cardiac arrest, administration of 2 to 4 g magnesium intravenously did not improve survival to hospital admission [180]. In a similar study involving inpatient cardiac arrest, magnesium did not improve return of spontaneous circulation, survival to 24 hours, or survival to hospital discharge [181].

Defibrillation:

A defibrillator is a device that delivers an electric shock to the heart muscle through the chest wall in order to restore a normal heart rate. There are different types of defibrillators are in use, those are manual external defibrillator monitor. In which health care provider will decide what charge (in joules) to use, based on guidelines and experience, and will deliver the shock through paddles or pads on the patient's chest. Automated external defibrillator are the simple-to-use units are based on computer technology which is designed to analyze the heart rhythm itself, and then advise the user whether a shock is required and implantable cardioverter-defibrillator also known as automatic internal cardiac defibrillation. These devices are implants, similar to pacemakers. They constantly monitor the patient's heart rhythm, and automatically administer shocks for various life threatening arrhythmias, according to the device's programming [182].

Defibrillation is the definitive treatment for the life-threatening cardiac arrhythmias, ventricular fibrillation and pulse less ventricular tachycardia. Defibrillation consists of delivering a therapeutic dose of electrical energy to the affected heart with a device called a defibrillator. This depolarizes a critical mass of the heart muscle, terminates the arrhythmia, and allows normal sinus rhythm to be reestablished by the body's natural pacemaker, in the senatorial node of the heart. Defibrillators can be external, transvenous, or implanted, depending on the type of device used or needed. Some external units, known as automated external defibrillators (AEDs), automate the diagnosis of treatable rhythms, meaning that lay responders or bystanders are able to use them successfully with little or in some cases no training at all. [183]

External electrical defibrillation remains the most successful treatment for pVT & VF. A shock delivered to the heart to uniformly and simultaneously depolarize a critical mass of the excitable myocardium. The objectives are to interfere with all reentrant arrhythmia and to allow any intrinsic cardiac pacemakers to assume the role of primary pacemaker.

Consequently, early defibrillation is vital; emergency medical services personnel can perform defibrillation at the scene, long before the patient could be seen at the emergency department (ED). In addition, the placement of AEDs in public places such as airports and casinos allows prompt use of these devices by trained laypersons. [17]

Defibrillation success rates decrease 5-10% for each minute after onset of VF. Success rates of 85% have been reported in strictly monitored settings where defibrillation was performed most promptly. [17]

Factors that affect the energy required for successful defibrillation include the following:

- Time from onset of pVT or VF to defibrillation
- Paddle size
- Paddle-to-myocardium distance: This is effected, for example, by obesity or mechanical ventilation
- Use of conduction fluid (e.g. disposable pads, electrode paste/jelly)
- Contact pressure
- Stray conductive pathways (e.g. electrode jelly bridges on skin)
- Previous shocks, which decrease defibrillation threshold
- The metabolic condition of the myocardium.[17]

The goal is to use the minimum amount of energy required to overcome the threshold of defibrillation. Excessive energy can cause myocardial injury and arrhythmias.

Larger paddles result in lower impedance, which allows the use of lower-energy shocks. Approximate optimal sizes are 8-12.5 cm for an adult, 8-10 cm for a child, and 4.5-5 cm for an infant. Position one paddle below the outer half of the right clavicle and one over the cardiac apex (V_4 -V_5). [17]

Before any defibrillation, remove all patches and ointments from the chest wall because they create a risk of fire or explosion. The patient must be dry and not in contact with metallic objects. Rescuers must remember to ensure the safety of everyone around the patient before each shock is applied. [17]

Defibrillation causes serum creatine phosphokinase levels to increase in proportion to the amount of electric energy delivered. If customary voltage is used to defibrillate a patient, the proportion of myocardial fraction (CK-MB) should remain within reference ranges unless an infraction has caused myocardial injury. [17]

A study was conducted to assess the patient's survival rate with cardiac arrest those who are subjected defibrillation health centers at London. A series of activities of health team members including medial, Para medical was observed during the defibrillation of patients in coronary care unit and intensive care unit. The probability of survival progressively decreased when defibrillation was delayed for more than 10 minutes. Study showed that the survival of the patient fibrillation among cardiac arrest patients was good when the fibrillating activity was rapid and fast. Study also stated delay in action recovery team members will lead poor recovery of patients. [184]

A systematic study done from Indian heart rhythm society in 2008. The objective was to assess the effectiveness of early defibrillation by nurse in coronary care nurse defibrillation programme. They say that that all episodes of early defibrillation that were delivered by CCU nurses from 1 October 2008 to 31 January 2009 were prospectively studied. It reported that there was surprisingly increased survival rate to 89% in early defibrillation compared with survival rate of 56% in delayed defibrillation given from doctors.[185]

A cross sectional study done from Central Queensland University in 2007 says, Evidence indicates that hospital nurse-initiated defibrillation improves survival following cardiac arrest. Accordingly, hospitals are changing their policies to permit nurses to initiate defibrillation. However, if nurse-initiated defibrillation is to be successful implemented, nurses' beliefs about this practice need to be understood.

Therefore, the aim of this study was to explore the attitudes of rural nurses towards defibrillation to assist in the development of nurse-initiated defibrillation programmes.[186]

A study was conducted on "treatment of ventricular tachycardia in patients with heart failure" in USA. The study suggested that management strategies for ventricular tachycardia include implantable cardioverter-defibrillator therapy, pharmacological therapy, catheter ablation techniques, ventricular assist device therapy and heart transplantation. The study concludes that ventricular tachycardia is a life threatening condition and it requires immediate treatment and effective management.[187]

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm.[188- 192] its routine use is therefore not recommended. It may be appropriate therapy only when used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. [193] Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, and then retract the fist immediately to create an impulse-like stimulus. There are rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm. [194]

Although the precordial thump is less appropriate for VF than for VT, it actually is appropriate in neither. Use it only for witnessed, monitored arrests in which no defibrillator is immediately available. [17]

2.13.3. Long-Term Treatment:

2.13.3.1. Implantable Cardioverter-Defibrillators

For long-term management of Ventricular tachycardia/fibrillation (VT/VF) an implantable cardioverter defibrillator (ICD) prolongs the lifespan of patients when used for primary or secondary prophylaxis of sudden cardiac death. [195 -197] It is indicated for secondary prevention in patients with a history of sustained VT/VF, and for primary prevention in patients with a history of heart failure or previous MI and left ventricular ejection fraction (LVEF) of 35% or less [48].

Survivors of VF that does not have a clear and readily reversible cause should be implanted with an ICD. Transvenous ICDs can be placed with minimal morbidity and mortality. Several multicenter trials have demonstrated the prophylactic value of ICD therapy in patients at high risk for VF.

In several studies that compared ICD placement with antiarrhythmic therapy in patients with VT/VF and/or prior cardiac arrest, ICD placement was shown to be associated with a significantly decreased mortality rate.[199- 201] However, ICD placement may also be appropriate in conjunction with antiarrhythmic therapy. Matsue et al demonstrated the benefit of ICD placement and medication in patients with vasospastic angina who had been resuscitated from lethal ventricular arrhythmia.[198]

The use of ICDs as primary prevention for VF has also been demonstrated in patients with LV dysfunction. Newer ICDs have pacing capabilities and have addressed bradyarrhythmias that either cause or complicate VT or VF. ICDs are indicated for the secondary prevention of VF and for the primary prevention of VF in patients with an LV ejection fraction of less than 35%, whether due to ischemic or non-ischemic cardiomyopathy. [202, 203]

2.13.3.2. Cardiac Surgery and Revascularization Procedures:

Cardiac surgery can be a primary treatment for VF via a variety of strategies. Surgical treatment in patients with ventricular arrhythmias and ischemic heart disease includes coronary artery bypass grafting (CABG). The Coronary Artery Surgery Study (CASS) illustrated that patients with significant coronary artery disease (CAD) and operable vessels who underwent CABG had a decrease in the incidence of VT/VF arrest compared with patients on conventional medical treatment. The reduction was most evident in patients who had 3-vessel disease and chronic heart failure. [204]

By itself, CABG prevents recurrent VF only if the ejection fraction is normal and ischemia was the cause of the arrest. Surgical treatment of ventricular arrhythmias in patients with non-ischemic heart disease includes excision of VT foci after endocardial mapping and excision of LV aneurysms. This is practiced very infrequently due to significant morbidity and limited efficacy.

Aortic valve replacement is associated with improved outcome in patients with hemodynamically significant valvular stenosis and well-preserved ventricular function. Mitral valve replacement is indicated for patients with mitral valve prolapse who have malignant tachyarrhythmias such as VT and VF associated with significant valvular regurgitation and LV dysfunction.

In patients with SCA or life-threatening VA presumed related to ischemia caused by anomalous origin of a coronary artery, repair or revascularization is performed to alleviate ischemia and reduce the recurrence of VA [205-209]

2.13.3.3. Catheter Ablation

Catheter ablation is an important treatment option for patients with VA when antiarrhythmic medications are ineffective, not tolerated, or not desired by the patient. Monomorphic VA usually have an origin or substrate that can be targeted for ablation. Ablation is an option for selected patients with polymorphic VT/VF only if an initiating PVC focus or substrate can be identified. The catheter ablation procedure usually involves attempts to induce VT by programmed electrical stimulation to confirm the diagnosis and guide ablation. Problems limiting success include inability to induce an arrhythmia for mapping (common with idiopathic VA), or origin of the arrhythmia from an inaccessible location in the myocardium (common in some cardiomyopathies). [147]

2.13.3.4. Diet and Activity

Patients with ischemic VT may benefit from low-cholesterol diets, low-salt diets, or both. Patients with idiopathic VT may notice a reduction in symptoms when stimulants (eg, caffeine) are avoided. [210] Fish oil supplementation does not reduce the risk of VT or VF in ICD patients with recent sustained ventricular arrhythmia. [211]

VT may be precipitated by increased sympathetic tone during strenuous physical exertion. One goal of successful VT management is to allow the patient to return to an active lifestyle through medications, ICD implantation, ablation therapy, or some combination thereof.

The 2006 ACC/AHA/ESC guidelines recommend that smoking be strongly discouraged in all patients who have, or who are thought to have, ventricular arrhythmias, aborted sudden cardiac death (SCD), or both. Cigarette smoking is an independent risk factor for SCD, typically from arrhythmia and regardless of underlying coronary heart disease, and smoking cessation significantly reduces the risk of SCD.[212]

A study was conducted on “possibilities of preparation omega 3 polyunsaturated fatty acids in the treatment of patients with ventricular arrhythmia and myocardial infarction” in Russia. The study involves 56 patients with ventricular extra systoles, from 500 to1000 per day. The study concluded that administration of omega 3 polyunsaturated fatty acids for 3 months reduced the number of premature ventricular contraction per day. These effects persisted after 6 months of treatment. [213]

2.13.4. Post-resuscitative Care:

Resuscitated patients must be admitted to an intensive care unit and monitored because high rate of early recurrence. Antiarrhythmic successfully used during resuscitation are usually continued. Maintenance infusions of amiodarone (0.5-1 mg/min) are the most commonly used therapies. Control any hemodynamic instability. Administer adrenalin as indicated.

Post-defibrillation arrhythmias (mainly atrioventricular [AV] blocks) have been reported in up to 24% of patients. The incidence is related to the amount of energy used for defibrillation.

Check for complications (eg, aspiration pneumonia, CPR-related injuries), and establish the need for emergent interventions (eg, thrombolytics, antidotes, decontamination).

Careful post-resuscitative care is essential to survival because studies have shown a 50% repeat in-hospital arrest rate for people admitted after a VF event. Multiple randomized trials have confirmed the benefit of treating myocardial ischemia, heart failure, and electrolyte disturbances.

Mild therapeutic hypothermia has been shown to improve neurologic outcomes and survival after out-of-hospital cardiac arrest and should be considered in appropriate patients.[214, 215] Traditionally, a target temperature of 32-34°C has been recommended. A study has shown, however, that in unconscious survivors of out-of-hospital cardiac arrest of presumed cardiac cause, hypothermia at a targeted temperature of 33°C did not confer a benefit as compared with a targeted temperature of 36°C.[216]

Patients require stabilization and monitoring for the possibility of a coexistent emergency or complication. Empiric beta-blockers are reasonable in many circumstances because of favorable properties discussed in Etiology. However, empiric antiarrhythmics, including amiodarone, should not supersede ICD placement unless control of recurrent VT is needed while the patient is hospitalized. [17]

Evaluation of ischemic injury to the central nervous system, myocardium, and other organs is essential. Survivors should undergo thorough diagnostic testing to establish the underlying etiology of the VF episode. If available, perform indicated interventions to improve long-term prognosis. [17]

2.14. Complications and adverse features:

The presence or absence of adverse symptoms or signs will dictate the appropriate immediate treatment for most arrhythmias. The following adverse features indicate that a patient is at high risk of early deterioration and death (‘unstable’), either because of the arrhythmia itself or because of underlying heart disease with the arrhythmia superimposed: [217]

- Shock – hypotension (systolic blood pressure <90 mm Hg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- Syncope – transient loss of consciousness due to global reduction in blood flow to the brain
- Myocardial ischaemia – typical ischaemic chest pain and/or evidence of myocardial ischaemia on 12-lead ECG
- Heart failure – pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).
- VT may deteriorate into VF.

Ventricular arrhythmias, including ventricular tachycardia (VT) and ventricular fibrillation (VF), are the leading cause of sudden cardiac death (SCD). [55]

2.15. Prognosis:

The prognosis is correlated with the type of arrhythmia and the type and degree of structural heart disease. NSVT, as well as frequent PVCs, have generally no impact on outcome. Patients with sustained ventricular arrhythmias have poorer short- and long-term prognosis. An in-hospital mortality rate of up to 50% has been reported in patients with sustained ventricular arrhythmias after surgery. Among patients who survive in-hospital sustained ventricular arrhythmias, up to 40% have a recurrence. As many as 20% of these patients die from cardiac causes within 24 months [218, 219].

ES is associated with significantly adverse prognosis, particularly in those patients with impaired cardiac function. However, the increased risk of mortality and hospitalization may be due to worsening heart disease in patients with ES, rather than the ES itself [220- 222]. Regardless, the increased mortality risk exists in patients who received an ICD for either primary or secondary prevention of sudden cardiac death (SCD).

Prognosis does not always correlate with left ventricular function. Patients with long QT syndrome, right ventricular dysplasia, or hypertrophic cardiomyopathy

may be at increased risk for sudden death despite relatively well-preserved left ventricular function. These possibilities should be considered in any patient with a strong family history of premature sudden death.[18]

VT can also result in sudden death. Patients in whom this occurs may first present with syncope.

- If treated rapidly, VT generally has a favorable short-term outcome.
- Long-term prognosis depends upon the underlying cardiac disease.

Hemodynamic collapse is more likely when underlying left ventricular dysfunction is present or when heart rates are very rapid. Diminished cardiac output may result in diminished myocardial perfusion, worsening inotropic response, and degeneration to ventricular fibrillation (VF), resulting in sudden death. [18]

Brugada syndrome: It is possible that some patients with what is thought to be primary VF may have Brugada syndrome; VF in these patients usually has no preceding symptoms; the prognosis is unfavorable, and the recurrence rate is as high as 33%. [17]

CHAPTER THREE

❖ Material &Methodology

3. Methodology

3.1. Study Design:

This study was hospital-based quasi-experimental study with a pre and posttest design conducted among nursing staff in critical care units. All subjects attended an educational intervention program on how to recognize and manage ventricular arrhythmias (ventricular fibrillation and pulseless ventricular tachycardia) as per ERC guidelines, to evaluate the effect of applying this program on nurses' knowledge and performance regarding the recognition and management of ventricular arrhythmias.

3.2. Study Area:

Khartoum State is one of the eighteen states of Sudan. Although it is the smallest state by area (22,142 km2), it is the most populous (5,274,321 in 2008 census) as it includes the nation's capital city. The city is located in the heart of Sudan at the confluence of the White Nile and the Blue Nile, where the two rivers unite to form the River Nile. The state lies between longitudes 31.5 to 34 °E and latitudes 15 to 16 °N.

It is surrounded by River Nile State in the north-east, in the north-west by the Northern State, in the east and southeast by the states of Kassala, Gedaref and Gezira, and in the west by North Kurdufan. The 2008 population census estimates the population of Khartoum state to be about 7,687,547, composed of various tribes of the Sudan. The temperature in summer ranges from 25 to 40 °C from April to June, and from 20 to 35 °C in the months of July to October.

Most of the population works in government service, the private sector, and banking. There is also a large number of merchants, and migrants and displaced people working in marginal activities.

3.3. Study setting:

The chosen areas for this study were:

1. Sudan Heart Center.
2. Khartoum Teaching Hospital.
3. Al-Shaab Teaching Hospital.

Those Hospitals serve approximately 500,000 clients per year; the following are brief descriptions of each hospital.

3.3.1. Sudan Heart Center:

A governmental cardiac center, owned and directed by the Sudanese Army, located in Khartoum state, Khartoum locality. It provides medical and surgical care for both adult and pediatric patients with heart diseases. Located in Khartoum city Arkaweet locality. The Noor Alayoun hospital is east of it and Africa Street and Future University from the west.

It is composed of cardiac medicine and cardiac surgery departments, adults and pediatrics section, divided into, Outpatient unit containing 6 beds, Cath lab unit containing 9 beds ,ICU containing 8 beds, CCU containing 8 beds , male and female wards of 15 beds each. It has an operating theatre, Laboratory and X-Ray department. It receives about 50 – 100 heart cases daily for referred cardiac problems and about 2 to 3 open heart surgeries are carried out daily for 5 days per week. Around 110 registered nurses provide bedside nursing care, including 45 critical care nurses.

3.3.2. Khartoum Teaching Hospital:

This is the oldest governmental teaching hospital in Sudan, located in a midtown of Khartoum and east of the faculty of medicine of Khartoum University, and west to Mack Nimer Street and Al-Shaab teaching hospital. It is surrounded from the north by a lot of private hospitals and Said Abdurrahman Street, from the south freedom bridge and train station.

The hospital now contains four sections; general surgery, pediatric surgery, fistula, and cosmetic section, and provides medical and surgical care for patients with medical and surgical conditions. There is an operating theatre, X- Ray, Laboratory departments and a blood bank.

The total number of hospital beds is more than 124 beds, 112 beds in the general ward and 12 beds in Critical care units. The total workforces of nurses include 73 registered nurses who provide bedside nursing care and 47 critical care nurses. The average number of patients seen per day is 100. It considered as an important teaching facility for many medical faculties in Sudan.

3.3.3. Al-Shaab Teaching Hospital:

Is the second biggest governmental teaching hospital in Sudan, located in a midtown of Khartoum and east of faculty of medicine of Khartoum University and Khartoum teaching hospital, and west to Mack Nimer Street and the dental hospital. It is surrounded from the north by a lot of private hospitals and Said Abdurrahman Street, from the south freedom bridge and train station.

It specializes in cardio-thoracic diseases both medicine and surgical specialties. It is composed of outpatient and emergency sections which receive fresh medical and surgical cardiac cases. The department of cardiac catheter with coronary care unit has a capacity of eight beds; the department of recovery has a capacity of eight beds. It contains a total of eight intensive and coronary care units. The intensive care unit has a capacity of nine beds, a high dependent unit with a capacity of four beds, and asthma care unit with an eight-bed capacity. Ward 1 of heart and chest (medicine) is for females with twenty beds. Ward 2 of heart and chest (medicine) is male and female with sixteen beds. Ward 3 of heart and chest (surgery) is also male and female with twenty beds and Ward 4 of heart and chest (medicine) is male with sixteen beds. Ward 9 of heart and chest is for male and female with twenty-four beds, Ward 10 of heart and chest is also a mixed ward with twenty-three beds. Pharmacy, laboratory, X-Ray department, seven specialist sections and clinics of the heart and chest equipped with echocardiography and ECG machines.

The average number of patients seen per day is 700 cardio-thoracic cases daily referred with problems and consultations. A total of 255 registered nurses provide bedside nursing care, including 136 critical care nurses. It is considered as an important teaching facility for many medical faculties in Sudan.

3.4. Study population:

The study population included all nursing staff working in the critical care units in these three hospitals at the mentioned study settings during the study period.

3.4.1. Inclusion criteria:

- Educational level a minimum of three years University diploma in nursing and above.
- Who have a job (Permanent or Contract) as critical care nurse.
- Available and freely willing to participate in this study.
- Commitment to attending all educational program about guidelines recognition on management of Ventricular arrhythmias.

3.4.2. Exclusion criteria:

- Educational level less than three years University qualification and experience.
- Part-time duty.
- Not available or willing to participate in this study.

- Lacking full commitment to the educational program about guideline recognition on management of Ventricular arrhythmias

3.5. Sampling Procedures and Sample Size:

3.5.1. Sampling frame:

This sample was chosen from a list of all critical care nurses who work in the three major governmental hospitals with critical care units as described above and summarized below in the below table.

3.5.2. Setting of the study:

Total coverage of three targeted hospitals for all Staff who work in critical care units (ICU, CCU, ACU, ICCU, HDU and OHS) was selected

Table (1) the representative full coverage sample:-

No	Cardiac center name	Frame	Proportionate sample
1	Sudan heart center	45 nurses	45 nurses
2	Khartoum teaching hospital	47 nurses	47 nurses
3	AL-Shaab teaching hospital	136 nurses	136 nurses
Total		**228 nurses**	**228 nurses**

From the above census done by the researcher for the nurses working in the study setting and fulfill the inclusion criteria during the study period, the sample was taken as a total coverage of 228 nurses.

3.6. Variables under Study

3.6.1. Nurse's knowledge regarding:

- Definition and causes of Pulseless VT and VF.
- Signs and symptoms of Pulseless VT and VF.
- Diagnostic method used for Pulseless VT and VF.
- Prevention and Complications of Pulseless VT and VF.
- Acute and long-term management for of Pulseless VT and VF.
- Interpret the Pulseless VT and VF from ECG Rhythm strip.

3.6.2. Nurses' Practice regarding:

- Interpret the pVT and VF from ECG the rhythm strip, and vital signs monitor.
- Use the ABCDE approach.
- Effective airway management.

- Effective breathing and ventilation.
- Correct delivery of high quality chest compressions
- Effective and safety use of manual defibrillator.
- Medication administration during Pulseless VT and VF management.

3.7. Data collection Tools:

A questionnaire was developed following the literature review which helped to select appropriate questions. These were adapted to form the questionnaire, which was developed by researcher and then piloted and pre-tested before use for the study. (Appendix1)

3.7.1. Questionnaire: A self-administered was developed by the researcher to assess nurses' knowledge regarding recognition and management of Ventricular arrhythmias (PVT and VF) guideline; it was composed of two sections.

I. The first part:

- Contained five questions about basic demographic data such as (Gender, age, years of experience, qualification degree, and attendance of training program regarding VF& Pulseless VT arrhythmia recognition and management).

II. The Second part:

- Included 16 questions related to basic knowledge about Ventricular arrhythmias (Pulseless VT and VF) to obtain information about Definition, Causes, signs & symptoms, diagnostic methods, prevention, complication, acute and long-term management and ECG Rhythm Strip. Moreover information about European Resuscitation Council standard guidelines for (Pulseless VT and VF) recognition and management.

The questionnaire was composed of 16 questions using a scoring system of the phrases (correct =1, Incorrect = 0), according to Likert scale system the response for the questions was ranged from very weak, weak, intermediate, Good to very good. For each question has an answer with four values.

The above range was dependent upon the values scored in all variables. Interval score was used for evaluation of the scores for the level of knowledge items which was evaluated by: very weak (0- 20%), weak (21-40%), intermediate (41- 60%), good (61- 80%), and very good (81- 100%).

The correct answer style was used for each question with four values, interpreted and judged according to same above rating as when the correct answer response is 20% and below, 21 to 40%, 41 to 60%, 61 to 80% and above 81%.

3.7.2. Observational Checklist:

An observational checklist was developed by the researcher to assess nurses' practice regarding recognition and management of ventricular arrhythmias (Pulseless VT and VF) guideline guided by European Resuscitation Council (ERC) 2015 guidelines and the base of literature review. (Appendix2)

Scale System for observational results:

The scale system had been described according to the participants' practices, rated, as done correctly, not done correctly, and not done at all.

Done correctly, not done correctly, and not done style was used for each check point interpreted and judged according to same above rating, the scale for checklist was scored into (Done Correctly= 2, not Done Correctly=1, and not done= 0).

The observational checklist includes:

1- Confirm cardiac arrest
2- Call resuscitation team
3- Perform uninterrupted CPR before and after defibrillation.
4- Confirm VF/pVT from the ECG
5- Steps and preparation before and after delivery of safe defibrillation
6- Administration Medication during Pulseless VT & VF Management

3.8. Operational Design

Operational design includes pilot study and data collection technique.

3.8.1. Pilot Study

A Pilot test was first carried out on a sample of 10 nurses from Ahmed Gassim Hospital - Heart Surgery and Kidney Transplant Center to determine the clarity of questions, effectiveness of test instructions, completeness of response sets, time required to complete the questionnaire, and success of data collection techniques.

The tools were examined by supervisor who indicated that some items needed to be modified, but overall were assured that each tool was appropriate to achieve the aim of the study. A pre-test was obtained by evaluating those 10 critical care nurses; the test was repeated after one week according to the knowledge and skill test tool.

Reliability of the instrument determined through using Pearson correlation coefficient by using Alpha Cronbach test (r = 0.821) for nurses level of knowledge and (r = 0.966) for nurses' practices. The level of the P value was (P = 0.04) which indicates statistical acceptable for the format.

3.8.1.1. The results of pilot were as the follows:

- The nurses understood the method used to fulfill each tool, and they indicated that some items needed to be modified
- Based on pilot results the modification was done and the researcher refined each tool, arranged the sequence of the questions under each item in the same part, to ensure consistency and logical flow of questions.
- Finally, making assurance that each tool as a whole achieved the aim of the study.
- The time required to fill the questionnaire was about 10-15 minutes

3.8.2. Data collection technique:-

3.8.2.1. Pretest data collection:

The data was collected in four phases firstly before implementation of the training program (pretest data). Pretest for the existing knowledge for nurses was carried out prior to the intervention using a self-administered questionnaire format; time offered was 15 minutes for each group (21 nurses), which was considered enough time for the participant to answer the questions.

The data was collected from 168 critical nurses out of 228 because the other 60 critical nurses were not available or willing to participate in this study.

Then each respondent in each group was observed using a checklist for their skills. Close supervision of the participants while applying the standard guidelines for Pulseless VT and VF recognition and management during the study period. Each group (21 nurses) was divided into four small groups under supervision of researcher or CPD Clinical Instructor, and provided a time of 10 minute for each participant:

Total: 168 Nurses / 21 Nurses = 8 Big groups

8 big groups * 52.5 Minute = 420 Minute

420 Minute /60 Minute = 7 Hours

Each group: 21 Nurses /4 small groups = 5.25 Nurses

5.25 Nurses * 10 Min = 52.5 Minute

52.5 Minute/ 60 Minute = 0.9 Hour

- The number of 21 nurses per group is a maximum number for candidates versus four clinical instructors according to the policy of Continuing Professional Development clinical training center.
- The researcher and three Continuing Professional Development Clinical Instructors carried out this part.

3.8.2.2. Posttest data collection:

After collection of pretest data the responders received the training. The same data collection tools were repeated three times; in the first time immediately after the training program finished. Then repeated again after one month and six months during a follow up phase to check retention of knowledge and practice.

3.9. Educational program:

A training program was designed based on actual needs assessment of nurses to improve their knowledge and practice regarding life threatening arrhythmias and European Resuscitation Council Standard Guidelines for (PVT and VF) Recognition and management .It was designed by the researcher in the light of European Resuscitation Council Standard Guidelines Last update 2015 ,and available researches and related literatures.

The Training program was implemented at the Continuing professional development (CPD) training center which is well equipped with all materials and class halls needed to have a successful training program. Three accredited clinical instructors were recruited to help the researcher in the delivery of practical section of this program and those clinical instructors later was apply the posttest assessment after 1 month & 6 months without any interference of the researcher to enhance have a real result.

The training program was applied in three phases as follows:

Phase one:

An orientation about the training program was been given firstly to each group to give the participants full idea about the phases of the application of the program in order to facilitate for their contribution. Orientation to the training program format, including the lecture's time, and the training materials. The intervention was been developed in English language to cover the relevant theoretical and practical aspects of VF & pVT recognition and management.

The intervention was implemented to nurses in eight groups each group was contain 21 nurses total number of nurses was 168 nurses. The program has been carry out in one group per day for two weeks for theoretical and clinical practice sites, each theoretical session was take two and half-hours, and clinical practice sessions was take three and half hours.

Pretest for the existing knowledge for nurses was been carried out prior to the intervention using self-administrative questionnaire format; time offered was 15 minutes for each group, which was considered enough time for the participants to answer the questions.

Pretest for the existing skills using the observational checklist format; time offered was 10 minutes for each participant. This part was been done by the researcher and accredited clinical instructors.

Phase two:

Orientation to the training program format, including the lecture's time, and the training materials.

Each lecture time was around forty five minutes, and there was about 15 minutes offered for discussion after each lecture for further clarification about what is missing or not understood by the participants. The breaks time was half hour for breakfast and 15 minute for Praying time.

Lectures titles were as mentioned below:

- Overview about Pulseless Ventricular tachycardia and fibrillation
- Basic Electrocardiogram (ECG) Interpretation.
- European Resuscitation Council Guidelines for Ventricular Fibrillation & pulseless Ventricular Tachycardia Management.

The course materials were provided as handouts.

Practical training was given as approved by European Resuscitation Council 2015 Guidelines, by using half body mannequin, complete body mannequin, ECG Simulator, defibrillator, and a set of airway management kit.

Each Participants group was divided into three station first station regarding effective airway management, second station regarding effective CPR technique and third station regarding safe use of defibrillator. By using three steps of demonstration for clinical training firstly clinical instructor demonstrate without comments, secondly clinical instructor demonstrate with his comments and explanations for the steps and then demonstration with candidate comments and explanations for the steps.

After that, each candidate started to implement specific skills in each station and at the end of rotation to all stations the candidates were implemented the standard guidelines for pVT & VF recognition and management under close supervision of researcher and clinical instructor.

Immediately after the training program was finished the same data collection tools were repeated by using the same self-administered questionnaire and the observational checklist formats.

Phase three:

A post-test and follow up phase was carried out with all the participants after one, and six months from the end of the program and from the first post-test the same self-administered questionnaire and the observational checklist formats were used.

3.10. Data Analysis:

The collected data was checked and cleaned for accuracy and consistency. After the data was coded and transferred to a specially designed format (excel master sheet) and a suitable database created for computer entry and further collation and statistical analysis.

Data was entered to Statistical Package for Social Science (SPSS) (version 21) database, in order to be analyzed according to the set plan. Both dependent and independent variables were displayed as frequency tables. Charts and graphs were only values of alpha equal to or less than 0.05 were considered as statistically significant. The results of categorical variables were presented with P values and confidence intervals.

Descriptive measures statistical measures used include count, percentage, and arithmetic mean, standard deviation, minimum and maximum ranges.

Frequency analysis, cross tabulation, and manual revision were used to detect any errors and through SPSS program.

3.11. Ethical Considerations:-

Permission was obtained from the Faculty of graduate studies scientific research committee. Following application, a letter of approval from the University was obtained to the study areas, and then permission was taken from each study area administrative authorities.

The study was explained to the participants in clear simple words and the participants were notified about the aims, method, expected outcome, benefits and result of this study, and those who accepted to participate in the study were include.

Written informed consent was obtained from each nurse who met the criteria and agreed to participate in this study prior to their inclusion. The privacy and dignity of nurse was been protected.

The participants in this study were assured of their confidentiality through de-identification coding and reports of data. Names of participants in this study were not used in any questionnaire or document.

All the nurses were released from work on the training day, as the agreement with administrative authorities in each hospital. Participants also understood that participation in the study would not affect them in anyway and they were free to withdraw from the study if they wished to do so.

Therefore all participants had the right to ask, to discontinue, and to refuse to answer any question of the study.

CHAPTER FOUR

❖ Results

4-Results

The results of the collected data through the questionnaire and checklist for this study after analyzed, the data presented as the following:

1. **The Questionnaire Results:**

The First part: Presented the demographic data of the study group.

The Second part: Presented the knowledge of the study group about (pVT & VF).

I. Questions about the basic knowledge regarding pulseless ventricular tachycardia and ventricular fibrillation

II. Questions about the knowledge regarding the management of pulseless ventricular tachycardia and ventricular fibrillation based on the standard ERC guidelines for 2015.

III. Questions about the knowledge regarding recognizing the Ventricular Tachycardia from the ECG rhythm-strip

The Third part: Presented the relation between the study variables.

The First part: The demographic data of the study group

Table (2):

The demographic data of the study group

Categories	Frequency	Percentage
Gender		
Male	38	23%
Female	130	77%
Total	**168**	**100%**
Age		
20 – 25 years	108	64%
26 – 30 years	35	21%
31 – 35 years	15	9%
>36 years	10	6%
Experience years in unit		
Less than one year	82	49%
1 – 3 years	55	33%
4 – 6 years	12	7%
7 – 9 years	19	11%
Degree of qualification		
Diploma	16	10%
Bachelor	139	83%
Post graduate	13	8%

Table (3):

Training program regarding (VF& Pulseless VT)

Item	Frequency	Percentage
Less than One year	43	26
Less than three years	20	12
More than Five years	4	2
I did not receive any training	101	60
Total	**168**	**100**

The Second part: The knowledge of the study group about (pVT & VF).

Table (4):

Nurses basic knowledge regarding pulseless ventricular tachycardia and ventricular fibrillation

Item	Pretest				Posttest 1				Posttest 2				Posttest 3			
	Not Know		Know		Not Know		Know		Not Know		Know		Not Know		Know	
	F	%	F	%	F	%	F	%	F	%	F	%	F	%	F	%
The definition	82	49	86	51	50	30	118	70	32	19	136	81	25	15	143	85
The common cause	55	33	113	67	24	14	144	86	13	8	155	92	10	6	158	94
The main sign	93	55	75	45	39	23	129	77	30	18	138	82	18	11	150	89
The main symptom	86	51	82	49	30	18	138	82	25	15	143	85	15	8.9	153	91
The useful diagnostic method	66	39	102	61	40	24	128	76	29	17	139	83	15	8.9	153	91
The common complication	57	34	111	66	35	21	133	79	25	15	143	85	25	15	143	85
The prevention	63	38	105	63	40	24	128	76	30	18	138	82	27	16	141	84

Figure (3):

Pretest frequencies for basic knowledge regarding pVT &VF

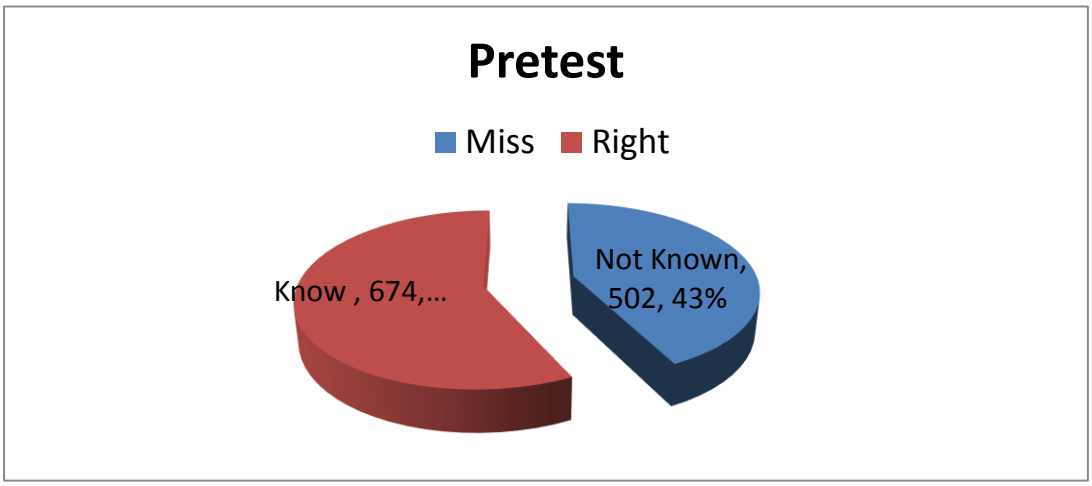


Figure (4):

Posttest 1 frequencies for basic knowledge regarding pVT &VF

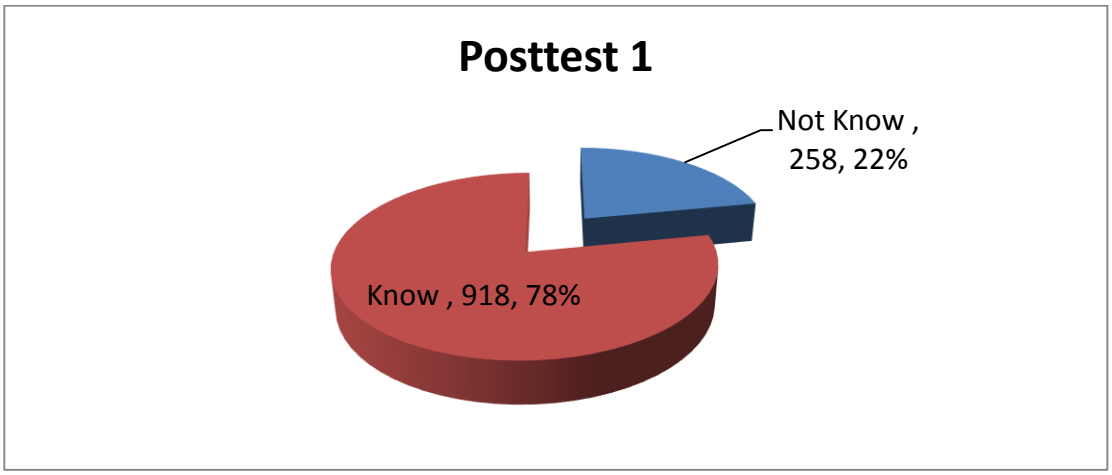


Figure (5):

Posttest 2 frequencies for basic knowledge regarding pVT &VF

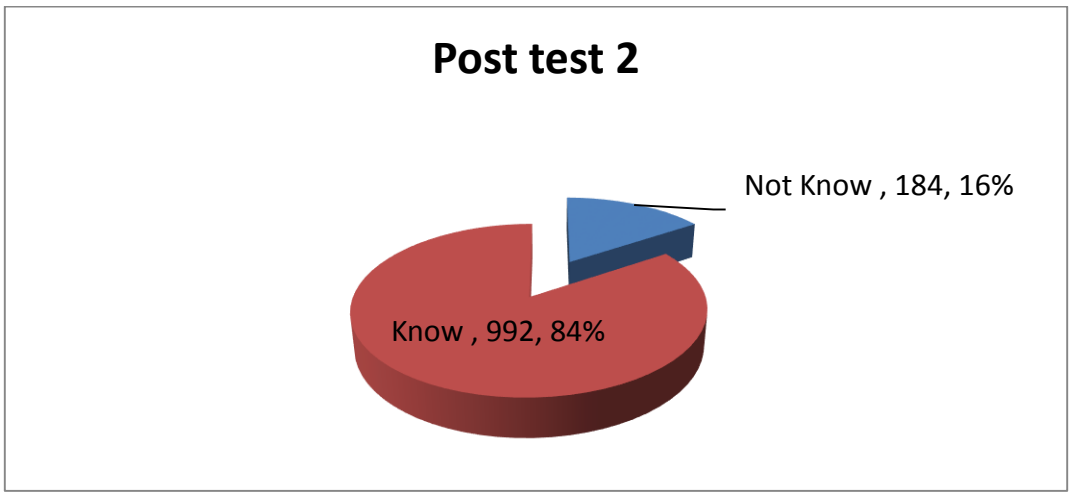


Figure (6):

Posttest 3 frequencies for basic knowledge regarding pVT &VF

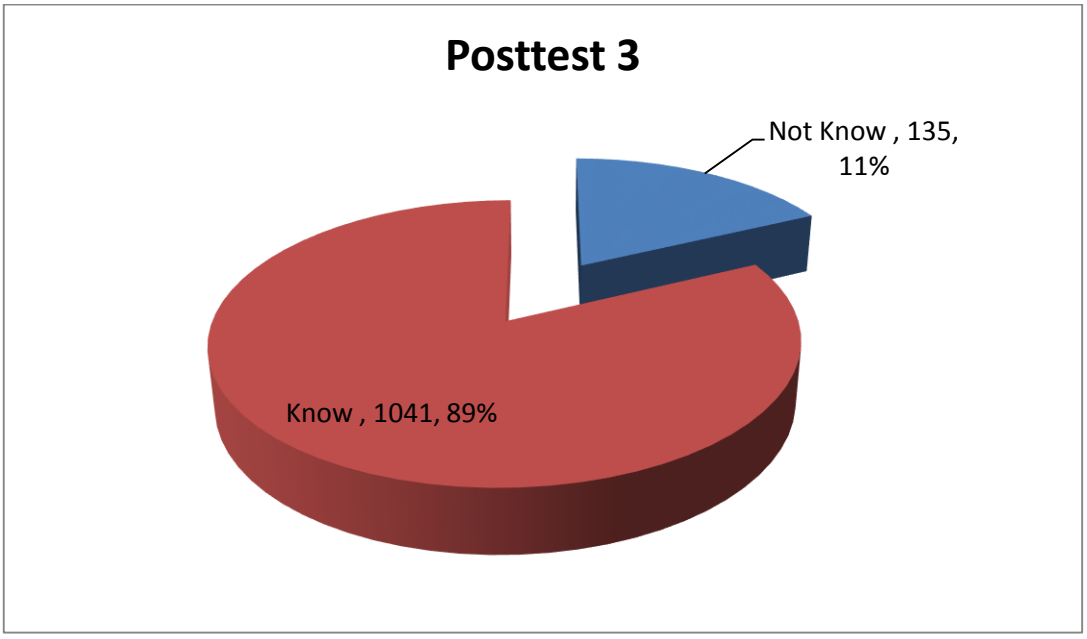


Table (5):

Nurses' knowledge about management of pulseless ventricular tachycardia and ventricular fibrillation based on the ERC standard guidelines for 2015

Item	Pretest				Posttest 1				Posttest 2				Posttest 3			
	Not Know		Know		Not Know		Know		Not Know		Know		Not Know		Know	
	F	%	F	%	F	%	F	%	F	%	F	%	F	%	F	%
The first step	105	63	63	38	60	36	108	64	30	18	138	82	35	21	133	79
The useful medication	76	45	92	55	50	30	118	70	40	24	128	76	22	13	146	87
When we start adrenaline	96	57	72	43	42	25	126	75	37	22	131	78	17	10	151	90
The appropriate energy	101	60	67	40	52	31	116	69	45	27	123	73	24	14	144	86
The immediate management	48	29	120	71	37	22	131	78	24	14	144	86	10	6	158	94
The long-term treatment	69	41	99	59	40	24	128	76	27	16	141	84	34	20	134	80

Figure (7): Pretest frequencies for knowledge regarding management and guidelines

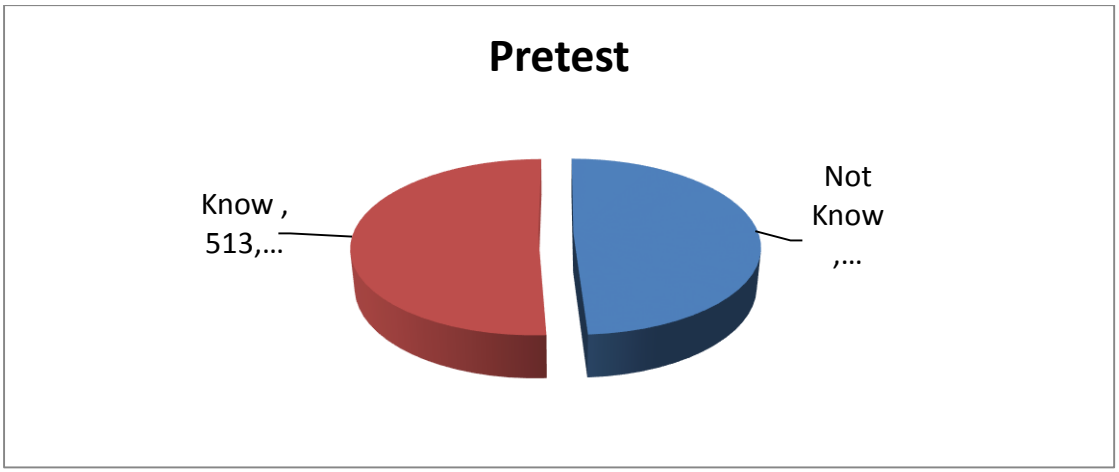


Figure (8): Posttest 1 frequencies for knowledge regarding management and guidelines

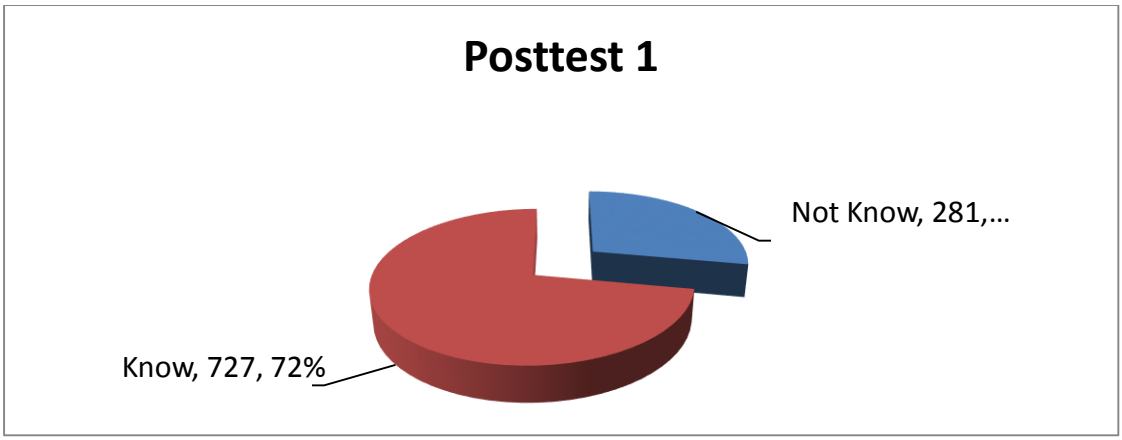


Figure (9): Posttest 2 frequencies for knowledge regarding management and guidelines

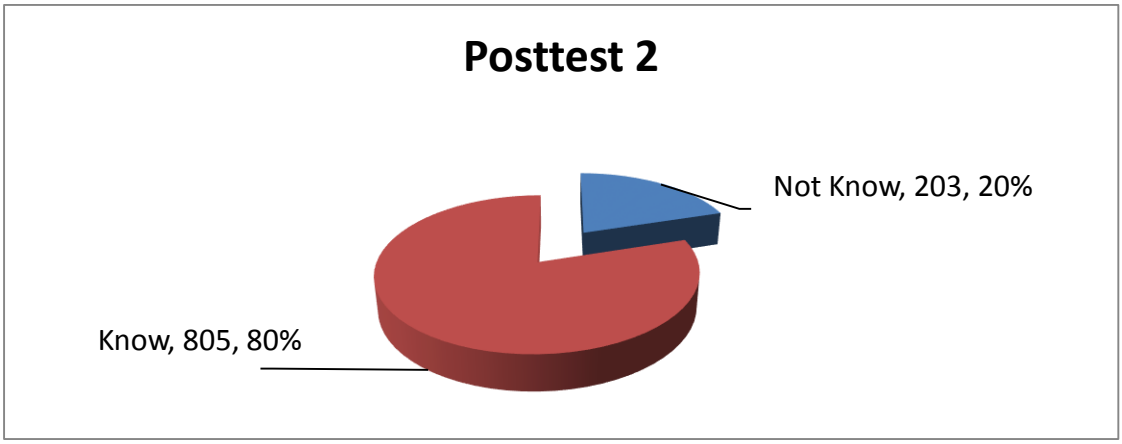


Figure (10): Posttest 3 frequencies for knowledge regarding management and guidelines

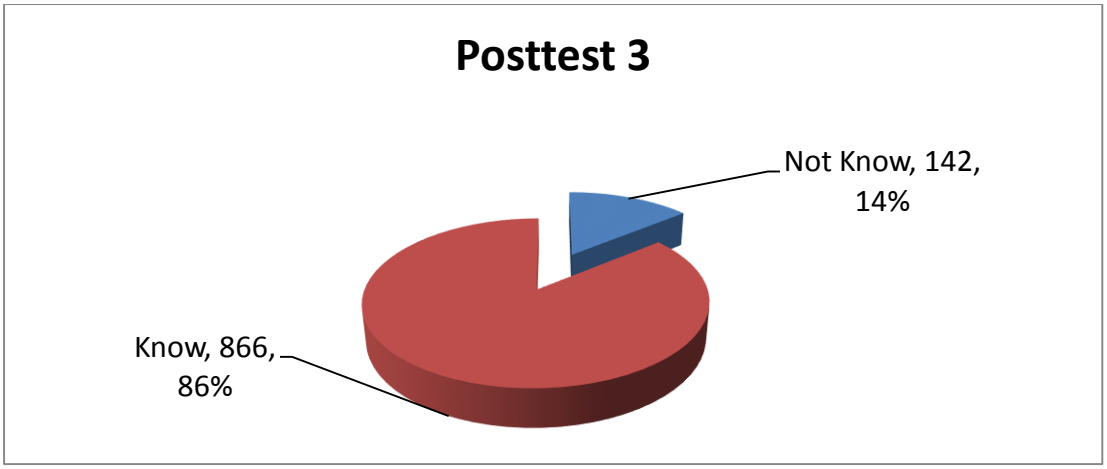


Figure (11): Pretest frequencies for basic knowledge, management and guidelines regarding pVT &VF

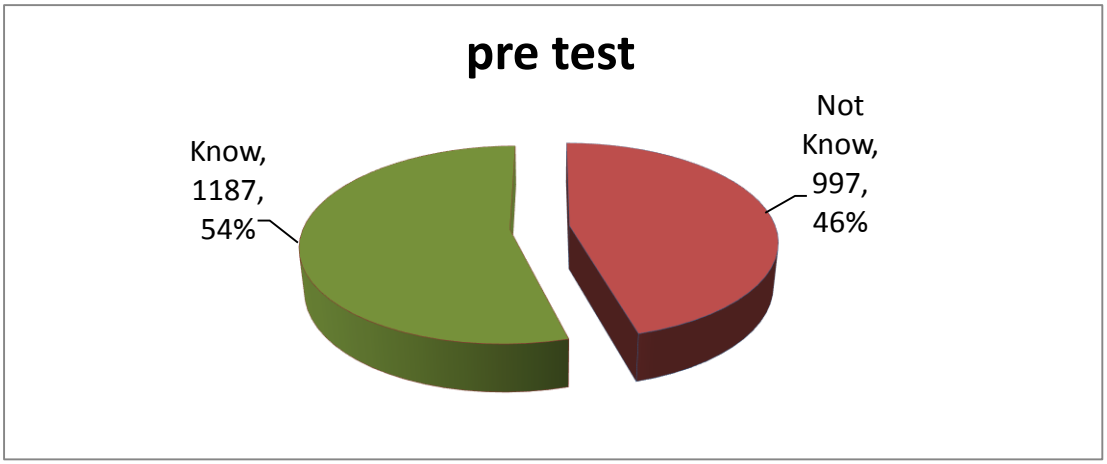


Figure (12): Posttest 1 frequencies for basic knowledge, management and guidelines regarding pVT &VF

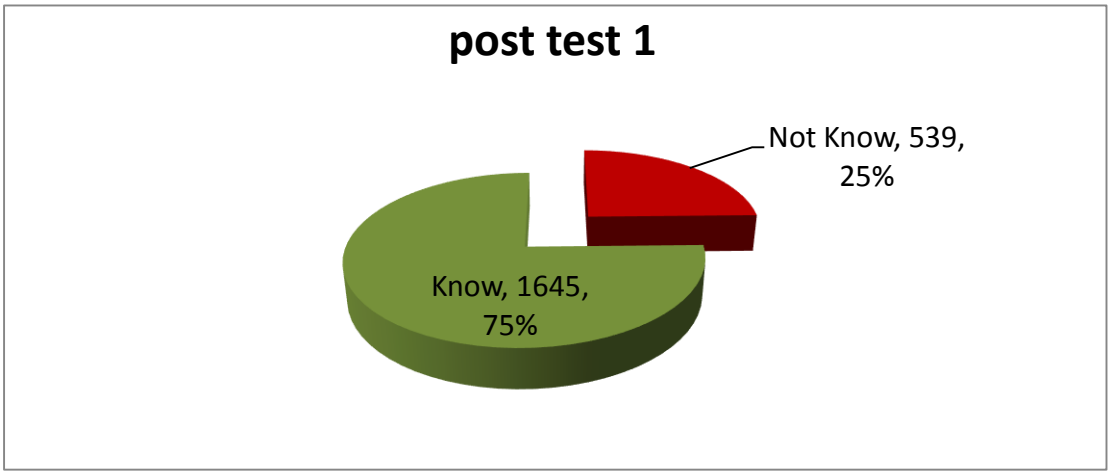


Figure (13): Posttest 2 frequencies for basic knowledge, management and guidelines regarding pVT &VF

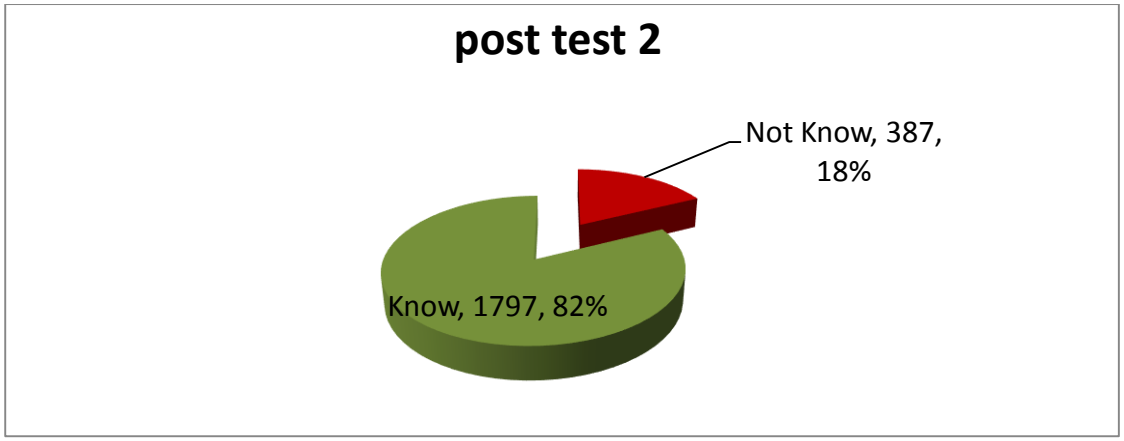


Figure (14): Posttest 3 frequencies for basic knowledge, management and guidelines regarding pVT &VF

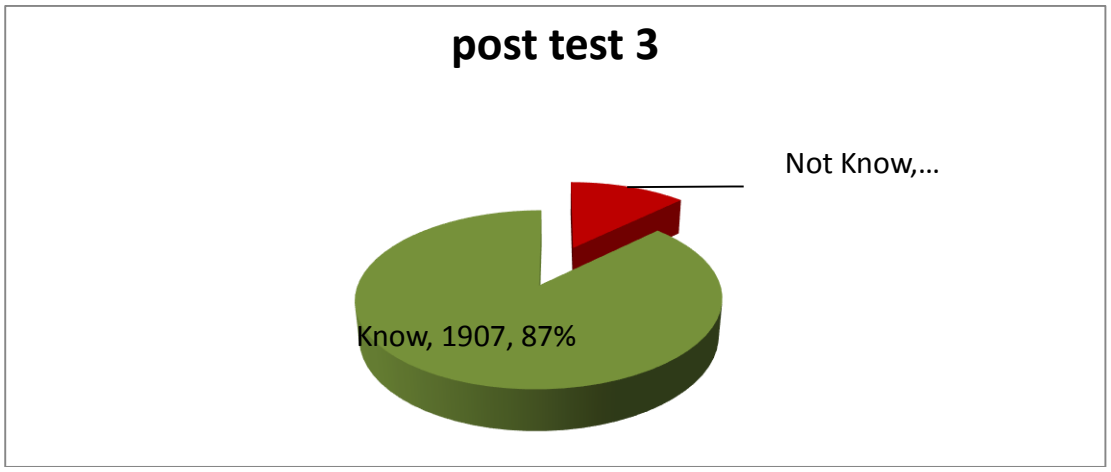


Table (6):

A comparison between nurses' knowledge regarding recognizing the Ventricular Tachycardia Ventricular Fibrillation from the ECG rhythm-strip in pre, post 1, post 2, and post 3 test of the implementation of the educational program

Ventricular Tachycardia rhythm	Not Know		Know		Total	
	F	%	F	%	F	%
Pretest Result	108	64%	60	36%	168	100%
Posttest 1 Result	57	34%	111	66%		
Posttest 2 Result	34	20%	134	80%		
Posttest 3 Result	37	22%	131	78%		
Ventricular Fibrillation rhythm						
Pretest Result	116	69%	52	31%	168	100%
Posttest 1 Result	64	38%	104	62%		
Posttest 2 Result	59	35%	109	65%		
Posttest 3 Result	52	31%	116	69%		

Figure (15):

Pretest frequencies for knowledge regarding recognizing of pVT &VF from ECG

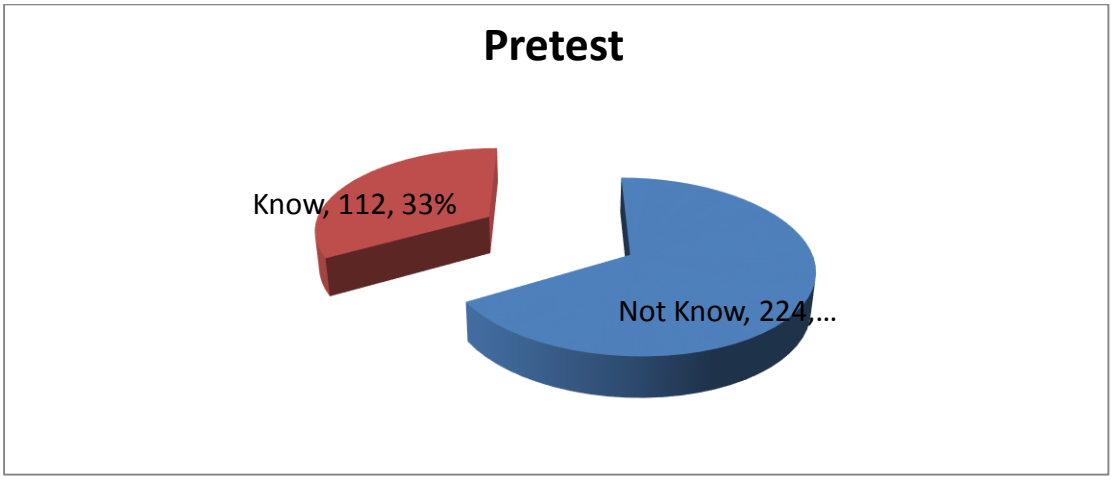


Figure (16):

Posttest 1 frequencies for knowledge regarding recognizing of pVT &VF from ECG

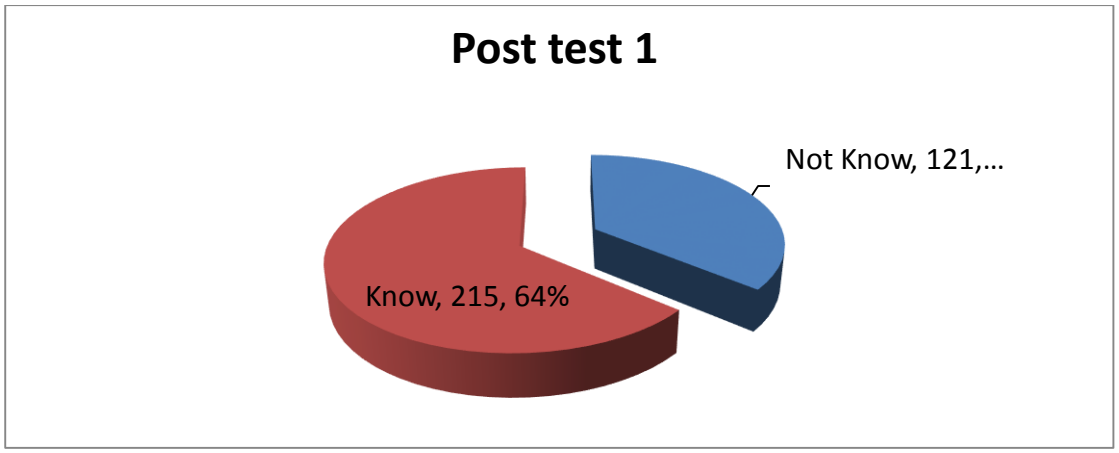


Figure (17):

Posttest 2 frequencies for knowledge regarding recognizing of pVT &VF from ECG

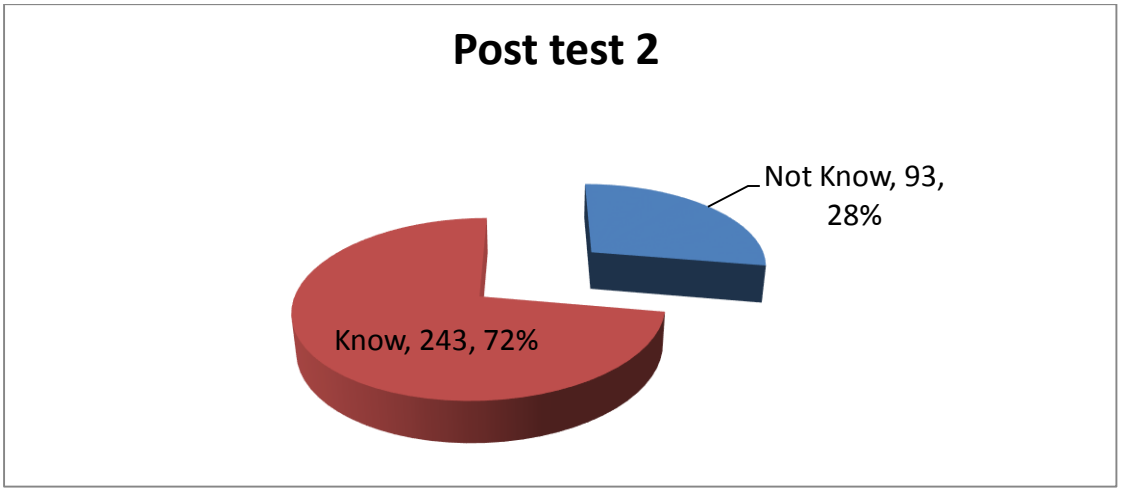


Figure (18):

Posttest 3 frequencies for knowledge regarding recognizing of pVT &VF from ECG

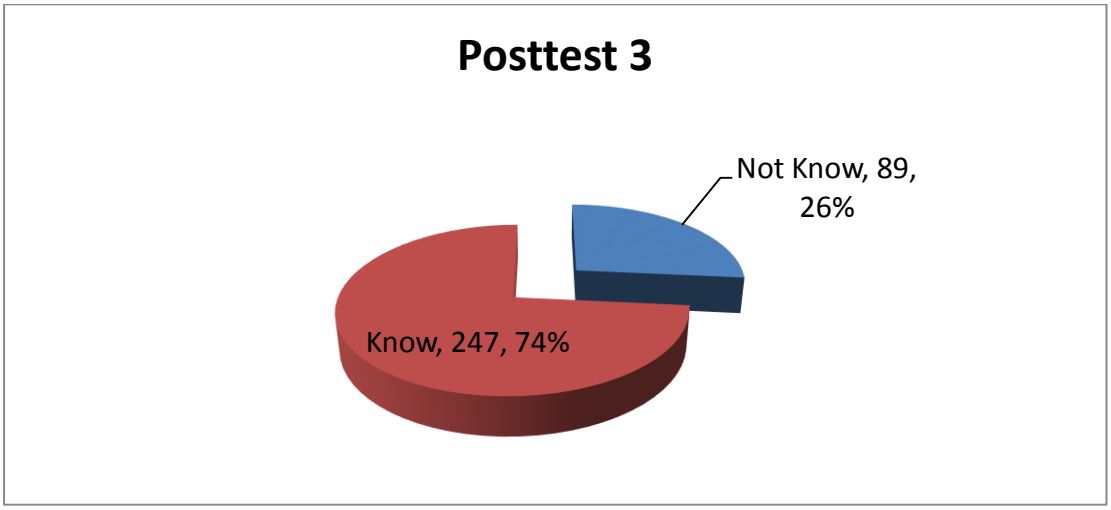


Table (7):

One-Sample Statistics pretest and posttest sample

One-Sample Statistics pre and post sample								
samples	N	Mean	Std. Deviation	direction	t	DF	Sig. (2-tailed)	Statistical declaration
pretest sample	168	.54	.17	Not know	3	167	(.001)**	Highly Sig
posttest sample1	168	.75	.15	Know	21	167	(.000)**	Highly Sig
posttest sample2	168	.82	.11	Know	37	167	(.000)**	Highly Sig
posttest sample3	168	.87	.09	Know	51	167	(.000)**	Highly Sig

Figure (19):

The mean of pretest sample and the mean of posttest 1 sample:

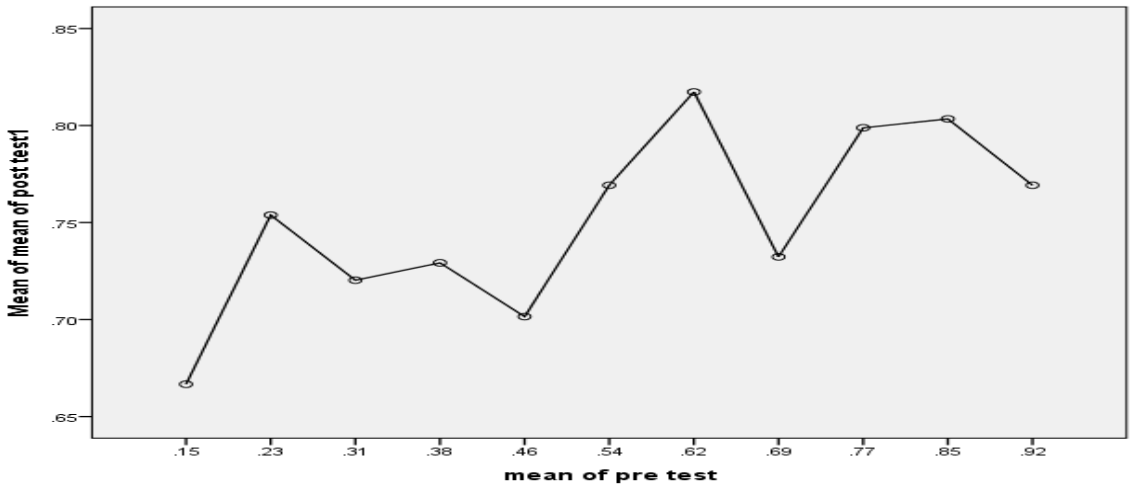


Figure (20):

The mean of pretest sample and the mean of posttest 2 sample:

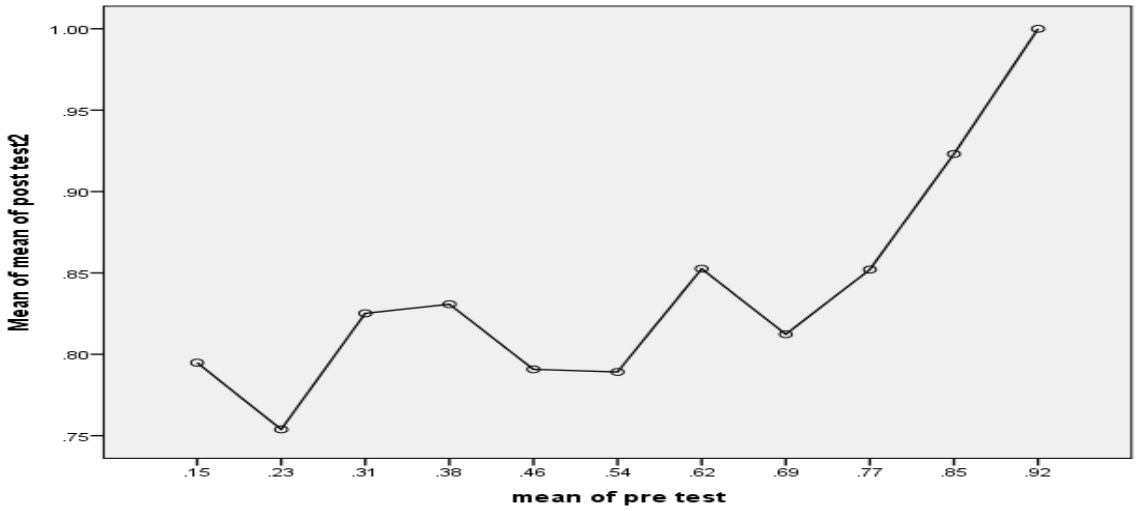
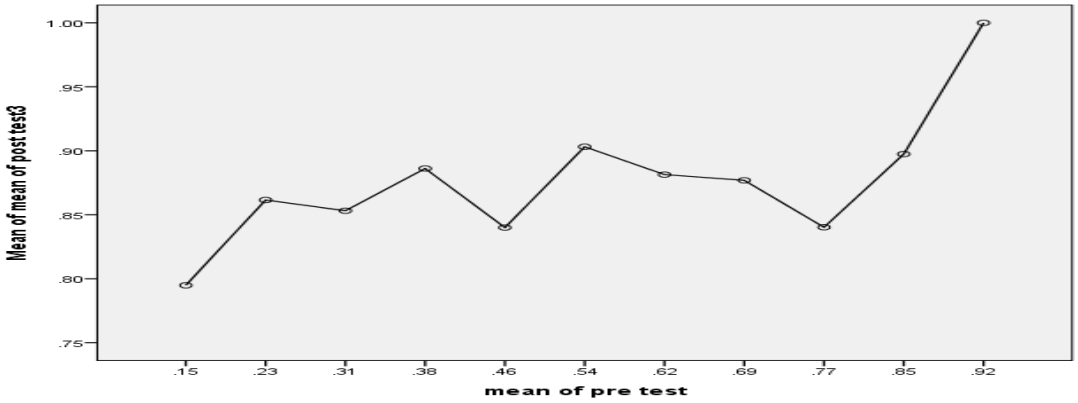


Figure (21):

The mean of pretest sample and the mean of post sample 3:



The Third part: The relation between the study variables

Table (8):

Frequencies between the gender and knowledge

Gender * Knowledge						
Gender	Knowledge					Total
	Very weak knowledge	weak Knowledge	Moderate knowledge	Good knowledge	Very good knowledge	
Male	1	9	21	69	52	152
Female	2	22	126	230	140	520
Total	3	31	147	299	192	672

Table (9):

Correlations between the gender and knowledge

Correlations	Gender & Knowledge
Correlation Coefficient	-.080*
Sig. (2-tailed)	.037
N	672

Table (10):

Chi-Square Tests between the gender and knowledge

Chi-Square Tests			
	Value	DF	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.980[a]	4	.062

Table (11):

Frequencies between the Age and knowledge

Age * Knowledge						
Age	Knowledge					
	Very weak knowledge	weak Knowledge	Moderate knowledge	Good knowledge	Excellent knowledge	Total
20 – 25 years	2	17	108	191	114	432
26 – 30 years	1	10	21	65	43	140
31 – 35 years	0	2	9	28	21	60
>36 years	0	2	9	15	14	40
Total	3	31	147	299	192	672

Table (12):

Correlations between the Age and knowledge:

Correlations	Knowledge & Age
Correlation Coefficient	.078*
Sig. (2-tailed)	.043
N	672

Table (13):

Chi-Square Tests between the Age and knowledge:

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	12.550[a]	12	.403

Table (14):

Frequencies between the Experience and knowledge

Experience years in unit * Knowledge						
Experience years in unit	Knowledge					
	Very weak knowledge	weak Knowledge	Moderate knowledge	Good knowledge	Excellent knowledge	Total
Less than 1 year	0	14	84	152	78	328
1 – 3 years	3	13	46	96	62	220
4 – 6 years	0	0	4	21	23	48
7 – 9 years	0	4	13	30	29	76
Total	3	31	147	299	192	672

Table (15):

Correlations between the Experience and knowledge

Correlations	Knowledge &Experience years in unit
Correlation Coefficient	.8123**
Sig. (2-tailed)	.001
N	672

Table (16):

Chi-Square Tests between the Experience and knowledge

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	28.436[a]	12	.005

Table (17):

Frequencies between the Degree of qualification and knowledge

Degree of qualification * Knowledge						
Degree of qualification	Knowledge					
	Very weak knowledge	weak Knowledge	Moderate knowledge	Good knowledge	Excellent knowledge	Total
Diploma	0	3	12	25	24	64
Bachelor	3	28	133	265	127	556
Post graduate	0	0	2	9	41	52
Total	3	31	147	299	192	672

Table (18):

Correlations between the Degree of qualification and knowledge

Correlations	Knowledge &Degree of qualification
Correlation Coefficient	.8144**
Sig. (2-tailed)	.000
N	672

Table (19):

Chi-Square Tests between the Degree of qualification and knowledge

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	76.809[a]	8	.000

Table (20):

Frequencies between the training program and knowledge

When you receive training program regarding (VF& Pulseless VT) Arrhythmia * Knowledge						
training	Knowledge					
	Very weak knowledge	weak Knowledge	Moderate knowledge	Good knowledge	Excellent knowledge	Total
Less than One year	1	5	33	65	68	172
Less than three years	0	5	18	34	23	80
More than Five years	0	0	1	6	9	16
Didn't receive training	2	21	95	194	92	404
Total	3	31	147	299	192	672

Table (21):

Correlations between the training program and knowledge

Correlations	Knowledge & training program
Correlation Coefficient	.7135**
Sig. (2-tailed)	.000
N	672

Table (22):

Chi-Square Tests between training program and knowledge

Chi-Square Tests			
	Value	DF	Asymp. Sig. (2-sided)
Pearson Chi-Square	25.368[a]	12	.013

2. **<u>checklist results:</u>**

Table (23):

The frequencies and percentages of the Pretest stage for the checklist

Pretest checklist		Not Done	not done correctly	Done correctly	Total
Confirm the cardiac arrest (ABC)	Frequency	103	49	16	168 100%
	Percent	61	29	10	
Call the resuscitation team	Frequency	107	39	22	
	Percent	64	23	13	
Perform uninterrupted chest compressions	Frequency	41	95	32	
	Percent	24	57	19	
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	Frequency	127	26	15	
	Percent	76	15	9	
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	Frequency	100	19	49	
	Percent	60	11	29	
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	Frequency	123	40	5	
	Percent	73	24	3	
The designated person selects the appropriate energy on the defibrillator and presses the charge button	Frequency	65	85	18	
	Percent	39	51	11	
Ensure that the rescuer giving the compressions is the only person touching the patient	Frequency	84	48	36	
	Percent	50	29	21	
Once the defibrillator is charged, tell the rescuer doing the CPR to “stand clear”; when clear, give the shock	Frequency	55	68	45	
	Percent	33	40	27	
After shock delivery immediately restart CPR using a ratio of 30:2	Frequency	125	35	8	
	Percent	74	21	5	
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	Frequency	109	43	16	
	Percent	65	26	10	
Pause CPR briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	Frequency	114	31	23	
	Percent	68	18	14	
If VF/pVT, repeat steps 6–12 above and deliver a second shock	Frequency	67	89	12	
	Percent	40	53	7	
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	Frequency	72	87	9	
	Percent	43	52	5	
Give adrenaline 1 mg IV	Frequency	91	35	42	
	Percent	54	21	25	
Amiodarone 300 mg IV while performing a further 2 min CPR	Frequency	129	35	4	
	Percent	77	21	2	
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	Frequency	82	76	10	
	Percent	49	45	6	
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	Frequency	125	37	6	
	Percent	74	22	4	

Table (24):

The mean, stander deviation and direction of the scale for the pretest checklist:

Pretest checklist	N	Mean	Std. Deviation	Direction
Perform uninterrupted chest compressions	168	1.95	.659	not done correctly
Once the defibrillator is charged, tell the rescuer doing the CPR to "stand clear"; when clear, give the shock	168	1.94	.772	not done correctly
The designated person selects the appropriate energy on the defibrillator and presses the charge button	168	1.72	.647	not done correctly
Ensure that the rescuer giving the compressions is the only person touching the patient	168	1.71	.798	not done correctly
Give adrenaline 1 mg IV	168	1.71	.843	not done correctly
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	168	1.70	.894	not done correctly
If VF/pVT, repeat steps 6–12 above and deliver a second shock	168	1.67	.604	not done correctly
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	168	1.63	.586	not done correctly
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	168	1.57	.605	not done correctly
Call the resuscitation team	168	1.49	.718	not done
Confirm the cardiac arrest (ABC)	168	1.48	.665	not done
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	168	1.46	.725	not done
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	168	1.45	.663	not done
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	168	1.33	.635	not done
After shock delivery immediately restart CPR using a ratio of 30:2	168	1.30	.555	not done
Resume CPR immediately; warn rescuers than the performing the CPR to "stand clear" and remove O2	168	1.30	.520	not done
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	168	1.29	.529	not done
Amiodarone 300 mg IV while performing a further 2 min CPR	168	1.26	.489	not done

Table (25):

The frequencies and percentages of the first posttest stage for the checklist

posttest checklist 1		Not Done	not done correctly	Done correctly	Total
Confirm the cardiac arrest (ABC)	Frequency	0	30	138	168
	Percent	0	18	82	
Call the resuscitation team	Frequency	0	47	121	
	Percent	0	28	72	
Perform uninterrupted chest compressions	Frequency	0	59	109	
	Percent	0	35	65	
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	Frequency	0	27	141	
	Percent	0	16	84	
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	Frequency	0	47	121	
	Percent	0	28	72	
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	Frequency	0	48	120	
	Percent	0	29	71	
The designated person selects the appropriate energy on the defibrillator and presses the charge button	Frequency	0	60	108	
	Percent	0	36	64	
Ensure that the rescuer giving the compressions is the only person touching the patient	Frequency	0	49	119	
	Percent	0	29	71	
Once the defibrillator is charged, tell the rescuer doing the CPR to stand clear; when clear, give shock	Frequency	0	59	109	
	Percent	0	35	65	
After shock delivery immediately restart CPR using a ratio of 30:2	Frequency	0	46	122	
	Percent	0	27	73	
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	Frequency	0	70	98	100%
	Percent	0	42	58	
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	Frequency	0	86	82	
	Percent	0	51	49	
If VF/pVT, repeat steps 6–12 above and deliver a second shock	Frequency	0	40	128	
	Percent	0	24	76	
If VF/pVT persists, repeat steps 6–9 & deliver third shock. Resume chest compressions immediately.	Frequency	0	64	104	
	Percent	0	38	62	
Give adrenaline 1 mg IV	Frequency	0	35	133	
	Percent	0	21	79	
Amiodarone 300 mg IV while performing a further 2 min CPR	Frequency	0	41	127	
	Percent	0	24	76	
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	Frequency	0	50	118	
	Percent	0	30	70	
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	Frequency	0	40	128	
	Percent	0	24	76	

Table (26):
The mean, stander deviation and direction of the scale for the posttest 1 checklist:

Posttest checklist 1	N	Mean	Std. Deviation	Direction
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	168	2.84	.368	Done correctly
Confirm the cardiac arrest (ABC)	168	2.82	.384	Done correctly
Give adrenaline 1 mg IV	168	2.79	.407	Done correctly
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	168	2.76	.427	Done correctly
If VF/pVT, repeat steps 6–12 above and deliver a second shock	168	2.76	.427	Done correctly
Amiodarone 300 mg IV while performing a further 2 min CPR	168	2.76	.431	Done correctly
After shock delivery immediately restart CPR using a ratio of 30:2	168	2.73	.447	Done correctly
Call the resuscitation team	168	2.72	.450	Done correctly
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	168	2.72	.450	Done correctly
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	168	2.71	.453	Done correctly
Ensure that the rescuer giving the compressions is the only person touching the patient	168	2.71	.456	Done correctly
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	168	2.70	.459	Done correctly
Perform uninterrupted chest compressions	168	2.65	.479	Done correctly
Once the defibrillator is charged, tell the rescuer doing the CPR to “stand clear”; when clear, give the shock	168	2.65	.479	Done correctly
The designated person selects the appropriate energy on the defibrillator and presses the charge button	168	2.64	.481	Done correctly
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	168	2.62	.487	Done correctly
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	168	2.58	.494	not done correctly
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	168	2.49	.501	not done correctly

Table (27):

The frequencies and percentages of the second posttest stage for the checklist

posttest checklist 2		Not Done	not done correctly	Done correctly	Total
Confirm the cardiac arrest (ABC)	Frequency	0	20	148	168
	Percent	0	11.9	88.1	100%
Call the resuscitation team	Frequency	0	44	124	
	Percent	0	26.2	73.8	
Perform uninterrupted chest compressions	Frequency	0	51	117	
	Percent	0	30.4	69.6	
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	Frequency	0	23	145	
	Percent	0	13.7	86.3	
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	Frequency	0	45	123	
	Percent	0	26.8	73.2	
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	Frequency	0	42	126	
	Percent	0	25.0	75.0	
The designated person selects the appropriate energy on the defibrillator and presses the charge button	Frequency	0	54	114	
	Percent	0	32.1	67.9	
Ensure that the rescuer giving the compressions is the only person touching the patient	Frequency	0	48	120	
	Percent	0	28.6	71.4	
Once the defibrillator is charged, tell the rescuer doing the CPR to “stand clear”; when clear, give the shock	Frequency	0	55	113	
	Percent	0	32.7	67.3	
After shock delivery immediately restart CPR using a ratio of 30:2	Frequency	0	42	126	
	Percent	0	25.0	75.0	
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	Frequency	0	55	113	
	Percent	0	32.7	67.3	
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	Frequency	0	40	128	
	Percent	0	23.8	76.2	
If VF/pVT, repeat steps 6–12 above and deliver a second shock	Frequency	0	33	135	
	Percent	0	19.6	80.4	
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume CPR immediately.	Frequency	0	59	109	
	Percent	0	35.1	64.9	
Give adrenaline 1 mg IV	Frequency	0	25	143	
	Percent	0	14.9	85.1	
Amiodarone 300 mg IV while performing a further 2 min CPR	Frequency	0	36	132	
	Percent	0	21.4	78.6	
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	Frequency	0	47	121	
	Percent	0	28.0	72.0	
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	Frequency	0	35	133	
	Percent	0	20.8	79.2	

Table (28):

The mean, stander deviation and direction of the scale for the posttest 2 checklist:

posttest checklist 2	N	Mean	Std. Deviation	Direction
Confirm the cardiac arrest (ABC)	168	2.88	.325	Done correctly
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	168	2.86	.345	Done correctly
Give adrenaline 1 mg IV	168	2.85	.357	Done correctly
If VF/pVT, repeat steps 6–12 above and deliver a second shock	168	2.80	.398	Done correctly
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	168	2.79	.407	Done correctly
Amiodarone 300 mg IV while performing a further 2 min CPR	168	2.79	.412	Done correctly
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	168	2.76	.427	Done correctly
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	168	2.75	.434	Done correctly
After shock delivery immediately restart CPR using a ratio of 30:2	168	2.75	.434	Done correctly
Call the resuscitation team	168	2.74	.441	Done correctly
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	168	2.73	.444	Done correctly
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	168	2.72	.450	Done correctly
Ensure that the rescuer giving the compressions is the only person touching the patient	168	2.71	.453	Done correctly
Perform uninterrupted chest compressions	168	2.70	.461	Done correctly
The designated person selects the appropriate energy on the defibrillator and presses the charge button	168	2.68	.468	Done correctly
Once the defibrillator is charged, tell the rescuer doing the CPR to “stand clear”; when clear, give the shock	168	2.67	.471	Done correctly
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	168	2.67	.471	Done correctly
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	168	2.65	.479	Done correctly

Table (29):

The frequencies and percentages of the third posttest stage for the checklist

posttest checklist 3		Not Done	not done correctly	Done correctly	Total
Confirm the cardiac arrest (ABC)	Frequency	0	25	143	168
	Percent	0	15	85	100%
Call the resuscitation team	Frequency	0	27	141	
	Percent	0	16	84	
Perform uninterrupted chest compressions	Frequency	0	29	139	
	Percent	0	17	83	
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	Frequency	0	15	153	
	Percent	0	9	91	
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	Frequency	0	27	141	
	Percent	0	16	84	
Resume CPR immediately; warn rescuers than the performing the CPR to "stand clear" and remove O2	Frequency	0	30	138	
	Percent	0	18	82	
The designated person selects the appropriate energy on the defibrillator and presses the charge button	Frequency	0	37	131	
	Percent	0	22	78	
Ensure that the rescuer giving the compressions is the only person touching the patient	Frequency	0	32	136	
	Percent	0	19	81	
Once the defibrillator is charged, tell the rescuer doing the CPR to "stand clear"; when clear, give the shock	Frequency	0	42	126	
	Percent	0	25	75	
After shock delivery immediately restart CPR using a ratio of 30:2	Frequency	0	30	138	
	Percent	0	18	82	
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	Frequency	0	45	123	
	Percent	0	27	73	
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	Frequency	0	18	150	
	Percent	0	11	89	
If VF/pVT, repeat steps 6–12 above and deliver a second shock	Frequency	0	17	151	
	Percent	0	10	90	
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	Frequency	0	42	126	
	Percent	0	25	75	
Give adrenaline 1 mg IV	Frequency	0	12	156	
	Percent	0	7	93	
Amiodarone 300 mg IV while performing a further 2 min CPR	Frequency	0	17	151	
	Percent	0	10	90	
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	Frequency	0	35	133	
	Percent	0	21	79	
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	Frequency	0	22	146	
	Percent	0	13	87	

Table (30):

The mean, stander deviation and direction of the scale for the posttest 3 checklist:

posttest checklist 3	N	Mean	Std. Deviation	Direction
Give adrenaline 1 mg IV	168	2.93	.258	Done correctly
Plan actions before pausing CPR for rhythm analysis and communicate these to the team	168	2.91	.286	Done correctly
If VF/pVT, repeat steps 6–12 above and deliver a second shock	168	2.90	.302	Done correctly
Amiodarone 300 mg IV while performing a further 2 min CPR	168	2.90	.302	Done correctly
Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse	168	2.89	.310	Done correctly
Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).	168	2.87	.338	Done correctly
Confirm the cardiac arrest (ABC)	168	2.85	.357	Done correctly
Call the resuscitation team	168	2.84	.368	Done correctly
Stop chest compressions; confirm VF/ pVT from the ECG and Pulse.	168	2.84	.368	Done correctly
Perform uninterrupted chest compressions	168	2.83	.379	Done correctly
Resume CPR immediately; warn rescuers than the performing the CPR to “stand clear” and remove O2	168	2.82	.384	Done correctly
After shock delivery immediately restart CPR using a ratio of 30:2	168	2.82	.384	Done correctly
Ensure that the rescuer giving the compressions is the only person touching the patient	168	2.81	.394	Done correctly
Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists	168	2.79	.407	Done correctly
The designated person selects the appropriate energy on the defibrillator and presses the charge button	168	2.78	.416	Done correctly
Once the defibrillator is charged, tell the rescuer doing the CPR to “stand clear”; when clear, give the shock	168	2.75	.434	Done correctly
If VF/pVT persists, repeat steps 6–9 and deliver a third shock. Resume chest compressions immediately.	168	2.75	.434	Done correctly
Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.	168	2.73	.444	Done correctly

Figure (22):

Pretest checklist frequencies for skills regarding implementation of standard guideline during management of pVT & VF

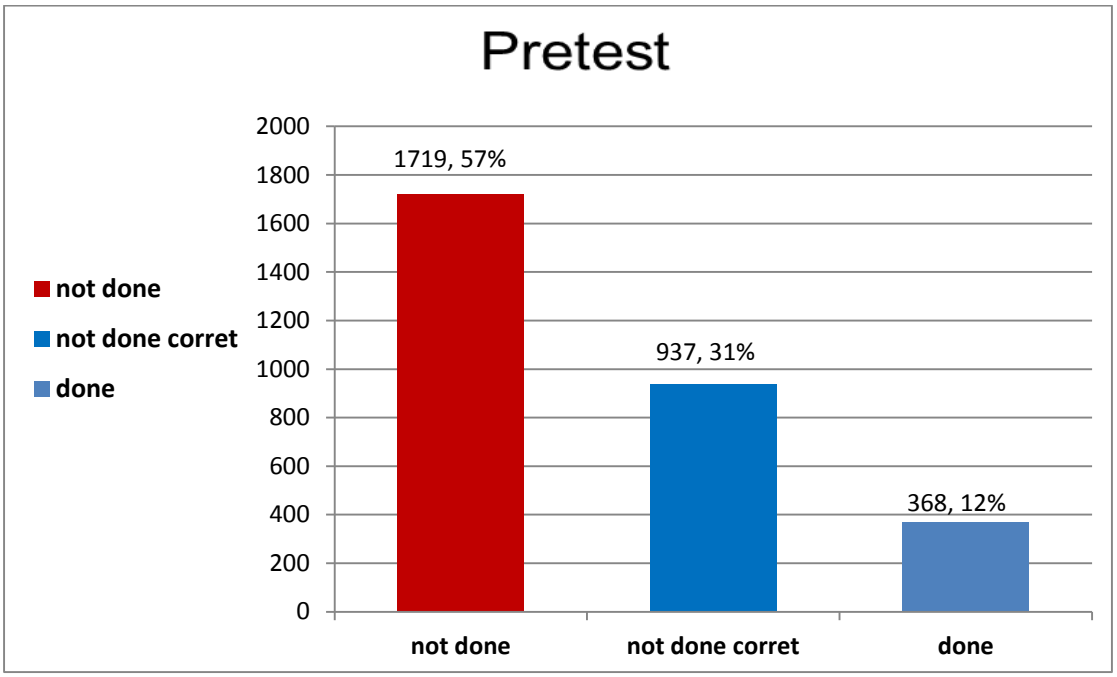


Figure (23):

Posttest1 checklist frequencies for skills regarding implementation of standard guideline during management of pVT & VF

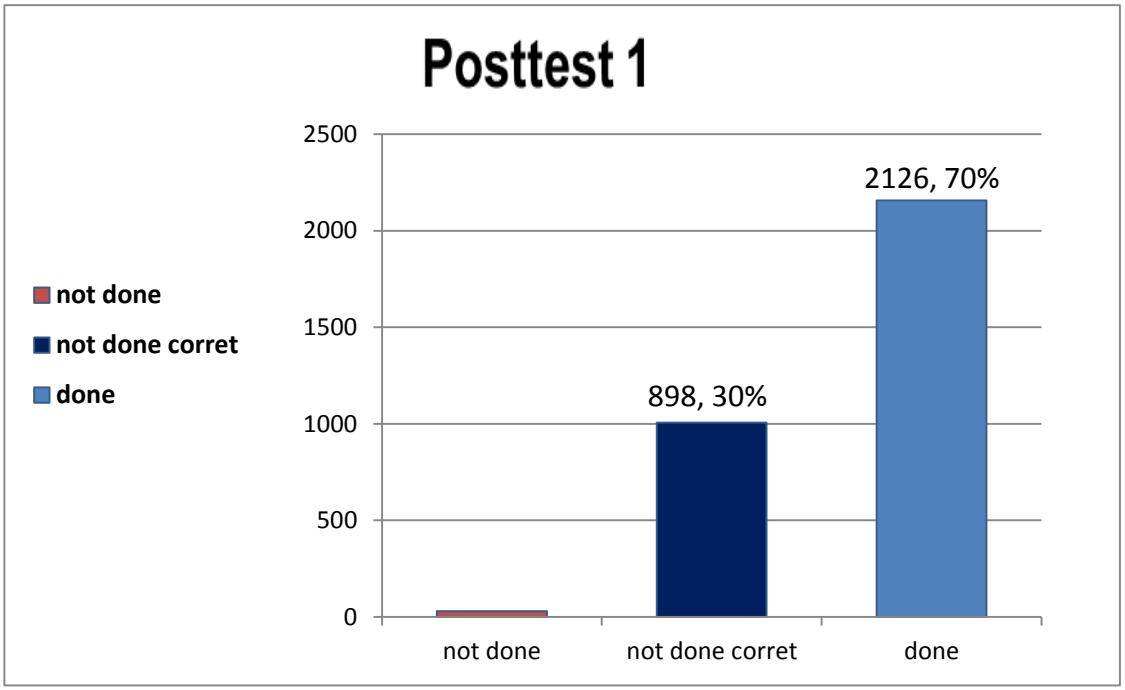


Figure (24):

Posttest 2 checklist frequencies for skills regarding implementation of standard guideline during management of pVT & VF

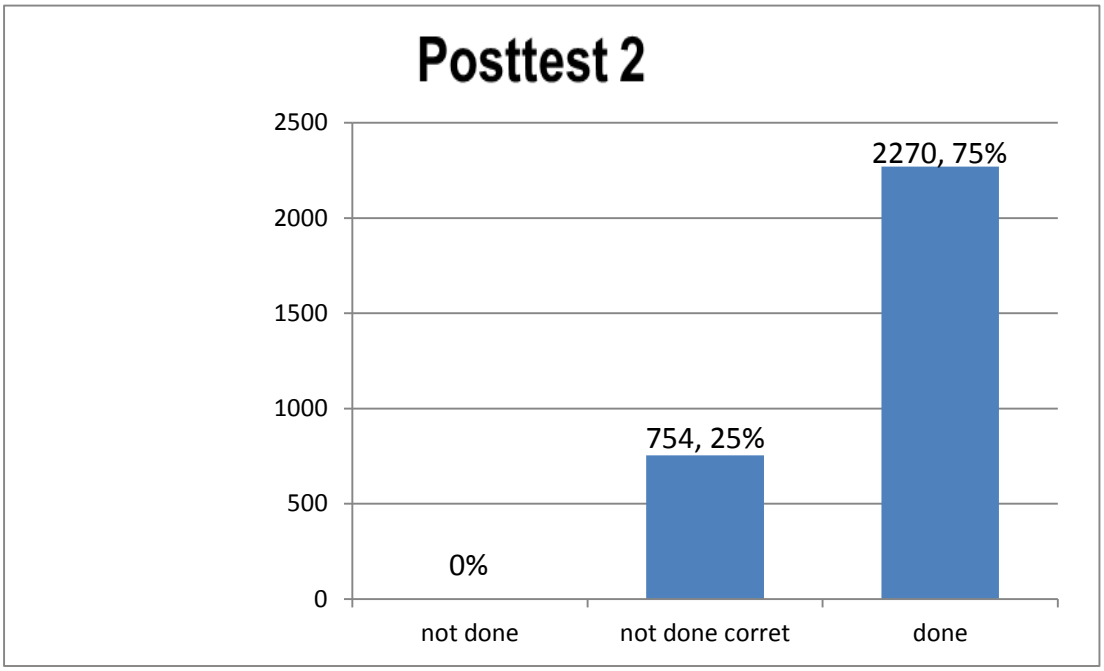
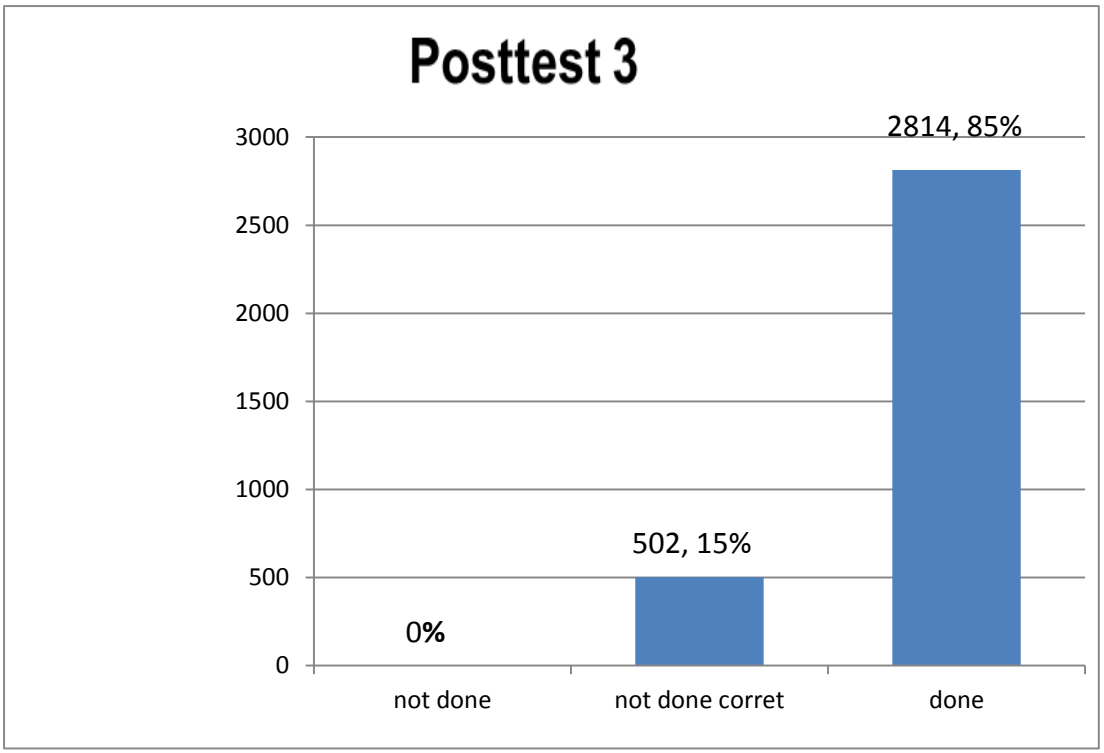


Figure (25):

Posttest 3 checklist frequencies for skills regarding implementation of standard guideline during management of pVT & VF



Test the hypothecs we use Paired sample (t) test with significant level (0.05)

Table (31):

Paired Samples Statistics for checklist

Paired Samples Statistics					
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	pretest checklist	1.44	168	.498	.038
	posttest checklist 1	2.79	168	.436	.034
Pair 2	posttest checklist 1	2.79	168	.436	.034
	posttest checklist 2	2.92	168	.277	.021
Pair 3	posttest checklist 2	2.92	168	.277	.021
	posttest checklist 3	3.00	168	0.000	0.000

Table (32):

Paired Samples Correlations for checklist

Paired Samples Correlations				
		N	Correlation	Sig.
Pair 1	pretest checklist & posttest checklist 1	168	-.044	.574
Pair 2	posttest checklist 1 & posttest checklist 2	168	-.095	.221
Pair 3	posttest checklist 2 & posttest checklist 3	168	-.000	.891

Table (33):

The mean and stander deviation of checklists

The mean and stander deviation to checklists				
	Mean	Std. Deviation	Std. Error Mean	Direction
pretest checklist	1.44	.498	.038	not done
posttest checklist 1	2.79	.436	.034	not done correctly
posttest checklist 2	2.92	.277	.021	Done correctly
posttest checklist 3	3.00	0.000	0.000	Done correctly

Table (34):

Paired Samples T Test for checklist

Paired Samples T Test									
		Paired Differences					T	DF	Sig. (2-ta)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	pretest checklist - posttest checklist 1	1.351	.676	.052	1.454	1.248	25.9	167	.000
Pair 2	posttest checklist 1- posttest checklist 2	.125	.538	.042	.207	.043	3.0	167	.003
Pair 3	posttest checklist 2- posttest checklist 3	.083	.277	.021	.126	.041	3.8	167	.000

CHAPTER FIVE

- Discussion
- Conclusion
- Recommendation

Discussion

The nursing care is important in survival and prevents the patients from Ventricular arrhythmias complications. Therefore, the competence of nurses about recognition and management of Ventricular arrhythmias is very crucial. The current study is effort to evaluate the Impact of an educational program on recognition and management of Ventricular arrhythmias (VF& pVT) guideline among critical care nurses. There are many studies related to the knowledge assessment of nurses regarding emergency management like cardiopulmonary resuscitation (CPR) but limited studies were conducted related to the knowledge assessment of nurses regarding ventricular arrhythmia (ventricular fibrillation/ pulseless ventricular tachycardia) recognition and management.

This study revealed that, among 168 critical care nurses (77 %) of study group were female, and (64 %) the median ages between (20-25) years, and only (6%) the median ages more than 36 years. Half of them (49 %) their years of experience less than one year, and just (11 %) have experience more than 7 years. which this finding means that the most of participants in this study who were working in critical care units are young, and not have long experience in nursing practice generally and especially in critical care units.

Moreover, the study indicated that the vast majority of nurses were bachelor holder (83%) whereas minimum numbers of the subjects were postgraduates (8%). The high percentage (60 %) did not receive in-service training course about recognition and management of pulseless VT and VF, (26%) were attend training program during last year. These findings indicated that nurses were working for many years which provide them to be expertise, but they were not exposed to any training course to enhance their skills or provide them with new guidelines. This clearly affects their performance and lead to poor outcome. This finding agreed with (*Lyneham et al)* conducted study to assess cardiopulmonary resuscitation knowledge among nurses working in Bahrain, the researcher find that in general, those who had less education and experience did not recall essential CPR knowledge. *Lyneham et al* study identified a significant problem with the knowledge surrounding CPR. More concerning was the lack of professional responsibility in dealing with this inadequacy [223].

Always in-service training is very important because it provides the nurses by updated guidelines and improving their skills in order to perform urgent response and effective management for pVT and VF patients. Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

guideline followed for many years and every five years this guideline reviewed and approved a new updated guideline. If nurses following this guideline from when having confirmed cardiac arrest, then throughout start CPR, up to delivered the defibrillation shocks safely, this will lead to effective management for pVT, & VF patient and shortly will return to his conscious level [224].

The present study revealed an increase in different knowledge aspects, and it was obvious that there was an improvement in nurses' knowledge in all posttest phases of an interventional program about the recognition and management of Ventricular arrhythmias (ventricular fibrillation/pulseless ventricular tachycardia). All the components of knowledge significantly improved in the first, second and third post-test (from 75%, to 82% and 87% in the third posttest) compared with (54%) in pretest evaluation. The fundamental knowledge regarding pulseless ventricular arrhythmia (ventricular tachycardia and ventricular fibrillation) significantly increased in posttest 1 (78%), to (84%) in posttest 2 and in posttest 3 the percentage was (89%) compared to this attribute in the pretest (57%). This finding disagreed with the study result submitted by (*Mohan Sanacy)*, 2010 who found that (88.88%) of nurses have above average level of knowledge regarding interpretation of life threatening arrhythmias and its emergency management [225]. While it has reinforced with (*Ayad M. Mousa, 2016)* study illustrated that the overall assessment of nurses' knowledge about VT was low; the high percentage 66.0% of nurses their knowledge less than 50% [226].

The nurses' knowledge showed low level of knowledge regarding the definition of ventricular arrhythmia (pVT &VF) question according to studied sample questionnaire. In addition, there was a poor knowledge regarding identify the main signs & symptoms. There was a deficient knowledge regarding common cause, diagnostic method, complication and prevention of ventricular arrhythmia (pVT &VF) according to sample questionnaire. In general, the overall assessment of samples basic knowledge was low in pretest stage. In accordance with this result, this study indicate that there is needed to improve the nurses' knowledge regarding fundamental knowledge about ventricular arrhythmia (pVT VF) such as definition, causes, signs, symptoms, complications and preventions of this arrhythmia.

The nurses' knowledge about the definition and signs & symptoms of ventricular arrhythmia (pVT &VF) significantly increased after the educational program. On the other hand, the intervention program enhanced the knowledge about the common cause, diagnostic method, complication and prevention of ventricular arrhythmia (pVT &VF) in the first, second and third posttest. In

concordance with this result, an experimental study was conducted on "effects of implementing nursing care standards for nurses caring for patients with cardiac arrhythmia" in Saudi Arabia. The sample includes 40 nurses working in Intensive coronary care unit. The study concluded that the majority of nurses had unsatisfactory knowledge and practice before implementation of standards and after applying the standards there were improvement [227]. In addition, Many acutely ill patients have 12 lead ECG recorded either on admission to hospital, either undergoing operations or when specific cardiac concerns have arisen. Traditionally, ECG have aided clinically diagnoses and, by learning how to understand and interpret them, nurse will know when they need to summon expert help (*Woodrow, 2010*) [228]. In critical care settings, ECG's provide nurses with information about the patient's electro-cardiac record in a manner that is easy and fast to use. Nurses who care for critically ill patients need to have a thorough understanding of the information provided by the ECG, which includes heart rate, regularity of the rhythm, interval measurements and characteristics of each individual waveform of the heart. Moreover, the life -threatening ventricular tachyarrhythmias such as VT or VF are likely to develop during the acute phase of ACS, and the occurrence of these arrhythmias has important effects on the prognosis of these patients [229].

In a comparison between nurses' knowledge in pre, post I, II, and post intervention III regarding the management of ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) based on the standard guidelines. The overall result shown that the half of participants in this program have poor knowledge about the management of ventricular arrhythmia in pretest evaluation, sequentially and dramatically changed to very good (86%) in posttest III phase. Which was remarkably pushing the concept of the important of the training program, and this result was clinically important because the good nursing management for ventricular arrhythmias should be done based on updated standard clinical guidelines. This finding was agree with study was conducted on "incidence and risk factors of arrhythmic events in catecholaminergic polymorphic ventricular tachycardia" in France. The study concluded that there is an increased incidence rate of cardiac arrhythmia and appropriate management is necessary to improve the patient outcome. [230] Also it was agree with study was conducted on "treatment of ventricular tachycardia in patients with heart failure" in USA. The study concludes that ventricular tachycardia is a life threatening condition and it requires immediate treatment and effective management. [231] Moreover, another study recommended that Nurses should have knowledge, experience and information toward patient with arrhythmia and specifically with VT because the patient is usually

(although not always) unresponsive and pulseless and need critical care that provided by skilled work team. [232]

In this study (38%) have good knowledge score regarding identification of the first step that nurse will do it when see ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) in the monitor in pre intervention stage. The result improved to becomes (64%) in the first post intervention, then improve more in the second posttest the percentage reached eighty two, and in the third posttest the result decreased to (79%), this findings coincided with (*Bhat et al*) study regarding the awareness of (BLS) among students, doctors and nurses of medical, dental, homeopathy and nursing colleges. The results were analyzed using an answer key prepared with the use of the Advanced Cardiac Life Support manual; no one among them had complete knowledge of BLS, majority of them (84.82%) had secured less than 50% marks. Awareness of BLS among students, doctors and nurses of medical, dental, homeopathy and nursing colleges is very poor. [233] But the results differ than "Life threatening arrhythmias: Knowledge and skills among nurses working in critical care settings at Muhimbili National Hospital", in Tanzania, the majority of the participants scored highly in their level of knowledge regarding life threatening arrhythmias, they scored poorly in most of the observed skills when identifying and treating this patient group [140].

In addition, (55%) have good level of knowledge regarding the useful medication for management of ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) in pretest. The result which changed to (70%) in posttest one, posttest two showed more improvement (76%), and in the posttest three shows great result (87%) have good knowledge about the useful medication for management of ventricular arrhythmias patients. Nevertheless, in pre-intervention assessment (57%) of nurses have poor level of knowledge regarding when the nurse will start to use adrenaline during treating the ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) patients, in post intervention 1 showed decreased to (25%) and decreased more in post intervention 2 to (22%) , and in the third post intervention the percentage reached (10%). The result showed the important of this program in reducing the poor knowledge regarding when the nurse will start adrenaline while treating the ventricular arrhythmias patient. This finding was collision with Cleary study among critical care nurses regarding initiation and administration of drugs for advanced life support in the absence of a medical practitioner, the results showed that the majority of nurses underwent an annual ALS

assessment and had current ALS accreditation. Nurses indicated that they felt educationally prepared and were confident to manage cardiopulmonary resuscitation without a medical officer; indeed, the majority had done so. The differences in practice issues for metropolitan, regional and rural nurses were highlighted. There is therefore clear evidence to suggest that amendments are appropriate and necessary, given the time critical nature of cardiopulmonary arrest [234].

The appropriate energy by using the biphasic defibrillator in the second & subsequent shock revealed that the nurses good knowledge was (40%) in pre intervention test, and showed improvement in post intervention I by (69%), post intervention II improved by (73%), and post intervention III reached to (86%). On the other hand, the nurses' knowledge regarding the immediate management for the ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) showed the correct answer were chosen by (71%) in pre intervention, (78%) in post intervention I, (86%) in post II, and (94%) in post III this good finding. This may be refers to that the immediate management for the ventricular arrhythmias is clear for critical care nurses. Moreover, the response regarding the long-term treatment for the patient has recurrent ventricular arrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) was good (59%) pre intervention, (76%) in post intervention I, (84%) in post intervention II and decreased to (80%) in post intervention III. The only intervention demonstrated to improve survival in patients at risk of SCD from ventricular arrhythmias is the implantable cardioverter defibrillator (ICD). It has indicated for secondary prevention in patients with a history of sustained VT/VF [48].

The nurses' knowledge showed low level (33%) regarding interpret and recognize the ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation) from the ECG rhythm-strip in pretest phase. In the first posttest the level jumped to (64%), then improve more to seventy two percent in the second posttest, and in the third posttest three quarter of participants were interpret and recognize the correct ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation) from the ECG rhythm-strip. The majority of the participant (64%) were not recognize the ventricular tachycardia (VT) rhythm from the ECG rhythm-strip in the pretest, were (36%) recognized the correct rhythm, and the result was improved to (66%) in post intervention I and changed to (80%) in post II, but in post intervention III decreased to (78%). In the same line, the result of the recognition of ventricular fibrillation (VF) rhythm from the ECG rhythm-strip showed (31%) as correct answer in pre intervention, (62%) in posttest I, (65%) in posttest II, and (69%) in posttest III. This result was frustrating because the recognition of

ventricular arrhythmias from the ECG rhythm-strip is very important and has direct effect to ventricular arrhythmias management and decrease the mortality. as recommended in one study was conducted in USA, this study concludes that electrocardiographic imaging can map the ventricular tachycardia activation sequence and identify the location and depth of ventricular origin in individual patients, allowing personalized treatment of patient with ventricular arrhythmia [49].

These findings get along to *(Florida et al)* study using a focus methodology to elicit the perceptions of nurses knowledge about the level of knowledge needed to recognize a cardiac arrhythmia. This study revealed a deficit in nurse's ability to recognize and identify specific arrhythmias including heart block, aberrant conduction, and tachyarrhythmia. [235]

On the other hand, the statistic results such as (mean, Std. deviation, t test and 2-tailed test) showed that the mean knowledge score at the post intervention phases was significantly higher than at the pre intervention for the study group. The greeter mean is post intervention III (.87) with STD Deviation (.09), the direction is (know), Sig. (2-tailed) of t test is (.000) is highly significant. The pre intervention result showed that the mean of pretest sample (.54) with STD Deviation (.17), the direction is (not know), Sig. (2-tailed) of t test is (.000) is highly significant.

In addition, the relation between the study variables revealed that there is independence relation between the gender and knowledge the Pearson Chi-Square was (0.062), there is independence relation between the age and knowledge Pearson Chi-Square was (0.403). Nevertheless, there is a strong statistically significant association between the degree of qualification and knowledge Correlation Coefficient is (.000), there is a strong statistically significant association between the training and knowledge Pearson Chi-Square was (0.013), moreover the duration of experience has a strong statistically significant association with the knowledge the sig of Correlation Coefficient is (.001).

The nursing skills were studied and tested based on the implementation of standard guidelines for the management of patients with ventricular arrhythmias (Pulseless Ventricular Tachycardia and Ventricular Fibrillation). The collected data after statistically tested showed markedly an improvement in the score from pre intervention to post intervention.

Overall result as shown in figure (22) regarding pre intervention frequencies, (57%) did not implement the most of standard guideline steps, (31%) did not implemented correctly, and only (12%) implemented correctly. In the first post intervention frequencies, the vast majority of

the participant (70%) were implemented these steps correctly, while (30%) did not implemented correctly. Three quarter of frequencies in the second post intervention phase were implemented the skills correctly and one quarter not implemented correctly, and zero percent not implement. Regarding the third post intervention result, shown that (85%) of frequencies were implemented the skills correctly and (15%) not implemented correctly, and (0%) of frequencies were not implement the standard guideline steps. These findings illustrate the significant change post this intervention program has made which shows the importance of this program.

In a comparison between nurses' skills in pre, post I, II, and post intervention III regarding the management of pulseless ventricular tachycardia and ventricular fibrillation. The main findings were as follow:

Confirmation of the cardiac arrest by using the (ABC) approach in pre intervention was not done by (61%) of participants, in post intervention III (0%) were not done, (15%) were not done correctly, and (85%) was done correctly. And calling the resuscitation team done by 22 participants (13%) in pre intervention phase, improved to (72%) in post intervention I to (74%) in posttest II and to (84%) in post intervention III.

Performing uninterrupted chest compressions not done correctly by 95 participants (57%) in pre intervention phase, decrease to (35%) in post intervention I decrease more to (30%) in posttest II and reach (27%) in post intervention III. While Planning actions before pausing CPR for rhythm analysis and communicate these to the team, in pre intervention phase was not done by (76%) of nurses, after the intervention program the percentage reaches Zero.

Stop chest compressions to confirm VF/ pVT from the ECG and Pulse was done correctly by one third of the critical care nurses in pretest; improve to two third in posttest I and II, and in the last posttest, the percentage reached more than eighty. Moreover, one hundred twenty three participants did not resume CPR immediately and warn rescuers than the performing CPR to "stand clear" and remove O2 in pre intervention phase, and the frequency decreased to zero in posttest phases.

The designated person selects the appropriate energy on the defibrillator and presses the charge button done correctly only by 18 participants in pretest. Improved to 108 participants in the first posttest, then to 114 participants in the second posttest, and improved more to 131 participants in

the third posttest phase. Whilst half of critical care nurses did not ensure that the rescuer giving the chest compressions is the only person touching the patient, only one-fifth of them do it correctly in pre intervention, in post intervention III not done by critical care nurses become zero, and more than four-fifths of them do it correctly.

Once the defibrillator is charged, tell the rescuer doing the CPR to "stand clear"; when clear, give the shock was not done correctly by forty percent of participant in pre intervention, in post intervention III decreased to twenty five percent while seventy five percent was do it correctly. Notwithstanding, after shock delivery immediately restart CPR using a ratio of 30:2 was done only by eight participants (5%) in pre intervention phase, improved to (73%) in post intervention I to (75%) in posttest II and to (82%) in post intervention III.

Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR, were not done by 125 participants (65%) in pre intervention phase, decreased to zero percent in post intervention I, II, and III. On the other hand, two-third of participant did not Pause CPR briefly to check the monitor, confirm VF/pVT from the ECG and Pulse in pre intervention phase and in post intervention III become zero percent, and who were do it correctly the percentage reached (89%). If VF/ p VT, repeat steps 6–12 above and deliver a second shock was done by (7%) of the nurses in pretest; improved to (76%) in posttest I and improved more to (80.4%) posttest II, and in the last posttest, the percentage reached (90%). In same line, if VF/p VT persists, repeat steps 6–9 and deliver a third shock and resume chest compressions immediately in pre intervention phase was not done by (73%) of nurses, after the intervention program the percentage reach Zero.

Give the patient adrenaline 1 mg IV done by 42 participants by (25%) in pre intervention phase, improved to (79%) in post intervention I to (85.1%) in posttest II and to (93%) in post intervention III. Whereas Pre intervention the percentage of nurses who do not give amiodarone 300 mg IV were (77%) and only (2%) who were correctly give amiodarone. In post intervention I (76%) were give amiodarone improved to (78.6%) in post intervention II, and post intervention III (90%) of nurses were correctly give amiodarone 300 mg, and (0%) do not give amiodarone 300 mg in all post interventions phase I, II, III.

Fifty percent of participant did not repeat 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists and the frequency decreased to zero in posttest phases in pre intervention phase. In addition give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min) were given by 6 critical care nurses (4%) in pretest phase, improved

to (76%) in the first posttest, improved more to (79.2%)in the second posttest, and in the third posttest the percentage reached (87%).

Table (33) revealed that the mean of pretest checklist is (1.44) direction (not done) with stander deviation (0.498). The mean of posttest checklist1 is (2.79) direction (not done correctly) with stander deviation (0.436). The mean of posttest 2 is (2.92) direction (don correctly) with stander deviation (0.021). The mean of posttest 3 is (3) direction (don correctly) with stander deviation (0.00).

Table (34) shown that the mean deferent between pre and post intervention I checklist is (1.351) with stander deviation (0.676) and PV (0.000) it is less than (0.05) highly significant, the mean increase in post intervention checklist 1. The pair 2 shown the mean deferent between the first post intervention & the second post intervention checklist is (0.125) with stander deviation (0.538) and PV (0.003) it's less than (0.05) highly significant, the mean increase in posttest checklist 2. Furthermore the mean deferent between the second post intervention checklist & the third post intervention checklist is (0.083) with stander deviation (0.277) and PV (0.000) it is less than (0.05) highly significant, the mean increase in post intervention checklist 3.

Conclusion

- The study shows that there was strong relationship between knowledge score and degree of qualification, receiving training, and duration of experience, while there is poor relationship between other demographic variables (gender, and age), and sample knowledge scores.
- The study shows that there was highly statistically significant association between the nurses' performance and implementation of this program in post I, post II and posttest III.
- The nurses' knowledge regarding the ventricular fibrillation and pulseless ventricular tachycardia about the basic knowledge and the management guidelines was improved after implementation of this interventional program.
    I. There was an increase in good knowledge regarding ventricular fibrillation and pulseless ventricular tachycardia about the definition, causes, signs & symptoms, etc.
    II. Nurses' knowledge regarding recognition of ventricular fibrillation and pulseless ventricular tachycardia from ECG increased after implementing the intervention program from weak in pre intervention to good post intervention III.
    III. There was an improvement in the nurses' knowledge regarding the management of pulseless ventricular tachycardia and ventricular fibrillation based on the standard guidelines.
- Overall nurses' skills regarding the ventricular fibrillation and pulseless ventricular tachycardia management guidelines improved from very week pre intervention, to good in post intervention I & II, to very good in post intervention III.
    I. There was an increase in mean of good skill of using ABC approach to confirm the cardiac arrest.
    II. There was improvement in nurses skills regarding perform uninterrupted chest compressions.
    III. There was obvious improvement in effective and safety use of manual defibrillator.
    IV. There was improvement in follow the medication administration policy during Pulseless VT &VF management.

Recommendation

Based on the finding of this study the researcher recommended that:

- Specific courses about the recognition and management of ventricular arrhythmias (ventricular fibrillation and pulseless ventricular tachycardia) must be establish for critical care nurses to enhance them to be more knowledgeably and skillfully in recognition and management of ventricular arrhythmias.
- Policies and regulations from Ministry of Public Health and Sudanese national council for medical and health professions should maintain optimum level of nurses' knowledge and skills regarding ventricular arrhythmias recognition and management, especially for critical care nurses.
- Continuing Professional Development training center (CPD) need to consider paying more attention about ECG interpretation especially about life threatening arrhythmias and management of ventricular arrhythmias patients.
- On the other hand, hospital management need to have a clinical nurse trainer to support and encourage nurses to translate acquired knowledge into practical skills, and designing and implementing CPD programs about ventricular threatening arrhythmia recognition and management.
- Besides that, there is need to have more staff that are senior in the critical care units, and to have Arrhythmia Care Coordinator (ACC), to guide patients through their illness and coordinate their care.
- Finally, more researches about nursing role in arrhythmias recognition and management should be encouraged, this research is the second and I hope it is not going to be the last.

APPENDIX

- References
- Research tools
- Teaching program

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بسم الله الرحمن الرحيم

SHANDI UNIVERSITY

FACULTY OF POSTGRADUATEAND SCIENTIFIC RESEARCH

ENDORSEMENT

I am Musab Fadlalla I working in primary health care Qatar State. I am conducting this research to assess the impact of an educational program on recognition and management of ventricular arrhythmias guideline among critical care nurses in Khartoum State. I greatly appreciate your participation in this research. The information I collect from you through questionnaires will help to design training policies and programs for the effective treatment of life-threatening arrhythmias in hospitals. The program you will receive help increase your knowledge and develop your skills, allowing you to learn how to recognize and treat life-threatening arrhythmias in hospitals. The questionnaire takes approximately 15 minutes to complete. The information you will write confidential and your answers will not be disclose to anyone who is not a researcher and none of your answers will be share with anyone inside or outside the hospital. Your participation will be as one of the volunteers in this research.

Do you have any questions about the search and the program?

……..

Name and number of the researcher: Musab Mohamed Ahmed Ali Fadlalla 0128467549

Agree ( ) Disagree ( )

PART ONE:

- Gender:-
  a) Male
  b) Female
- Age:-
  a) 20 – 25 years
  b) 26 – 30 years
  c) 31 – 35 years
  d) >36 years
- Experience years in unit:-
  a) Less than one year
  b) 1 – 3 years
  c) 4 – 6 years
  d) 7 – 9 years
  e) >10 years

- Degree of qualification:-
    a) Diploma
    b) Bachelor
    c) Post graduate
- When you receive training program regarding (VF& Pulseless VT) Arrhythmia recognition and management:-
    a) Less than One year
    b) Less than three years
    c) More than Five years
    d) I did not receive any training

PART TWO:

Choose the most correct answer:-

1) (VF& Pulseless VT) Arrhythmia means-
    a) Fast atrium rate whether regular or irregular
    b) Chest pain
    c) Palpitation
    d) Fast ventricular rate whether regular or irregular

2) The common cause of (VF& Pulseless VT) arrhythmias is-
    a) Electrolyte disturbance
    b) Coronary artery disease
    c) Cardiomyopathy
    d) All correct

3) The main signs of (VF& Pulseless VT) arrhythmias is:-
    a) Low oxygen saturation
    b) Syncope
    c) Chest pain & Palpitations
    d) ECG changes & pulse absent

4) The main symptoms of (VF& Pulseless VT) arrhythmias is:-
    a) Low oxygen saturation
    b) Syncope
    c) Chest pain & Palpitations
    d) ECG changes & pulse absent

5) The useful diagnostic method for (VF& Pulseless VT) arrhythmias is-

a) Check cardiac enzyme
b) Chest X-ray
c) An ECG monitoring
d) Echocardiography

6) The common complication of (VF& Pulseless VT) arrhythmias is:

a) Stroke
b) Heart failure
c) Sudden Cardiac Death (SCD)
d) Plural effusion

7) Prevention of (VF& Pulseless VT) arrhythmias by:

a) Stop smoking& controlling cholesterol level
b) Physical exercise
c) Avoid crowded places and sugar intake
d) Maintain good fluid & nutrition intake

8) What is the first step you will do it when you see (VF& VT) arrhythmias in monitor:-

a) Check electrolyte
b) Check cardiac enzyme
c) Check the pulse
d) Make ECG

9) The useful medications for management of (VF& Pulseless VT) arrhythmias is-

a) Digoxin
b) Amiodaron
c) Atropine
d) Captopril

10) When we start adrenaline for patient with (VF& Pulseless VT) arrhythmias:-

a) After First shock
b) After Second shock
c) After Third shock
d) After Fifth shock

11) The appropriate energy by using the biphasic defibrillator in the second & subsequent shock is-

a) 100 joule
b) 200 joule
c) 300 joule
d) 400 joule

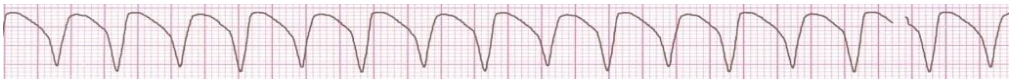
12) The immediate management for (VF& Pulseless VT) arrhythmias is-

a) Cardiopulmonary resuscitation (CPR) ONLY
b) Defibrillation ONLY
c) Defibrillation then Cardiopulmonary resuscitation (CPR)
d) Coronary bypass surgery (CABG)

13) The long-term treatment for the patient with recurrent (VF& Pulseless VT) is:

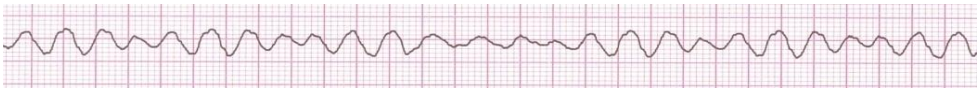
a) Cardiopulmonary resuscitation (CPR)
b) Pacemakers
c) Coronary bypass surgery (CABG)
d) ICD (implantable cardioverter-defibrillator)

14) This rhythm is:



a) Ventricular Fibrillation (VF)
b) Asystole
c) Ventricular Tachycardia (VT):
d) Supra-Ventricular Tachycardia (SVT)

15) This rhythm is:



a) Ventricular Fibrillation (VF)
b) Asystole
c) Ventricular Tachycardia (VT):
d) Supra-Ventricular Tachycardia (SVT).

Check List

Steps Treatment of shockable rhythms (VF/pVT)	DC	NDC	ND
1. Confirm cardiac arrest (ABC)			
2. Call resuscitation team.			
3. Perform uninterrupted chest compressions			
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.			
5. Stop chest compressions; confirm VF/pVT from the ECG and Pulse.			
6. Resume chest compressions immediately; warn all rescuers other than the individual performing the chest compressions to "stand clear" and remove any oxygen delivery device as appropriate.			
7. The designated person selects the appropriate energy on the defibrillator and presses the charge button.			
8. Ensure that the rescuer giving the compressions is the only person touching the patient.			
9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to "stand clear"; when clear, give the shock.			
10. After shock delivery immediately restart CPR using a ratio of 30:2			
11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.			
12. Pause briefly to check the monitor, confirm VF/pVT from the ECG and Pulse.			
13. If VF/pVT, repeat steps 6–12 above and deliver a second shock.			
14. If VF/pVT persists, repeat steps 6–9 above and deliver a third shock. Resume chest compressions immediately.			
15. Give adrenaline 1 mg IV			
16. Amiodarone 300 mg IV while performing a further 2 min CPR.			
17. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.			
18. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).			

DC: Done correctly

NDC: Not Done correctly

ND: Not Done

Educational Program

Regarding recognition and management of ventricular arrhythmia (Pulseless Ventricular Tachycardia and Ventricular Fibrillation) guideline among critical care nurses

Program Objective:-

The purpose of this program is to provide nurses with up-to-date knowledge of risk factors for development of ventricular arrhythmias, recommended therapies for the immediate management of arrhythmias, and indications of complications, early identification of problems, and appropriate management.

Learning Objectives:-

Upon the completion of this program, the nurse should be able to:

- Define basic concepts of normal cardiac conduction, including cellular events.
- Recognize the normal electrocardiogram (ECG) waveform and differentiate between NSR, VF, VT and asystole.
- Identify ventricular Tachycardia and ventricular fibrillation, including definition, underlying pathophysiology, signs and symptomsm diagnostic methods, complication, and prognosis.
- Outline the assessment, diagnosis, and risk stratification process for patients who have, or are deemed to be at risk for, ventricular arrhythmias.
- List key points in the American Heart Association's Chain of Survival, including the use of defibrillators.
- Summarize key points in the emergency management of life-threatening ventricular arrhythmias for patients, as recommended by Advanced Cardiovascular Life Support (ACLS) guidelines.

Topic	Duration	Tools	Objective
Overview about Pulseless Ventricular tachycardia and fibrillation	30 Min	Computer, projector, PowerPoint	• Background, Statistic, Important of VT& VF management
Basic Electrocardiogram (ECG) Interpretation	60 Min	Computer, projector, PowerPoint, Photos	• Conduction System, Physiology • Recognize normal ECG waveform PQRST. • Recognize VF/ VT from ECG.
European Resuscitation Council Guidelines for Ventricular Fibrillation & pulseless Ventricular Tachycardia Management	60 Min	Computer, projector, PowerPoint	• Chain of Survival , The algorithm for (VF& VT) management, Importance of high quality chest compressions , Safe Use of Defibrillator, • Administration of drugs during (VF/VT) management, potentially reversible causes of (VF/VT).

Practical-secti

Topic	Duration	Tools	Objective
European Resuscitation Council Guidelines for Ventricular Fibrillation & pulseless Ventricular Tachycardia Management	210 Min	Manikin, ECG Simulator, Defibrillator, Emergency trolley, Airway Management Kit	• Implement of effective airway management. • Implement of high quality chest compressions. • Implement safe use of Defibrillator. • CASTeach.

Assess the impact of Educational program for nurses' application of standard guidelines for life threating arrhythmias (VT& VF) recognition and management

MUSAB M.AHMED ALI

Program Objective

The purpose of this program is to provide nurses with up-to-date knowledge of risk factors for development of ventricular arrhythmias, recommended therapies for the immediate management of arrhythmias, and indications of complications , early identification of problems, and appropriate management .

Learning Objectives

Upon completion of this program, you should be able to:

- Define basic concepts of normal cardiac conduction, including cellular events.
- Recognize the normal electrocardiogram (ECG) waveform and differentiate between NSR, VF, VT and asystole.
- Identify ventricular Tachycardia and ventricular fibrillation, including definition, underlying pathophysiology, signs and symptomsm diagnostic methods, complication, and prognosis.

- Outline the assessment, diagnosis, and risk stratification process for patients who have, or are deemed to be at risk for, ventricular arrhythmias.
- List key points in the American Heart Association's Chain of Survival, including the use of defibrillators.
- Summarize key points in the emergency management of life-threatening ventricular arrhythmias for patients, as recommended by Advanced Cardiovascular Life Support (ACLS) guidelines.

Practical Objectives :-

Upon completion of this practical section, you should be able to perform this skills :

- Use the ABCDE approach to assessing and treating the deteriorating and (VF/pVT) patients.
- Use the effective airway management and use the airway adjust during management of (VF/pVT) patient.
- Use the effective and safety use of manual defibrillator during management of (VF/pVT) patient.
- Demonstrate and implement the CAST (all skills above together) during management of (VF/pVT) patient.

Ventricular tachycardia

- Ventricular tachycardia is a rapid, abnormal rhythm that originates in the ventricles and takes over or usurps the heart's normal rhythm.
- Ventricular tachycardia can occur in persons with normal hearts but is more likely to occur in the presence of ischemic or non-ischemic heart disease. It may also develop as a side effect of certain medications .
- The patient may report symptoms ranging from dizziness, lightheadedness, or feeling faint to more severe symptoms of syncope, sudden loss of consciousness, and sudden cardiac death

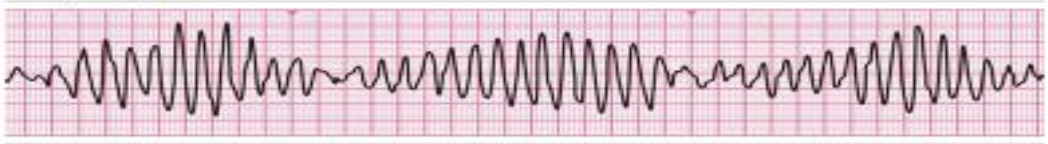
- **There are different types of VT that may vary greatly in the following characteristics:**

- **Duration**
- **Appearance of the QRS complexes**
- **Underlying mechanism**
- **Site or origin of the VT**
- **Impact on cardiac output**

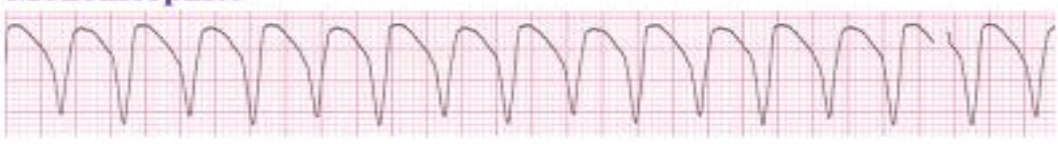
- duration; VT may be described as:
  - sustained or non-sustained.
- clinical presentation; VT may be described as:
  - stable or unstable

- Shape and configuration of the QRS complex: VT may be described as :-
  - Monomorphic or Polymorphic

Polymorphic:

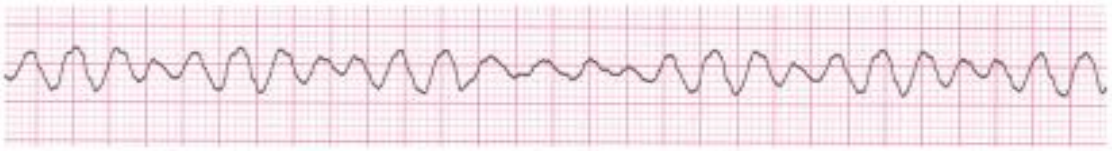


Monomorphic:



Ventricular Fibrillation

- Fibrillation is an uncontrolled twitching or quivering of muscle fibers (fibrils). When it occurs in the lower chambers of the heart, it is called ventricular fibrillation.
- During ventricular fibrillation, blood is not pumped from the heart, sudden cardiac death results.

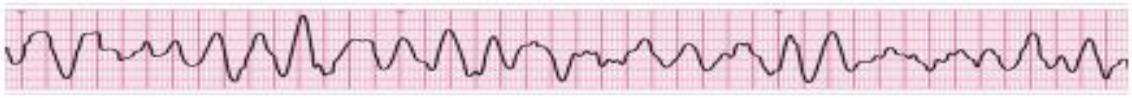


- The most common cause of VF is a heart attack. However, VF can occur whenever the heart muscle does not get enough oxygen. A person who has a VF episode can suddenly collapse or become unconscious, because the brain and muscles have stopped receiving blood from the heart.

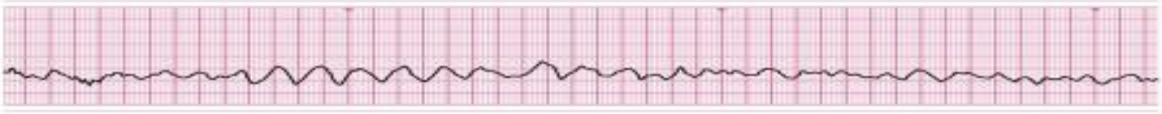
- The following symptoms may occur within minutes to 1 hour before the collapse (Chest pain, Dizziness, Nausea, Rapid heartbeat, Shortness of breath).

Ventricular Fibrillation

Coarse Ventricular Fibrillation (VF)



Fine Ventricular Fibrillation (VF)



ECG Interpretation

MUSAB M.AHMED ALI

BY THE END OF THIS SESSION YOU WILL BE ABLE TO:

- Recognize Normal Impulse Conduction system
- Impulse Conduction system & the ECG
- Known Pacemakers of the Heart
- *Use the 6 steps approach to recognise the ECG rhythm*
- Recognize the normal rhythm of the heart - "Normal Sinus Rhythm."
- Recognize the Ventricular arrhythmias

NORMAL IMPULSE CONDUCTION

Sinoatrial node

↓

AV node

↓

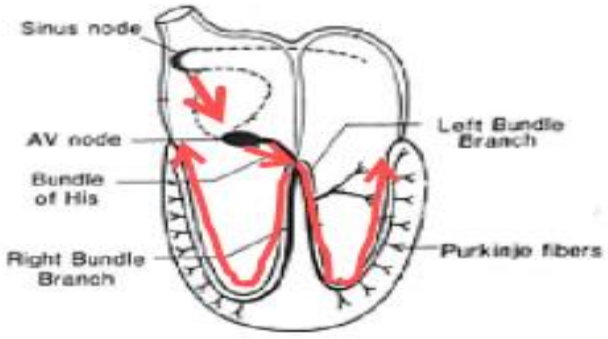
Bundle of His

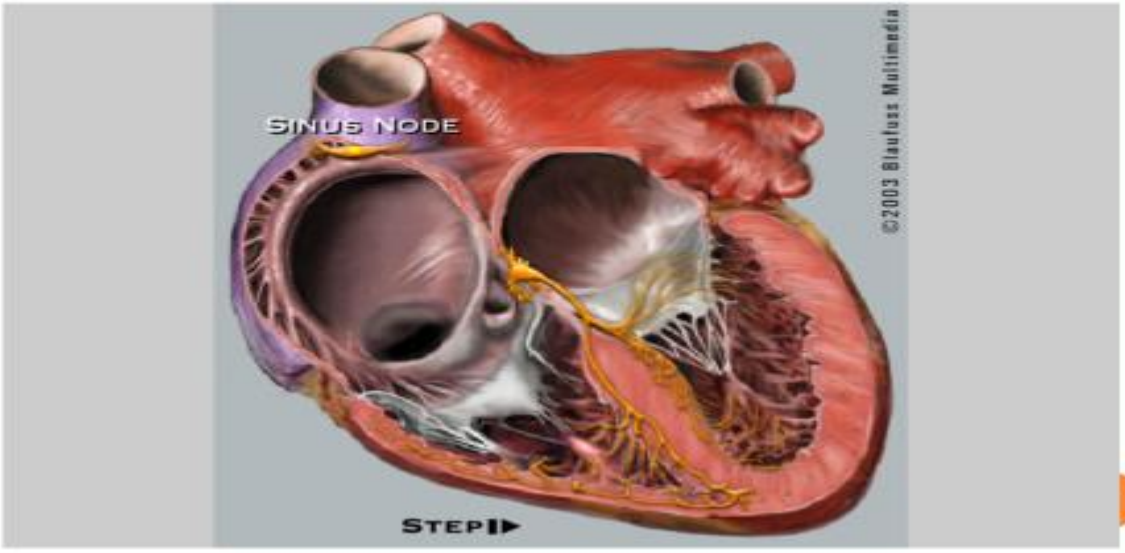
↓

Bundle Branches

↓

Purkinje fibers





Impulse Conduction & the ECG

Sinoatrial node

↓

AV node

↓

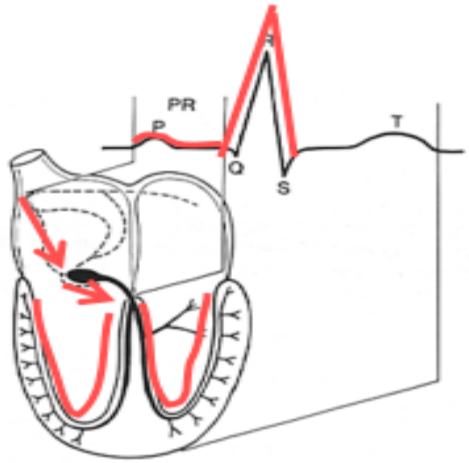
Bundle of His

↓

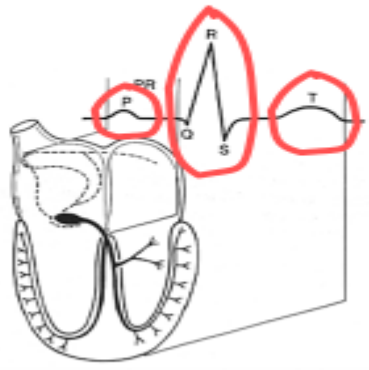
Bundle Branches

↓

Purkinje fibers



The "PQRST"



- P wave - Atrial depolarization
- QRS - Ventricular depolarization
- T wave - Ventricular repolarization

PACEMAKERS OF THE HEART

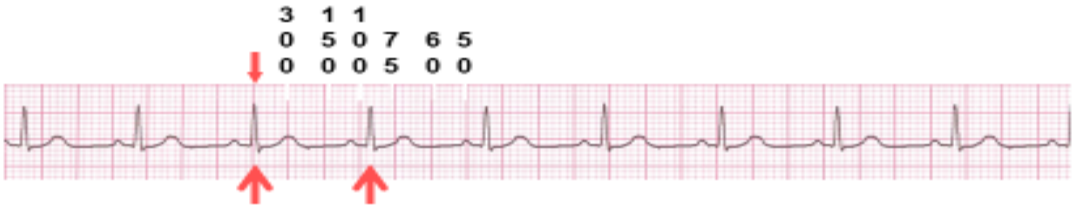
- SA Node - Dominant pacemaker with an intrinsic rate of 60 - 100 beats/minute.
- AV Node - Back-up pacemaker with an intrinsic rate of 40 - 60 beats/minute.
- Ventricular cells - Back-up pacemaker with an intrinsic rate of 20 - 45 bpm.

PRINCIPLES OF ECG RHYTHM RECOGNITION

- *The 6-stage approach to rhythm recognition :*

1. Is there any electrical activity?
Yes Vs No
2. What is the ventricular (QRS) rate?
Calculate Rate
3. Is the QRS rhythm regular or irregular?
Regular Vs irregular
4. Is the QRS width normal or prolonged?
Broad Vs Narrow QRS.
5. Is atrial activity present?
Yes Vs No
6. How is it related to ventricular activity?
Any P wave follow by QRS,& P-R interval.

STEP 1: CALCULATE RATE



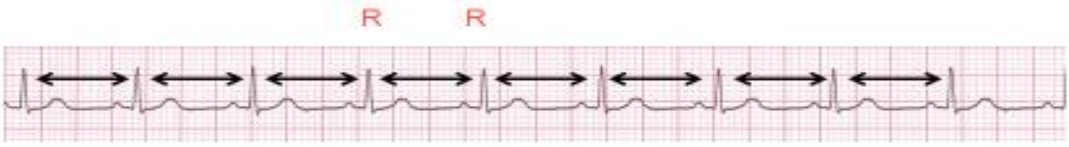
- Option 2 (cont)
  - Memorize the sequence:

    300 - 150 - 100 - 75 - 60 - 50

Interpretation?

Approx. 1 box less than 100 = 95 bpm

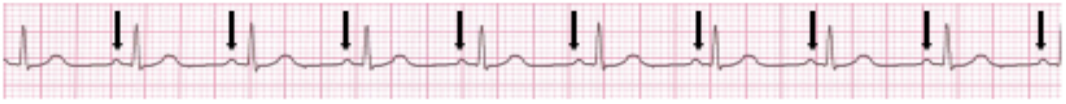
STEP 2: DETERMINE REGULARITY



- Look at the R-R distances (using a caliper or markings on a pen or paper).
- Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular? Irregularly irregular?

Interpretation? *Regular*

STEP 3: ASSESS THE P WAVES

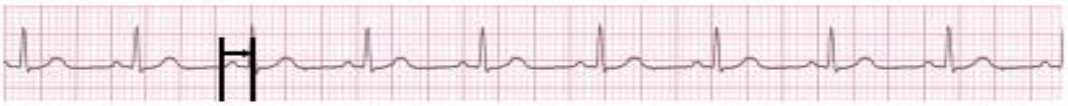


- Are there P waves?
- Do the P waves all look alike?
- Do the P waves occur at a regular rate?
- Is there one P wave before each QRS?

Interpretation?

Normal P waves with 1 P wave for every QRS

STEP 4: DETERMINE PR INTERVAL

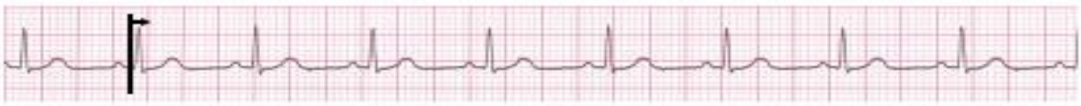


- Normal: 0.12 - 0.20 seconds.
  (3 - 5 boxes)

Interpretation?

0.12 seconds

STEP 5: QRS DURATION

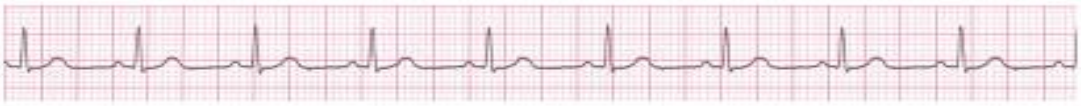


- Normal: 0.04 - 0.12 seconds.
  (1 - 3 boxes)

Interpretation?

0.08 seconds

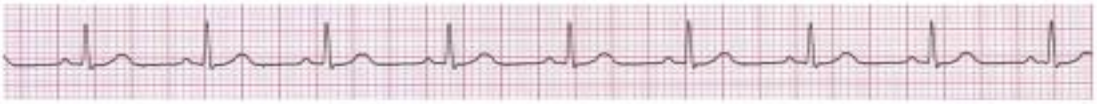
NORMAL SINUS RHYTHM (NSR)



Rate	90-95 bpm
Regularity	regular
P waves	normal
PR interval	0.12 s
QRS duration	0.08 s

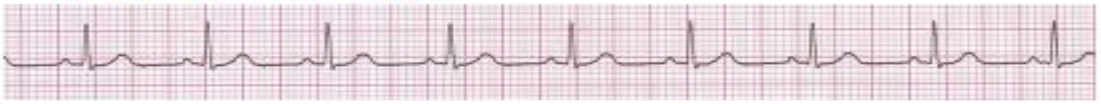
Interpretation? *Normal Sinus Rhythm*

Normal Sinus Rhythm (NSR)



- Etiology: the electrical impulse is formed in the SA node and conducted normally.
- This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.

NSR Parameters



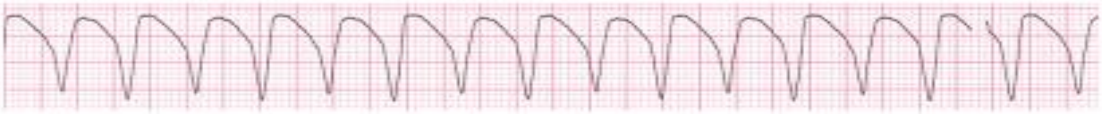
- Rate 60 - 100 bpm
- Regularity regular
- P waves normal
- PR interval 0.12 - 0.20 s
- QRS duration 0.04 - 0.12 s

Any deviation from above is sinus tachycardia, sinus bradycardia or an arrhythmia

Ventricular Arrhythmias

- *Ventricular Tachycardia*
- *Ventricular Fibrillation*
- *Asystole*

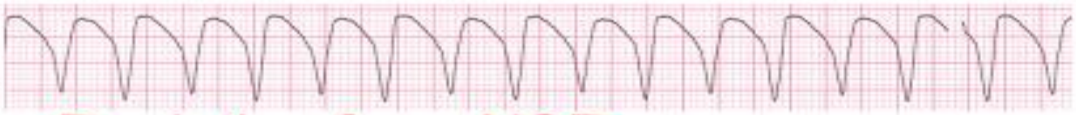
Ventricular Tachycardia



- Rate? 160 bpm
- Regularity? regular
- P waves? none
- PR interval? none
- QRS duration? wide (> 0.12 sec)

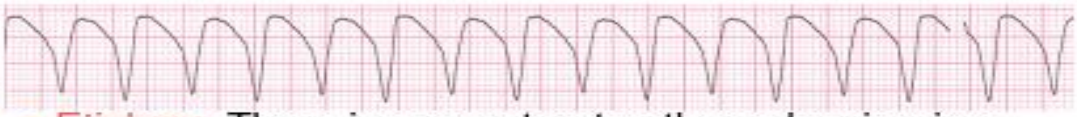
Interpretation? *Ventricular Tachycardia*

VENTRICULAR TACHYCARDIA



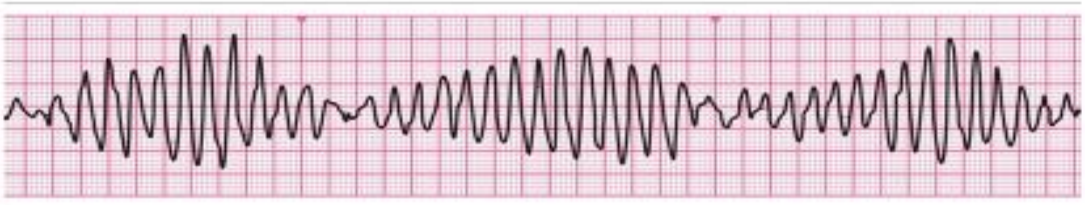
- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).

VENTRICULAR TACHYCARDIA

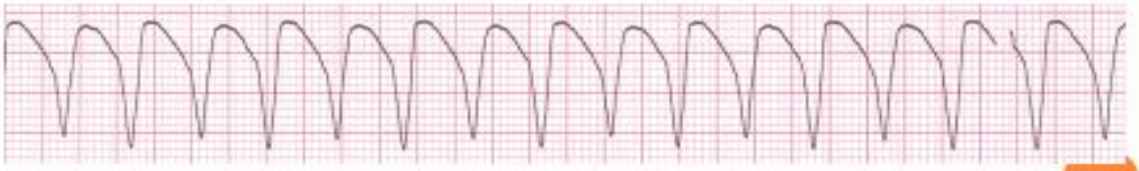


- Etiology: There is a re-entrant pathway looping in a ventricle (most common cause).
- Ventricular tachycardia can sometimes generate enough cardiac output to produce a pulse; at other times no pulse can be felt.

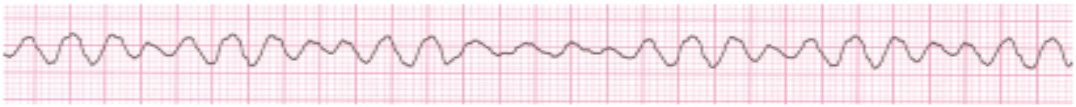
Polymorphic:



Monomorphic:



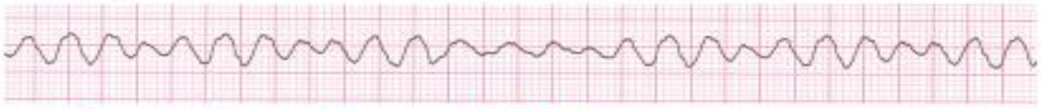
VENTRICULAR FIBRILLATION



- Rate? none
- Regularity? irregularly irreg.
- P waves? none
- PR interval? none
- QRS duration? wide, if recognizable

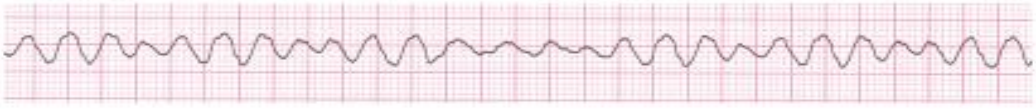
Interpretation? *Ventricular Fibrillation*

VENTRICULAR FIBRILLATION



- Deviation from NSR
  - Completely abnormal.

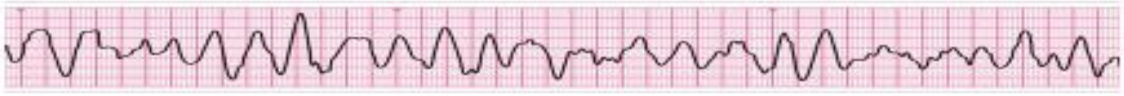
VENTRICULAR FIBRILLATION



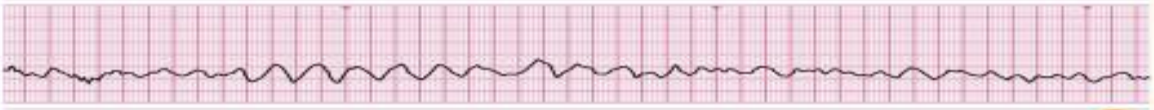
- Etiology: The ventricular cells are excitable and depolarizing randomly.
- Rapid drop in cardiac output and death occurs if not quickly reversed

VENTRICULAR FIBRILLATION

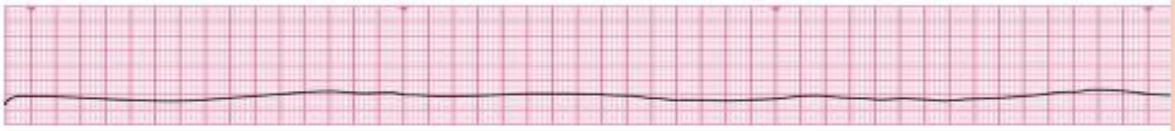
Coarse Ventricular Fibrillation (VF)



Fine Ventricular Fibrillation (VF)



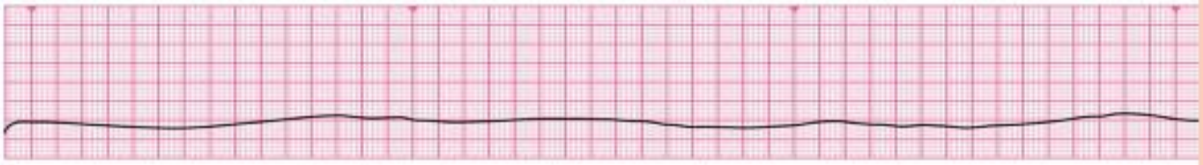
ASYSTOLE



- Rate? none
- Regularity? none
- P waves? none
- PR interval? none
- QRS duration? none

Interpretation? *Asystole*

ASYSTOLE:



Asystole:

- Absent ventricular (QRS) activity Atrial activity (P waves) may persist.
- Rarely a straight line trace.

KEY LEARNING POINTS

- ***Now by the end of this session you able to:***

- **Recognize Normal Impulse Conduction system**
- **Impulse Conduction system & the ECG**
- **Known Pacemakers of the Heart**
- ***Use the 6 steps approach to recognise the ECG rhythm***
- **Recognize the normal rhythm of the heart - "Normal Sinus Rhythm."**
- **Recognize the Ventricular arrhythmias**

Ventricular Fibrillation & pulseless Ventricular Tachycardia Management

MUSAB M.AHMED ALI

BY THE END OF THIS SESSION THE TRAINER WILL ABLE TO:

- **Known the Chain of survival .**
- Describe the ABCDE approach survey to recognise and treat patients at risk of cardiac arrest
- **Known the ERC 2015 In-hospital cardiac arrest algorithm.**
- **Known the ERC 2015 algorithm for (VF& pVT) management**
- Known the Importance of high quality chest compressions
- **Known how to Safety use the Defibrillator**

BY THE END OF THIS SESSION THE TRAINER WILL ABLE TO: CONTINUE

- Known the appropriate administration of adrenergic agents and antiarrhythmic,& other agents drugs during (VF/pVT) management.
- **Known the Potentially reversible causes of (VF/pVT).**
- Known the Post-resuscitation care.

Early recognition prevents:

- Cardiac arrests and deaths
- Admissions to ICU
- Inappropriate resuscitation attempts

THE ABCDE APPROACH

Airway

Breathing

Circulation

Disability

Exposure

ABCDE APPROACH

Underlying principles:

- Complete initial assessment
- Treat life-threatening problems
- Reassessment
- Assess effects of treatment/interventions
- Call for help early
- Communicate effectively - use the Situation, Background, Assessment, Recommendation (**SBAR**) or Reason, Story, Vital signs, Plan (**RSVP**) approach.

ABCDE APPROACH

First steps

- **Personal safety**
- First look at the patient in general to see if the patient appears unwell.
- **If the patient is awake, ask "How are you?". If the patient appears unconscious or has collapsed, shake him and ask "Are you alright?"**
- This first rapid 'Look, Listen &Feel" of the patient should take about 30S
- **If the patient is unconscious, unresponsive, &is not breathing normally start CPR according to the resuscitation guidelines**
- Vital signs
  - Respiratory rate, SpO_2, pulse, BP, GCS, temperature
- **Insert an intravenous cannula as soon as possible.**

ABCDE approach

AIRWAY

Causes of airway obstruction:

- CNS depression
- Blood
- Vomit
- Foreign body
- Trauma
- Infection
- Inflammation
- Laryngospasm
- Bronchospasm

ABCDE approach

AIRWAY

Recognition of airway obstruction:

- Talking
- Difficulty breathing, distressed, choking
- Shortness of breath
- Noisy breathing
  - Stridor, wheeze, gurgling
- See-saw respiratory pattern, accessory muscles

ABCDE approach

AIRWAY

Treatment of airway obstruction:

- Airway opening
  - Head tilt, chin lift, jaw thrust
- Simple adjuncts
- Advanced techniques
  - e.g. LMA, tracheal tube
- Oxygen

ABCDE approach

BREATHING

Recognition of breathing problems:

- **Look**
  - Respiratory distress, accessory muscles, cyanosis, respiratory rate, chest deformity, conscious level
- **Listen**
  - Noisy breathing, breath sounds
- **Feel**
  - Expansion, percussion, tracheal position

ABCDE approach

BREATHING

Treatment of breathing problems:

- Airway
- Oxygen
- Treat underlying cause
  - e.g. antibiotics for pneumonia
- Support breathing if inadequate
  - e.g. ventilate with bag-mask

+

ABCDE approach

CIRCULATION

Causes of circulation problems:

- **Primary**
  - Acute coronary syndromes
  - Arrhythmias
  - Hypertensive heart disease
  - Valve disease
  - Drugs
  - Inherited cardiac diseases
  - Electrolyte/acid base abnormalities
- **Secondary**
  - Asphyxia
  - Hypoxaemia
  - Blood loss
  - Hypothermia
  - Septic shock

ABCDE approach

CIRCULATION

Recognition of circulation problems:

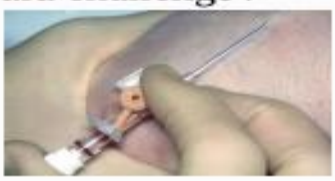
- Look at the patient
- Pulse - tachycardia, bradycardia
- Peripheral perfusion - capillary refill time
- Blood pressure
- Organ perfusion
  - Chest pain, mental state, urine output
- Bleeding, fluid losses

ABCDE approach

CIRCULATION

Treatment of circulation problems:

- Airway, Breathing .
- Oxygen .
- IV/IO access, take bloods .
- Treat cause .
- Fluid challenge .



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ABCDE approach

CIRCULATION

Acute Coronary Syndromes

- Unstable angina or myocardial infarction
- Treatment
  - Aspirin 300 mg orally (crushed/chewed)
  - Nitroglycerine (GTN spray or tablet)
  - Oxygen guided by pulse oximetry
  - Morphine (or diamorphine)
- Consider reperfusion therapy (PCI, thrombolysis)

ABCDE approach

DISABILITY

Recognition

- AVPU or GCS
- Pupils

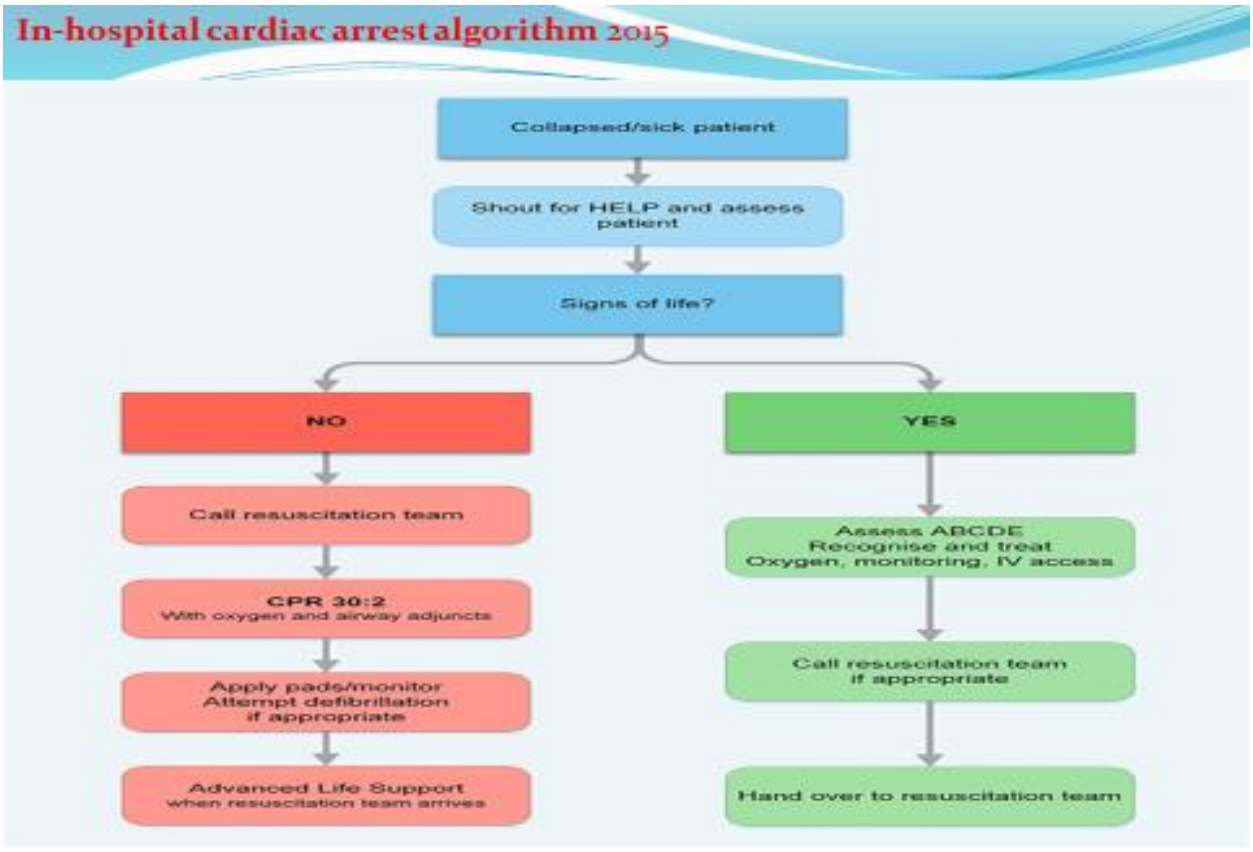
Treatment

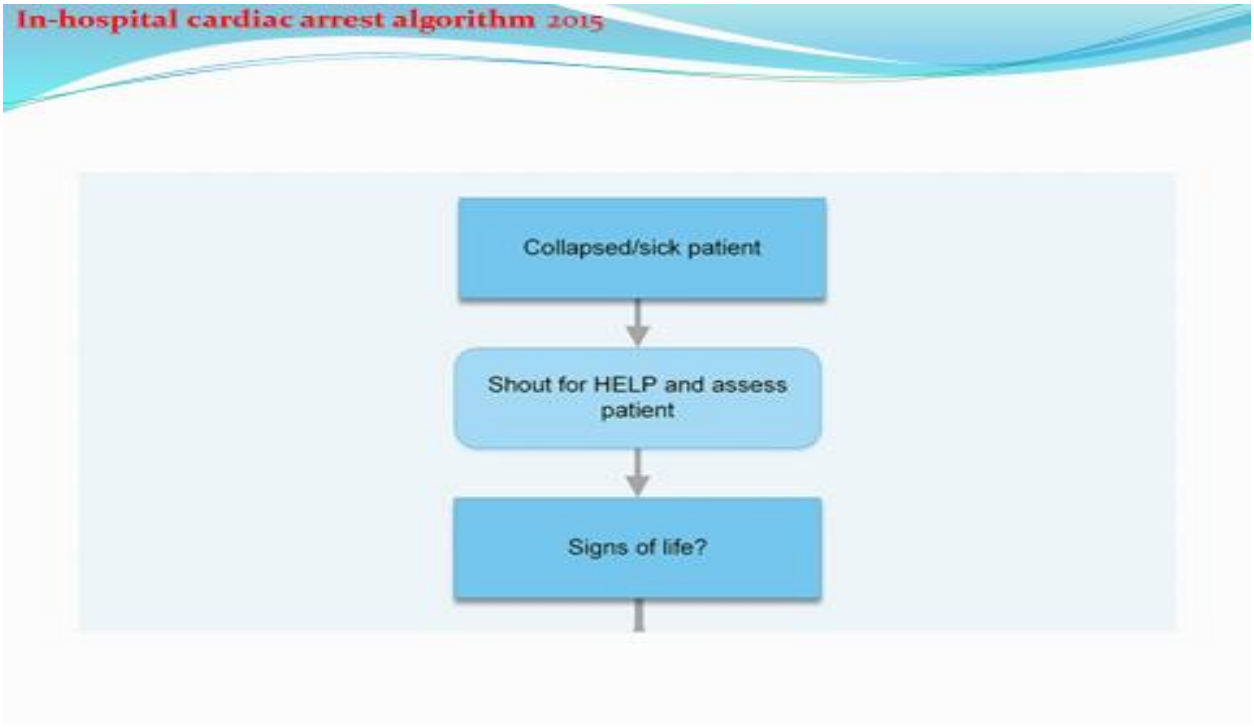
- ABC
- Treat underlying cause
- Blood glucose
  - If < 4 mmol l^{-1} give glucose
- Consider lateral position
- Check drug chart

ABCDE approach

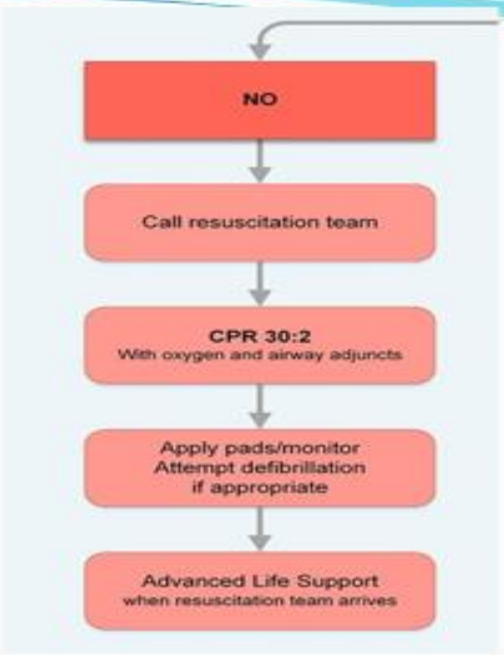
EXPOSURE

- Remove clothes to enable examination
  - e.g. injuries, bleeding, rashes
- Avoid heat loss
- Maintain dignity

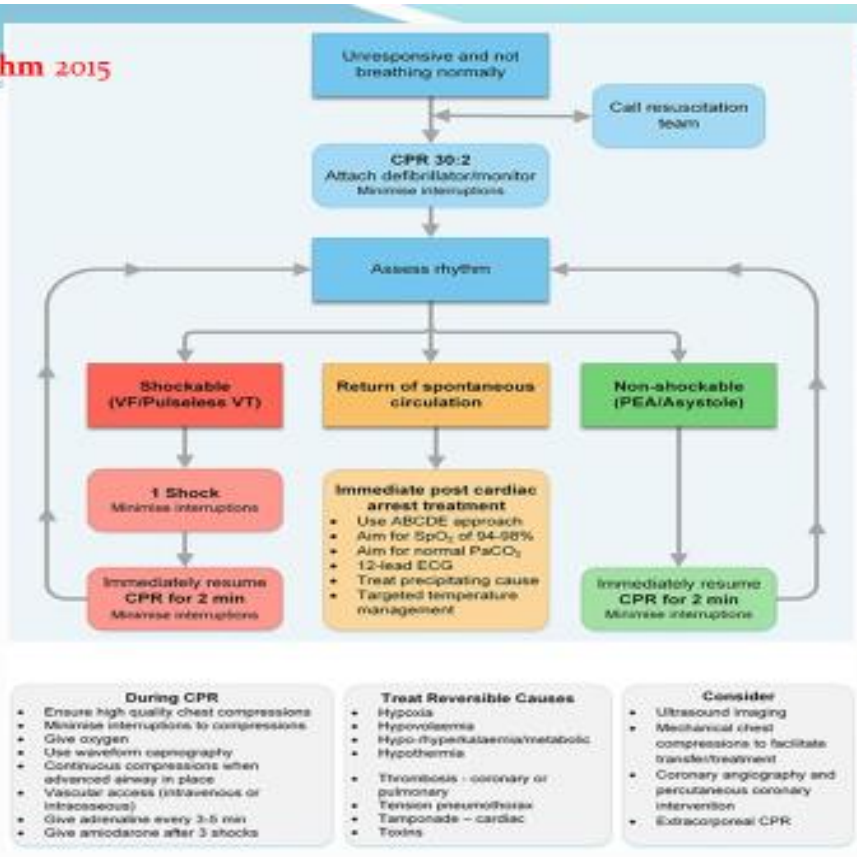

In-hospital cardiac arrest algorithm 2015
Collapsed/sick patient
Shout for HELP and assess patient
Signs of life?
NO
Call resuscitation team
CPR 30:2
With oxygen and airway adjuncts
Apply pads/monitor
Attempt defibrillation if appropriate
Advanced Life Support when resuscitation team arrives
YES
Assess ABCDE
Recognise and treat
Oxygen, monitoring, IV access
Call resuscitation team if appropriate
Hand over to resuscitation team

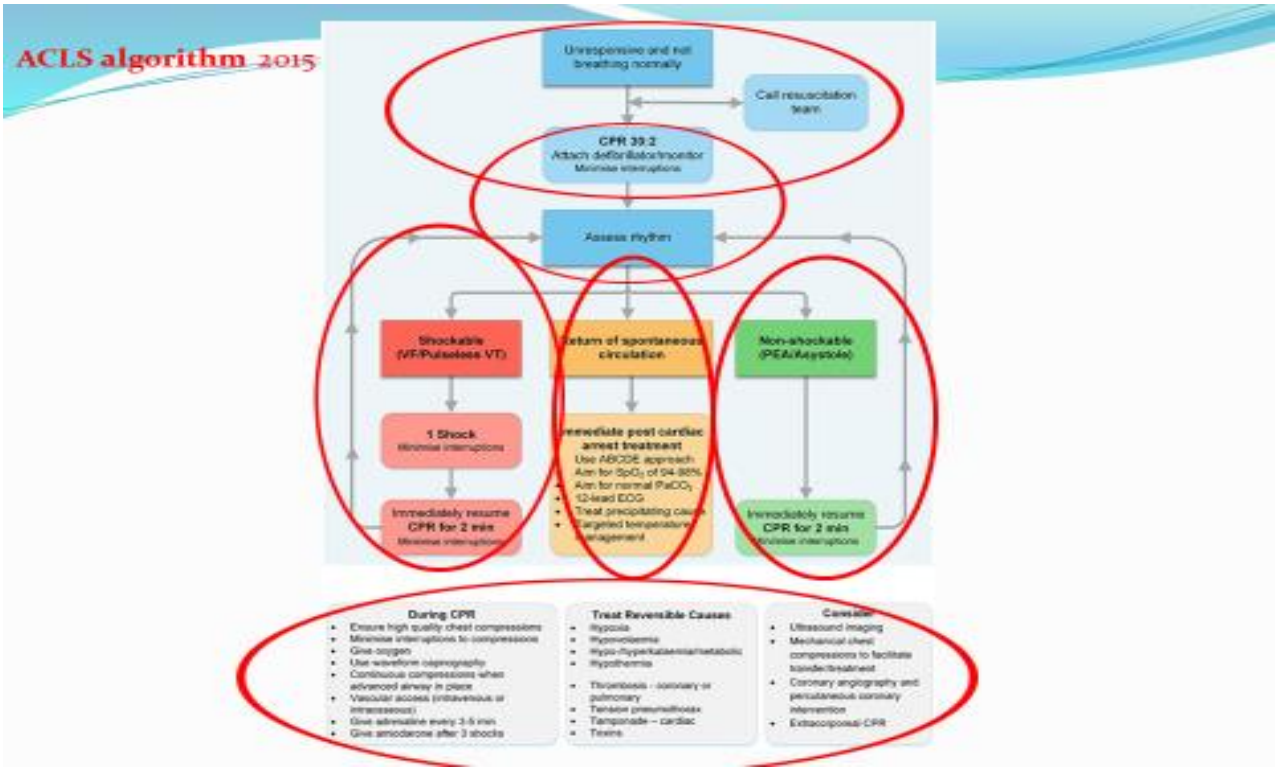

In-hospital cardiac arrest algorithm 2015
Collapsed/sick patient
Shout for HELP and assess patient
Signs of life?

In-hospital cardiac arrest algorithm 2015



ACLS algorithm 2015





Unresponsive?
Not breathing or
only occasional gasps

To confirm cardiac arrest...

- Patient response
- Open airway
- Check for normal breathing
  - Caution agonal breathing
- Check circulation
- Check for signs of life

Unresponsive?
Not breathing or
only occasional gasps

Cardiac arrest confirmed

Call
resuscitation team
CODE BLU TEAM

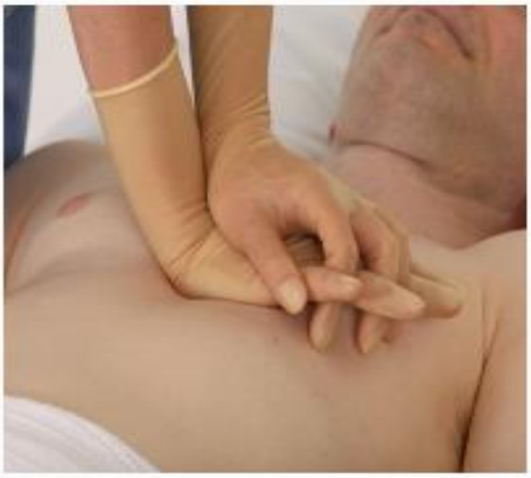
Unresponsive?
Not breathing or
only occasional gasps

Cardiac arrest confirmed

Call
resuscitation team

CPR 30:2
Attach defibrillator / monitor
Minimise interruptions

Chest compression

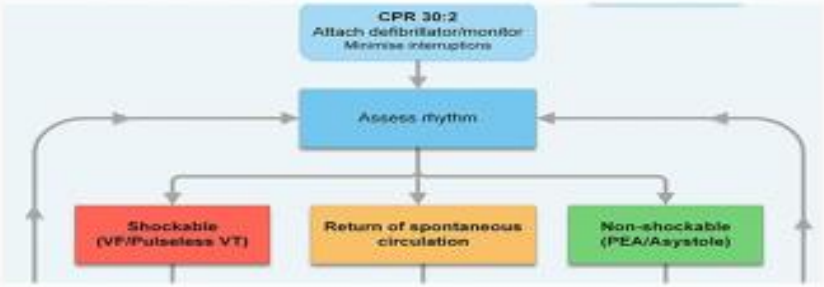


- 30:2
- Compressions
  - Centre of chest
  - 5-6 cm depth
  - 2 per second (100-120 min)
- Maintain high quality compressions with minimal interruption
- Continuous compressions once airway secured
- Switch compression provider every 2 min to avoid fatigue

During CPR

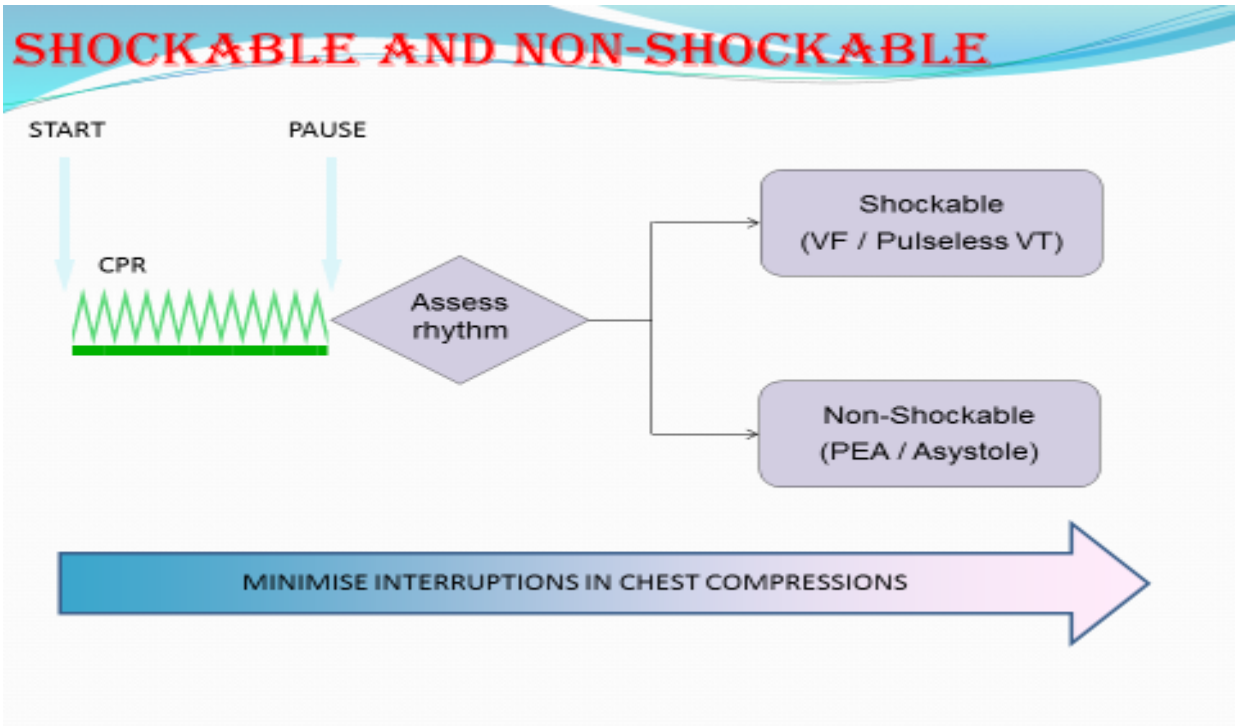
- **Ensure high-quality CPR: rate, depth, recoil**
- **Plan actions before interrupting CPR**
- **Give oxygen**
- **Consider advanced airway and capnography**
- **Continuous chest compressions when advanced airway in place**
- **Vascular access (intravenous, intraosseous)**
- **Give adrenaline every 3-5 min**
- **Correct reversible causes**
- **Drugs and advanced airways are still included among pVT/VF interventions, but are of secondary importance to early defibrillation and high quality, uninterrupted chest compressions 2015**

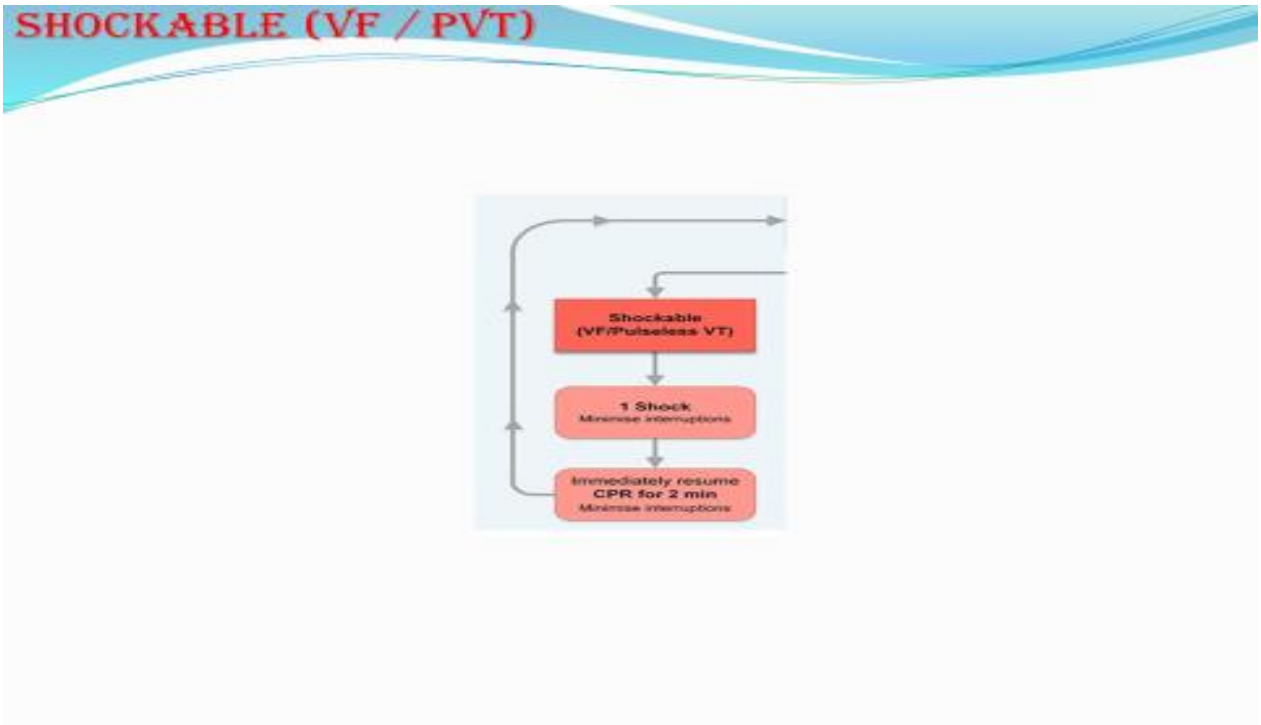
DEFIBRILLATION

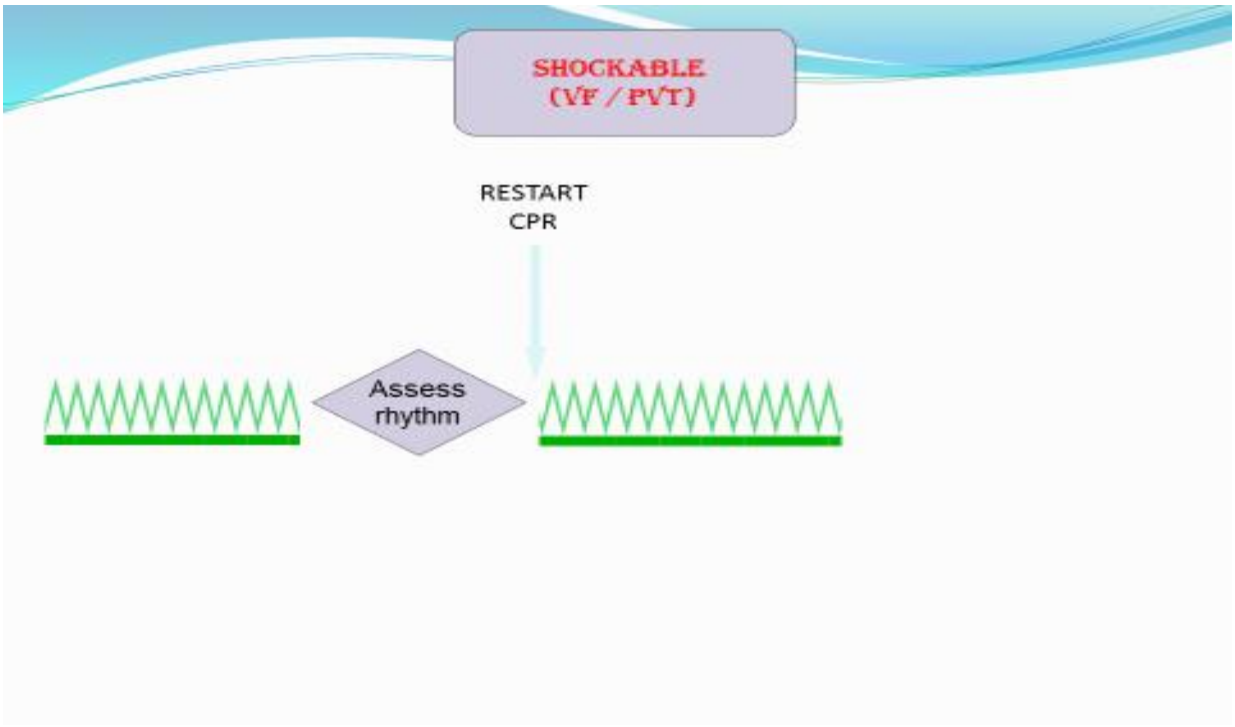


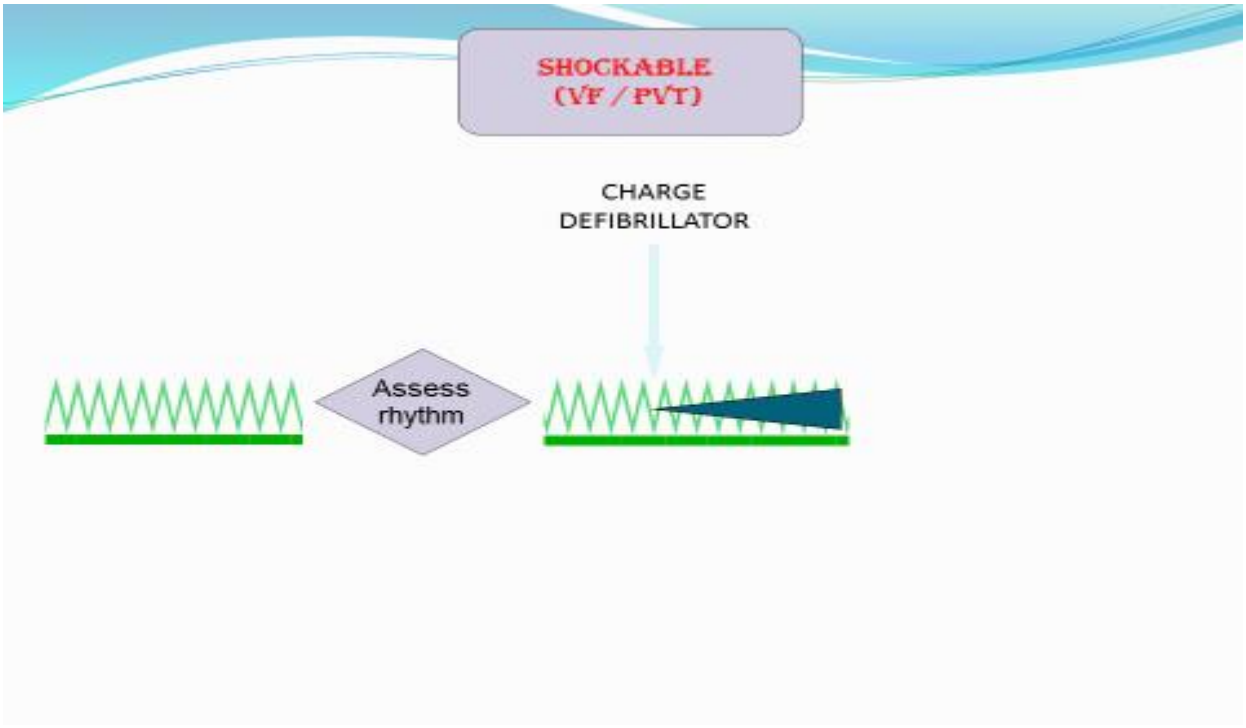
DEFIBRILLATION

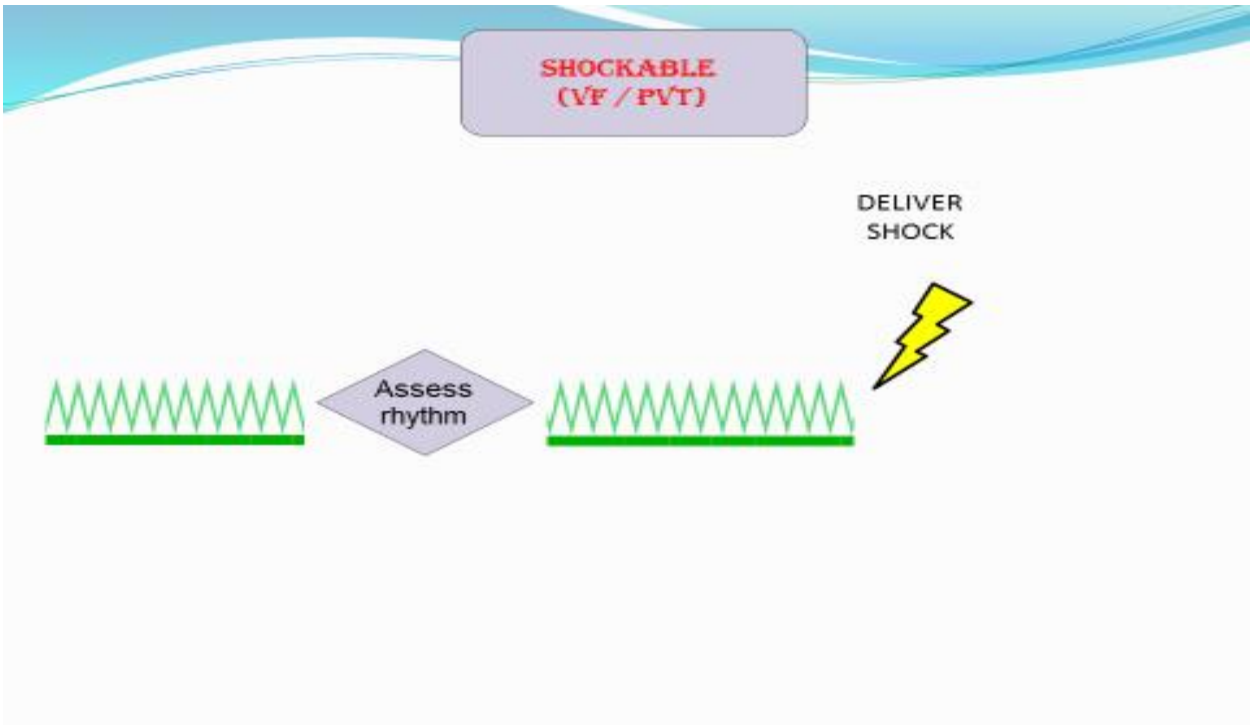
- Plan all pauses in chest compressions
- Brief pause in compressions to check rhythm
- Do chest compressions when charging
- Ensure no-one touches patient during shock delivery
- Very brief pause in chest compressions for shock delivery
- Resume compressions immediately after the shock

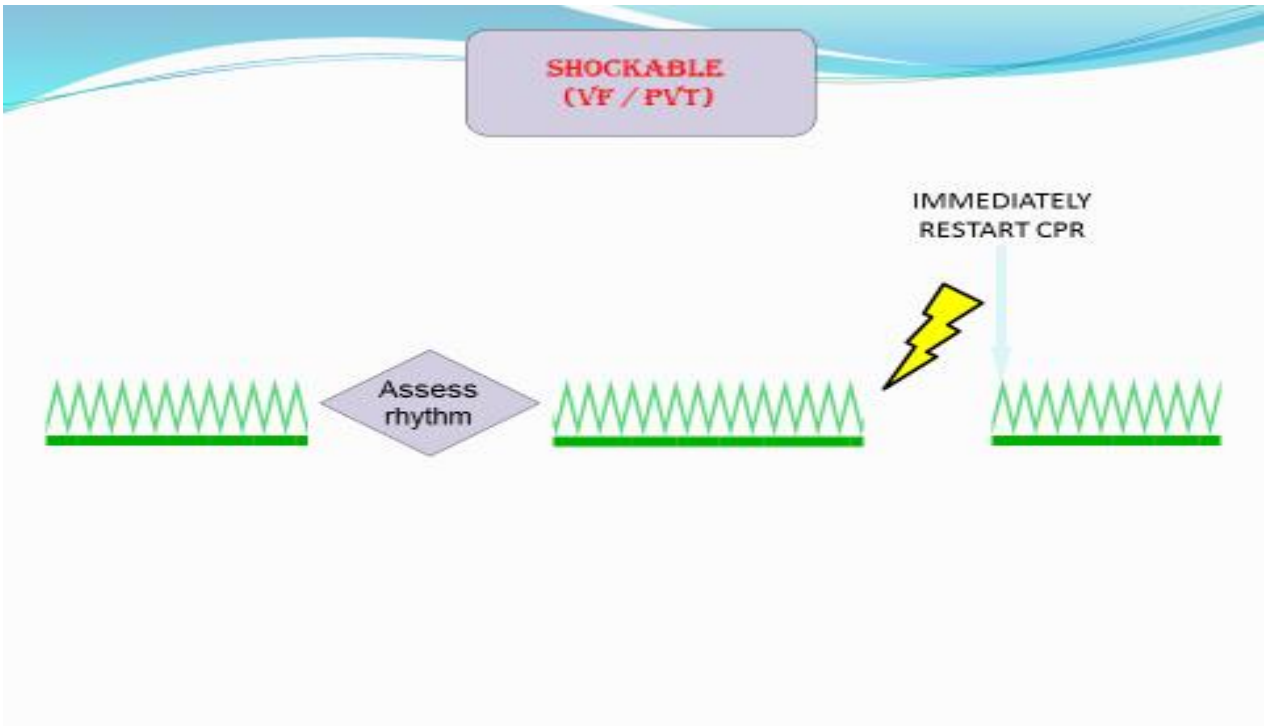

SHOCKABLE AND NON-SHOCKABLE
START
PAUSE
CPR
Assess rhythm
Shockable (VF / Pulseless VT)
Non-Shockable (PEA / Asystole)
MINIMISE INTERRUPTIONS IN CHEST COMPRESSIONS

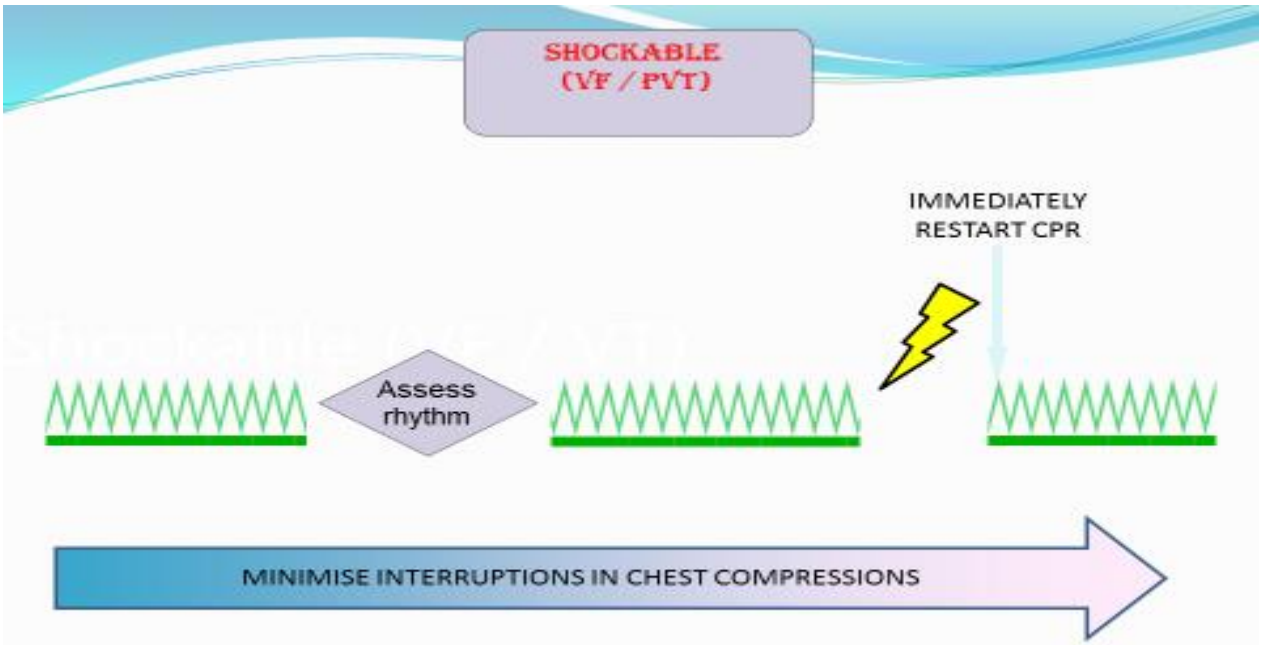

SHOCKABLE (VF / PVT)
Shockable (VF/Pulseless VT)
1 Shock
Immediately resume CPR for 2 min


SHOCKABLE
(VF / PVT)
RESTART
CPR
Assess
rhythm


SHOCKABLE
(VF / PVT)
CHARGE
DEFIBRILLATOR
Assess
rhythm


SHOCKABLE
(VF / PVT)
DELIVER
SHOCK
Assess
rhythm


SHOCKABLE
(VF / PVT)
IMMEDIATELY
RESTART CPR
Assess
rhythm



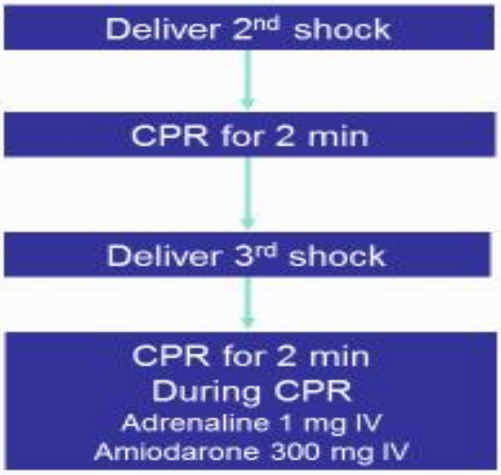
MANUAL DEFIBRILLATION ENERGIES

- Vary with manufacturer
- Check local equipment
- If unsure, deliver highest available energy

➢ DO NOT DELAY SHOCK

- Energy levels for manual defibrillators on this course

(VF/VT) ALGORITHM



(VF/VT) ALGORITHM

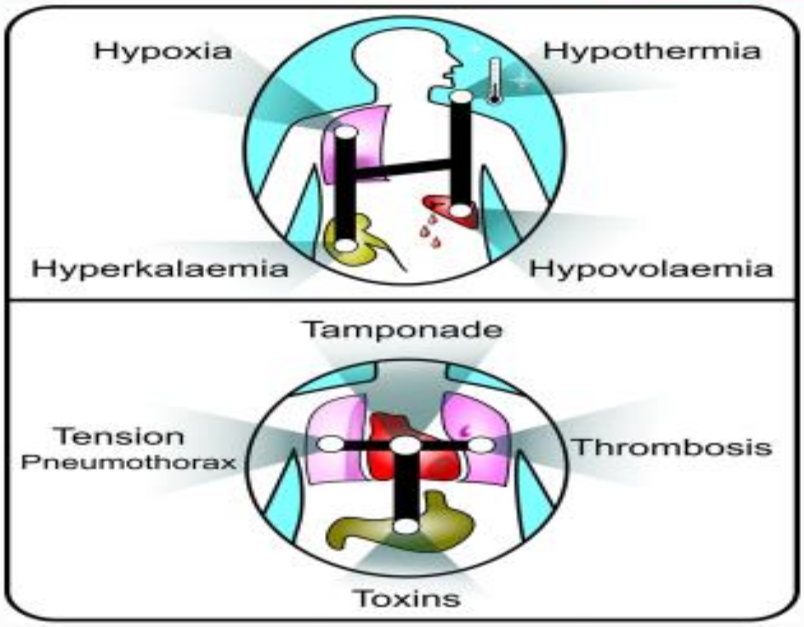
DRUGS DURING (VF/PVT)

- **Adrenaline**
  - 1 mg IV if VF/VT persists after the third shock.
  - Repeat every 3-5 min thereafter if VF/VT persists.
- **Amiodarone**
  - 300 mg by bolus injection.
  - Then 150 mg may be given for recurrent or refractory VF/VT
  - Followed by an infusion of 900 mg over 24 h (ROSC)
- **Lidocaine**
  - 1 mg/kg, If amiodarone is not available
  - Do not exceed a total dose of 3 mg kg-1 during the first hour

(VF/VT) DRUGS

- **Magnesium**
  - Magnesium sulphate 8 mmol (4 ml of a 50% solution) for refractory VF
- **Sodium bicarbonate**
  - 50 mmol if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose.
- **Calcium**
  - 10 ml 10% calcium chloride.

REVERSIBLE CAUSES



HYPOXIA

- Ensure patent airway
- Give high-flow supplemental oxygen
- Avoid hyperventilation

AIRWAY AND VENTILATION

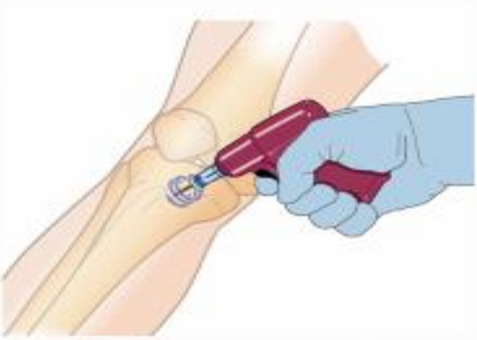
- Secure airway:
  - Supraglottic airway device e.g. LMA.
  - Tracheal tube (ETT)
- Do not attempt intubation unless trained and competent to do so
- Once airway secured, if possible, do not interrupt chest compressions for ventilation
- Avoid hyperventilation

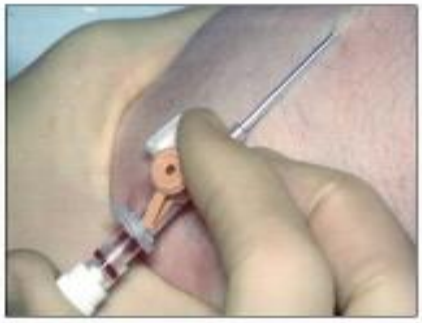
HYPOVOLAEMIA

- Seek evidence of hypovolaemia
  - History
  - Examination
    - Internal haemorrhage
    - External haemorrhage
    - Check surgical drains
- Control haemorrhage
- If hypovolaemia suspected give intravenous fluids

VASCULAR ACCESS

- Peripheral versus central veins
- Intraosseous





HYPO/HYPERKALAEMIA AND METABOLIC DISORDERS

- Near patient testing for K^+ and glucose
- Check latest laboratory results
- Hyperkalaemia
  - Calcium chloride
  - Insulin/dextrose
- Hypokalaemia/ Hypomagnesaemia
  - Electrolyte supplementation

HYPOTHERMIA

- Rare if patient is an in-patient
- Use low reading thermometer
- Treat with active rewarming techniques
- Consider cardiopulmonary bypass

TENSION PNEUMOTHORAX

- Check tube position if intubated
- Clinical signs
  - Decreased breath sounds
  - Hyper-resonant percussion note
  - Tracheal deviation
- Initial treatment with needle decompression or thoracostomy

TAMPONADE, CARDIAC

- Difficult to diagnose without echocardiography
- Consider if penetrating chest trauma or after cardiac surgery
- Treat with needle pericardiocentesis or resuscitative thoracotomy

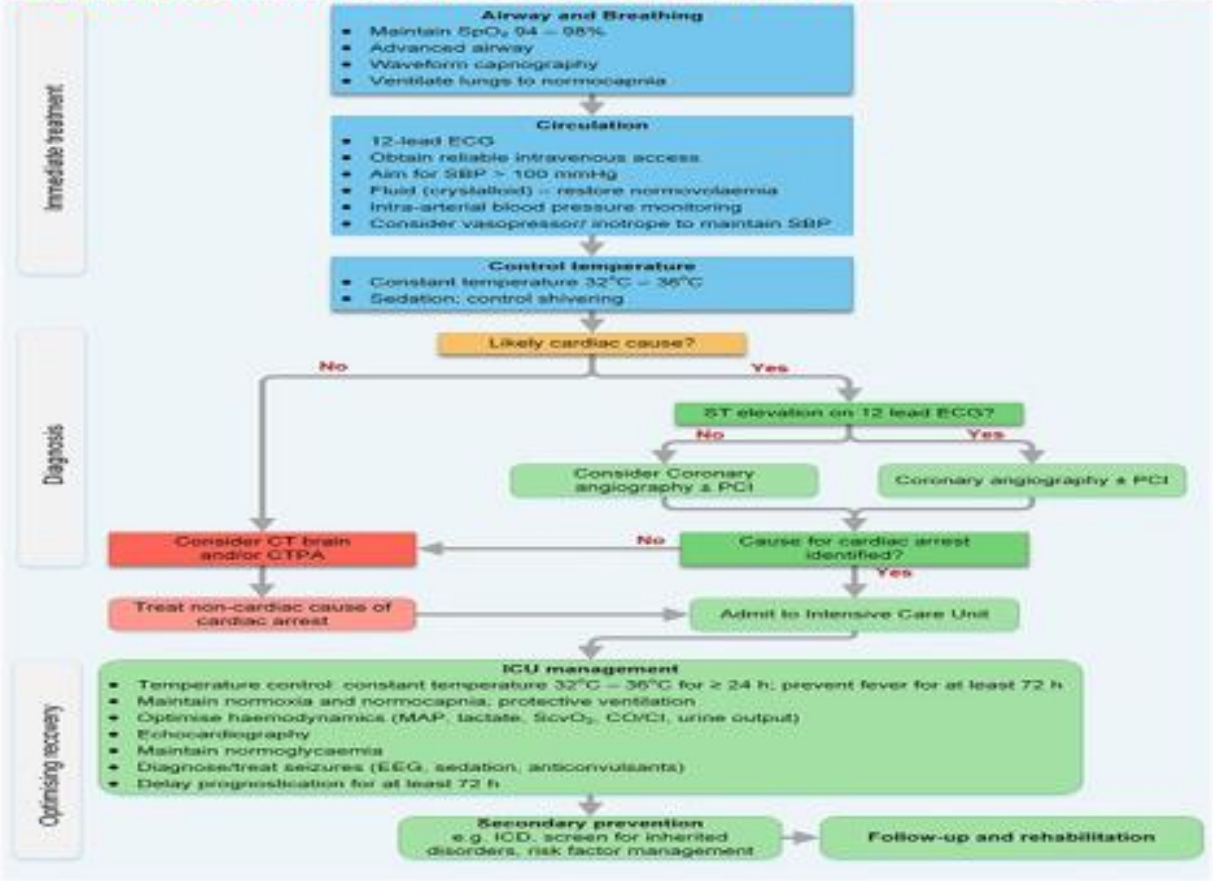
TOXINS

- Rare unless evidence of deliberate overdose
- Review drug

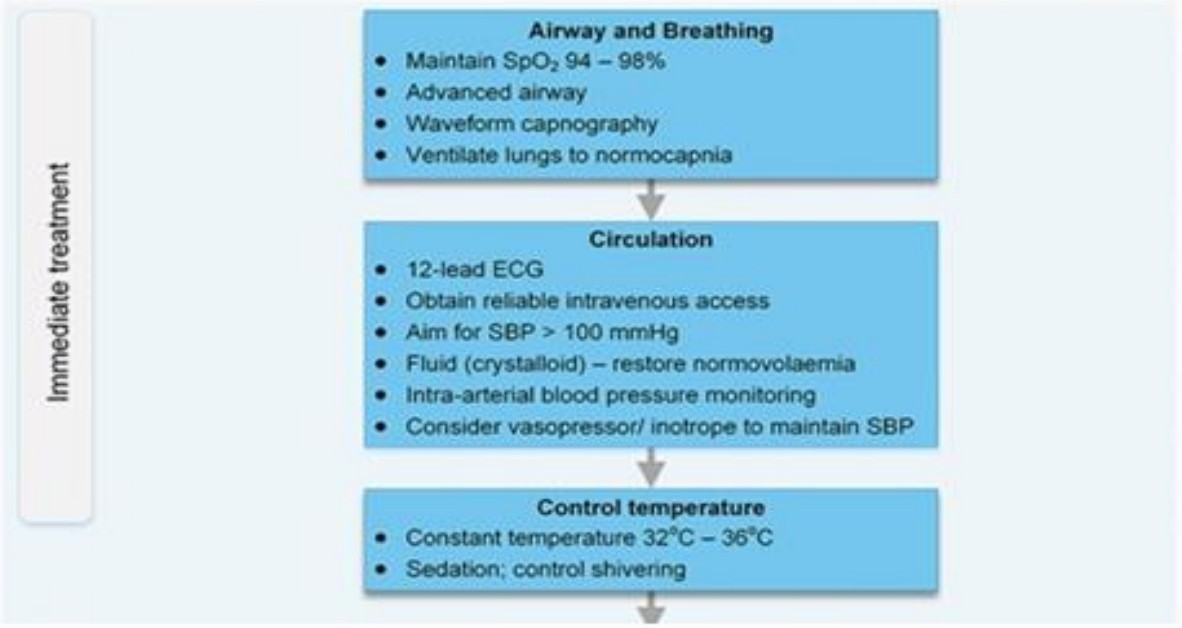
THROMBOSIS

- If high clinical probability for PE consider fibrinolytic therapy
- If fibrinolytic therapy given continue CPR for up to 60-90 min before discontinuing resuscitation

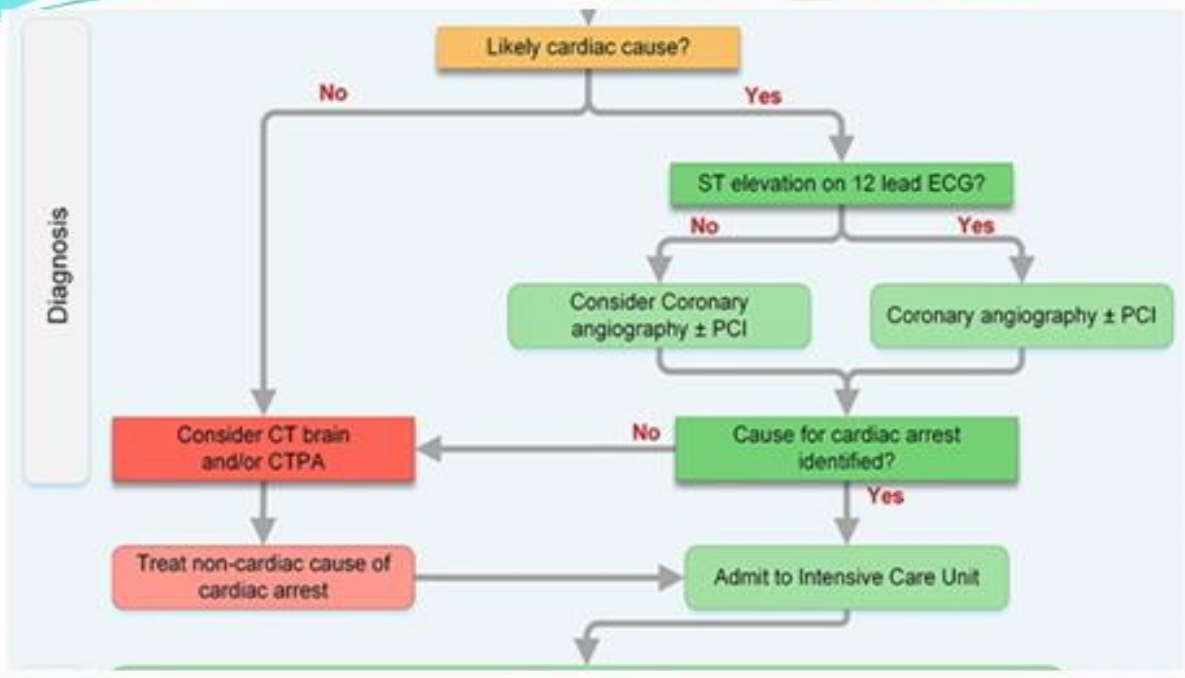
Post-resuscitation care algorithm 2015



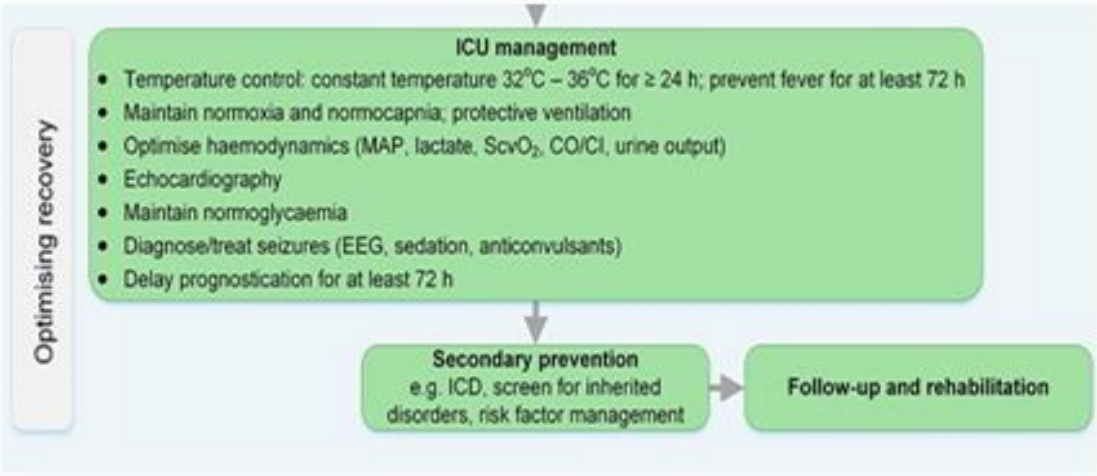
Immediate Post-resuscitation Treatment:



Post-resuscitation Diagnosis :



Optimising Recovery Post-resuscitation:



IMMEDIATE POST VT& VF TREATMENT

Airway

- Assess patency,
- Open airway
- Maintain patency,
- Consider definitive airway

Breathing

- Assess breathing
- Manage and give O_2
- Monitor by pulse SpO2 & ABGs

Circulation

- PR, BP, Capillary refill time.
- Manage accordingly
- Attach to rhythm Monitor

Disability

- Pupils
- Glucose check
- AVPU scale

Exposure

RESUSCITATION TEAM

- Roles planned in advance
- Identify team leader
- Importance of non-technical skills
  - Task management
  - Team working
  - Situational awareness
  - Decision making
- Structured communication
  - SBAR or RSVP

ANY QUESTIONS?


Thank you
let us to demonstrate