Update and Recent Trends in Management of Cardiac Asystole

Protocol For an Essay
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By

Reda Ibrahim Eldesoky Elkohafi

M.B., B.Ch

Under supervision of

Prof. Dr. Azza Youssef Ibrahim
Professor of Anesthesiology and Intensive Care Medicine
Faculty of Medicine –Ain Shams University

Dr. Hesham Mohammed Elazzazi
Assistant Professor of Anesthesiology and Intensive care Medicine
Faculty of Medicine –Ain Shams University

Dr. Dalia Abdelhamid Nasr
Assistant Professor of Anesthesiology and Intensive care Medicine
Faculty of Medicine –Ain Shams University

Faculty of Medicine
Ain Shams University
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List of Abbreviations

Medical Terms

ABC	Airway, Breathing, and Circulation
ABCDE	Airway, Breathing, Circulation, Disability, and Exposure
ABG	Arterial Blood Gases
ACC	American College of Cardiology
ACCP	American College of Chest Physician
ACE	Angiotensin Converting Enzyme
ACLS	Advanced Cardiac Life Support
AED	Automatic External Defibrillator
AHA	American Heart Association
AIDS	Acquired Immuno Deficiency Syndrome
AV	Atrio Ventricular
ASA	American Society of Anesthesiologists
BLS	Basic Life Support
BTS	British Thoracic Society
CAB	Chest compressions, Airway, Breathing
CABG	Coronary Artery Bypass Grafting
CAD	Coronary Artery Disease
CAH	Congenital Adrenal Hyperplasia
CCU	Coronary Care Unit
CCM	Critical Care Medicine
CT	Computerized Tomography
CHF	Congestive Heart Failure
CPR	CardioPulmonary Resuscitation
CPVT	Catecholaminergic Polymorphic Ventricular Tachycardia
CVD	Cardiovascular disease
CVVD	Continuous Veno-Venous Hemofiltration
DC	Direct Current
DM	Diabetes Melllitis
DNAR	Do Not Attempt Resuscitation
DVT	Deep Venous Thrombosis
ECC	Emergency Cardiovascular Care Committee
ECG	ElectroCardioGram
EF	Ejection Fraction
EMS	Emergency Medical Support

EPS	Electrophysiology Studies
FiO_2	Fraction of inspired oxygen
GCS	Glasgow Coma Scale
HTN	Hypertension
ICD	Implantable cardioverter defibrillator
ICU	Intensive Care Unit
IHD	Ischamic Heart Disease
IM	Intramuscular
INR	International Normalized Ratio
IO	Intraosseous
IV	Intravenous
IVCD	IntraVentricular Conduction Disturbances
LBBB	Left Bundle Branch Block
LMWH	low-Molecular-Weight Heparin
LPFB	left Posterior Fascicular Block
LVEF	Left Ventricular Ejection Fraction
LVEDV	Left Ventricular End Diastolic Volume
JVP	Jugular Venous Pressure
MAP	Mean Arterial Pressure
MI	Myocardial Infarction
MRA	Magnetic Resonance Angiography
MRI	Magnetic Resonance Imaging
NRCPR	National Registry of Cardiopulmonary Resuscitation
PA	Pulmonary Artery
PE	Pulmonary thromboembolism
RBBB	Right Bundle Branch Block
PEA	Pulseless Electrical Activity
PHA	PseudoHypoAldosteronism
PVCs	Premature Ventricular Contractions
RCTs	Randomized Controlled Trials
RRT	Renal Replacement Therapy
ROSC	Return of Spontaneous Circulation
RV	Right Ventricle
RVOT	Right Ventricular Outflow Tract
SA	Sino-Atrial
SACT	Sino-Atrial node Conduction Time

SCA	Sudden Cardiac Arrest
SCCM	Society of Critical Care Medicine
SCD	Sudden Cardiac Death
SNRT	Sinus Node Recovary Time
SLE	Systemic Lupus Erythematosus
TMT	Treadmill Test
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia
WPW	Wolf Parkinson White
S3/S4	Third/ Fourth heart sound
SC	Subcutaneous

International terms

K^+	Potassium
mEq/L	milliEquivalent/Litre
mg/kg	milligram/kilogram
mmole	millimole
mmHg	millimeter Mercury
Na	Sodium
PaO_2	Partial pressure of oxygen
$PaCO_2$	Partial pressure of carbon dioxide
pH	Negative logarithm of hydrogen ion concentration
α	Alpha
ß	Beta

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Introductions

Asystole is a direct form of cardiac arrest in which the heart stops beating. There is no systole and there is no electrical activity in the heart. The heart is at a total standstill. In the past, the asystolic heart usually could not be restarted. (William et al., 2008**).**

Asystole is defined as the absence of myocardial electrical activity, It should be confirmed by switching between several leads or changing the position of the defibrillation paddles. Most patients with asystole present in a code situation. Persons outside of the hospital who are found to have asystole by the initial responding teem usually have it as a result of profound myocardial ischemia The possibility of a successful outcome in this situation is extremely small. (Brian et al., 2013).

Asystole may be due to profound parasympathetic suppression of both atrial and ventricular activities, stunning of the myocardium due to electrical defibrillation, complete heart block, or prolonged myocardial ischemia. Also, many of the causes of pulseless electrical activity may also lead to asystole.(Brian et al., 2013).

Cardiovascular disease is the most common cause of sudden cardiac arrest, which causes over 60% of adult coronary heart disease deaths, Survival from cardiac arrest depends on a sequence of interventions ,the Chain of Survival comprising (1) early recognition and call for help, (2) early cardiopulmonary resuscitation (CPR), (3) early defibrillation, and (4) postresuscitation care. The division between basic life support (BLS) and

Advanced life support (ALS) is arbitrary the resuscitation process is a continuum. (David et al.,2010).

CPR should be performed immediately on any person who has become unconscious and is found to be pulseless. Assessment of cardiac electrical activity via rapid “rhythm strip” recording can provide a more detailed analysis of the type of cardiac arrest, as well as indicate additional treatment options. Cardiopulmonary resuscitation (CPR) consists of the use of chest compressions and artificial ventilation to maintain circulatory flow and oxygenation during cardiac arrest. Although survival rates and neurologic outcomes are poor for patients with cardiac arrest, early appropriate resuscitation involving early defibrillation and appropriate implementation of post cardiac arrest care lead to improved survival and neurologic outcomes. (Catharine et al., 2013).

Studies have shown that survival falls by 10-15% for each minute of cardiac arrest without CPR delivery. Bystander CPR initiated within minutes of the onset of arrest has been shown to improve survival rates 2- to 3-fold, as well as improve neurologic outcomes at 1 month. (Yasunaga et al,.2010).

Epidemiology and Pathophysiology

Sudden cardiac death (SCD) accounts for 300,000 to 400,000 deaths every year in the United States.The incidence of SCD is 54 to 55 per 100,000 persons (Chugh et al,.2004). Zheng and colleagues reported 63% of all cardiac deaths as SCD (Zheng et al,.2001).The proportion of cardiovascular death from SCD has remained constant over the past several years despite the fact that mortality from cardiovascular cause has decreased (Bakhtiar and Maziar.,2008).

The most common etiology for SCD is coronary artery disease (CAD) followed by cardiomyopathies (Fig,1) . Acute insults including hypoxia, ischemia, acidosis, electrolyte imbalances, and toxic effects of certain drugs may act on the structural substrate and produce arrhythmias leading to SCD and cardiac arrest. (Huikuri et al,.2001).

The presenting rhythm in cardiac arrest is variable, with new studies suggesting a decreasing incidence of VT/VF (21%-32%) for cardiac arrest and a higher incidence of asystole and pulseless electrical activity (PEA) . (Hallstrom et al., 2006),(Ong et al 2006).

In a multicenter, randomized trial studying out of hospital cardiac arrest, 31% of subjects presented with an initial rhythm of VT/VF. In another study with a cohort of 783 out-of-hospital cardiac arrest subjects, 22% presented with an initial rhythm of VT/VF.The National Registry of Cardiopulmonary Resuscitation (NRCPR) reported 25% of initial rhythm in

14,720 victims of in-hospital cardiac arrest as VT/VF.A heart in VT/VF is thought to deteriorate to PEA and asystole with time, conditions which are less responsive to treatment. (Bakhtiar and Maziar.,2008)

The temporal sequence of cardiac arrest can be understood by a 3-phased time sensitive model as proposed by Weisfeldt and Becker (Fig 2). These phases include electrical (lasting 0 to 4 minutes from time of cardiac arrest), circulatory (lasting approximately 4 to 10 minutes from time of cardiac arrest), and metabolic (lasting> 10 minutes from time of cardiac arrest), and they require specific treatment. During the electrical phase, defibrillation is the most effective treatment for cardiac arrest. In the circulatory phase, good quality CPR gains increasing importance along with defibrillation. In the third and final metabolic phase, there is global ischemic injury, where therapeutic strategies that focus on metabolic derangements are critical. Therapeutic hypothermia for comatose survivors of SCD may assist in neurologic recovery at this stage. (Bakhtiar and Maziar.,2008)

Patients with cardiac arrest present both in hospital and out of hospital. The majority of SCDs occur at home and are witnessed by relatives of cardiac arrest victims. In a prospective study of out of hospital SCDs conducted in Europe, bystander interviews were conducted by emergency physicians on site after return of spontaneous circulation (ROSC) or death. The study identified 406 cardiac arrest patients out of 5831 rescue missions. In 72% of the cardiac arrest patients, events occurred at home. Of the witnessed cardiac arrest victims, only 14% received bystander resuscitation even though 66% of witnesses were relatives of the victim. Most notably,

55% of SCD victims reported cardiac symptoms 1 hour prior to collapse. These symptoms included chest pain, syncope, and dyspnea. The majority of SCD victims have a known history of either cardiovascular disease (CVD) or cardiac symptoms. However, almost half of the patients will present without any symptoms and will present as unresponsive with no spontaneous respirations or pulse. (Bakhtiar and Maziar.,2008).

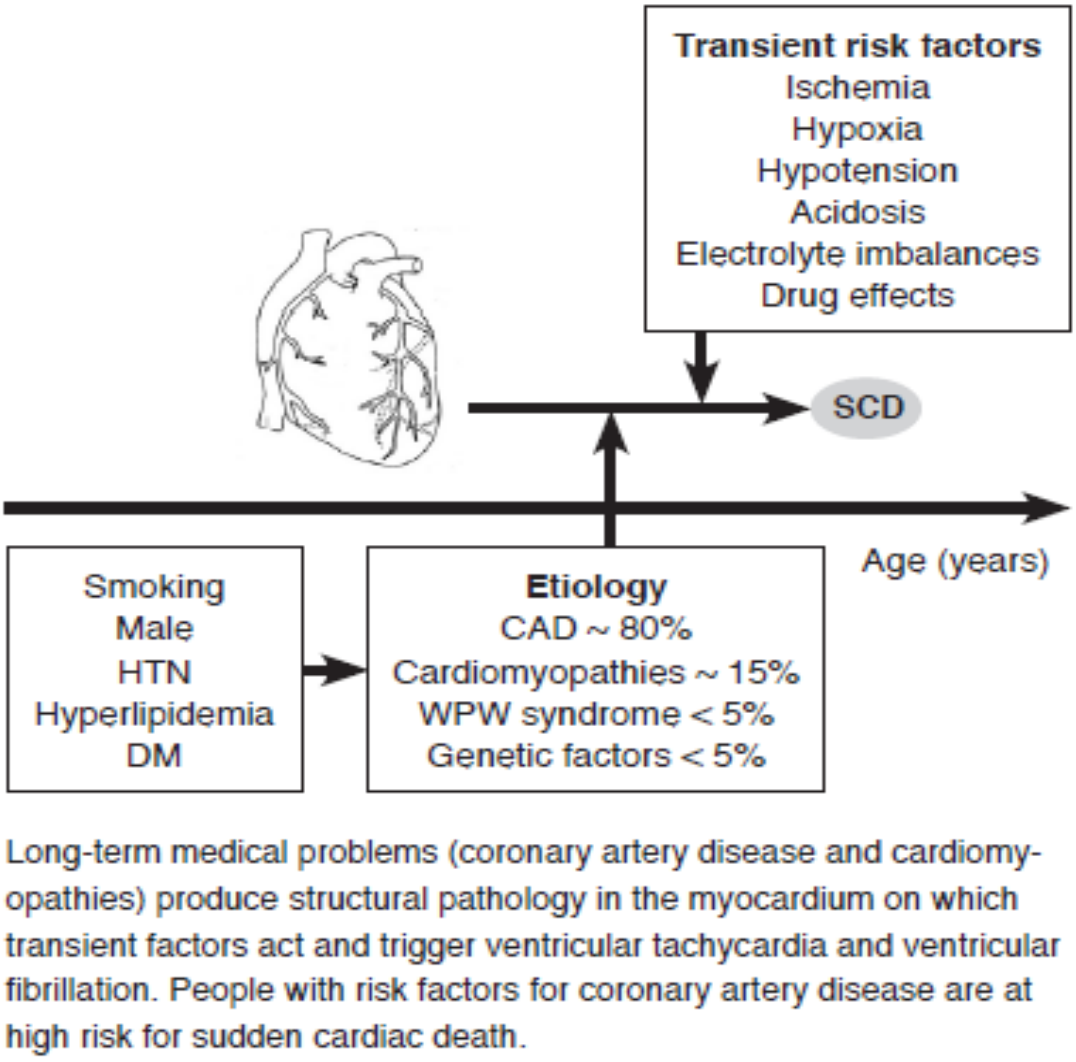
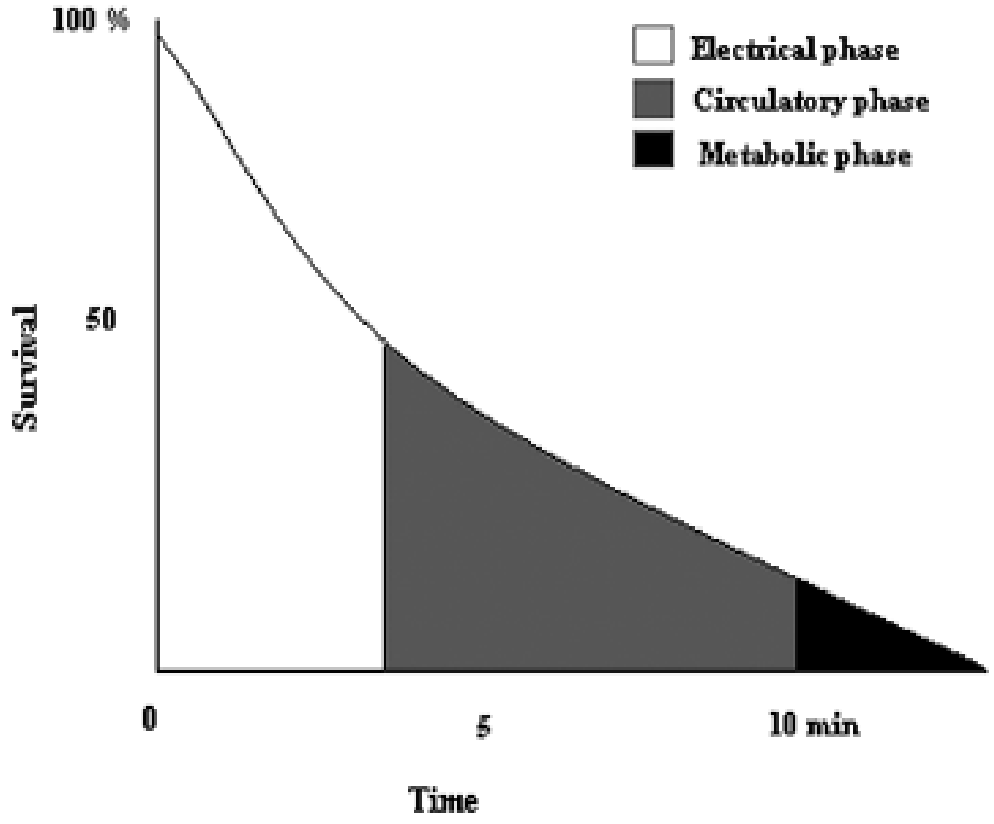


Figure 1. A Confluence Of Risk Factors Act Together To Produce Sudden Cardiac Death Arrest (Weisfeld and Becker. 2002)



This model predicts 50% survival rate for defibrillation provided in the electrical phase where electrical phase = 0 to 4 minutes, circulatory phase = 4 to 10 minutes, and metabolic phase > 10 minutes (based on the model described by Weisfeldt and Becker. JAMA. 2002).

Figure 2. Graphic Representation Of The 3-Phase Time Sensitive Model Of Cardiac Arrest Arrest (Weisfeld and Becker. 2002)

cardiac causes of cardiac asystole

I. Suppression of both Atria and Ventricular activities

1 . Sinus Node Dysfunction
2 . AV Conduction Disturbances
3. Intraventricular Conduction Disturbances (IVCD)

II. Prolonged Myocardial Ischemia

III. Stunning of Myocardium due to Electrical Defibrillion

1. Ventricular Fibrillation

I- Suppression of both Atrial and Ventricular activities

1- Sinus Node Dysfunction.

Sinus node dysfunction encompasses any dysfunction of the sinus node and includes inappropriate sinus bradycardia, SA exit block , SA arrest , and tachycardia bradycardia syndrome. (Myerburg and Castellanos. 2011)

A. Clinical presentation;

There is a wide range of presentations, and some patients' disease may be asymptomatic.

1. Syncope and presyncope are the most dramatic presenting symptoms. Fatigue, angina, and shortness of breath are more subtle consequences of sinus node dysfunction.

2. In the tachycardia bradycardia syndrome, the primary complaint may be palpitation. Documentation of the arrhythmia may be difficult because of the sporadic and fleeting nature of the problem.

B. Etiology;

The intrinsic and extrinsic causes of sinus node dysfunction. Idiopathic degenerative disease is the most common cause of intrinsic sinus node dysfunction, and the incidence increases with age. Acute coronary syndromes are a common cause of bradyarrhythmias, occurring in 25% to 30% of patients with myocardial infarction (MI) (Brian et al., 2013).Table 1,Table 2.

(Table 1) Etiology of ***Intrinsic causes*** sinus node dysfunction. (Brian et al., 2013)

Intrinsic causes Sinus node dysfunction	
Idiopathic degenerative disease	Hypertension
Coronary artery disease	Cardiomyopathy
Infiltrative disorders	Collagen vascular disease
Inflammatory processes	Surgical trauma
Musculoskeletal disorders	Congenital heart disease

(Table 2) Etiology of Extrinsic causes sinus node dysfunction. (Brian et al., 2013)

Extrinsic causes Sinus node dysfunction	
B-BIocking agents	Calcium channel blocking agents
Digoxin	Sympatholytic antihypertensives
Antiarrhythmic drugs	Excessive vagal tone
Carotid sinus syndrome	Increased intracranial pressure
Hyperkalemia	Hypothermia