INTRODUCTION

Cardiac arrest:

Definition

Cardiac arrest, also known as cardiopulmonary arrest, is the cessation of normal circulation of the blood due to failure of the heart to contract effectively.[1] Medical personnel may refer to an unexpected cardiac arrest as a sudden cardiac arrest (SCA). A cardiac arrest is different from (but may be caused by) a heart attack, where blood flow to the muscle of the heart is impaired.[2]

Cardiac arrest is caused when the heart's electrical system malfunctions. In cardiac arrest death results when the heart suddenly stops working properly. This is caused by abnormal, or irregular, heart rhythms (called arrhythmias). The most common arrhythmia in cardiac arrest is ventricular fibrillation. This is when the heart's lower chambers suddenly start beating chaotically and don't pump blood. Death occurs within minutes after the heart stops. Arrested blood circulation prevents delivery of oxygen to the body. Lack of oxygen to the brain causes loss of consciousness, which then results in abnormal or absent breathing. Brain injury is likely if cardiac arrest go untreated for more than five minutes.[3][4] For the best chance of survival and neurological recovery, immediate and decisive treatment is imperative.[5]

For patients with cardiac arrest, survival rates and neurologic outcomes are poor, though early appropriate resuscitation, involving cardiopulmonary resuscitation (CPR), early defibrillation, and appropriate implementation of post–cardiac arrest care, leads to improved survival and neurologic outcomes. Targeted education and training regarding treatment of cardiac arrest directed at emergency medical services (EMS) professionals has significantly increased cardiac arrest survival rates.[6]

Etiology:

Ventricular fibrillation (VF) constitutes the most common electrical mechanism in cardiac arrest, and is responsible for 65 to 80% of occurrences. Another 20-30% is caused by severe Bradyarrhythmias, pulseless electrical activity (PEA) and asystole.[7,8]

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Among adults, ischemic heart disease is the predominant cause. At autopsy 30% of victims show signs of recent myocardial infarction. Other conditions include structural abnormalities, arrhythmias and cardiomyopathies. Secondary cardiac arrest may be elicited by non-cardiac conditions such as hypoxia from a variety of causes, overwhelming infection (sepsis), massive pulmonary embolus, cardiac tamponade, shock, pneumothorax, ventricular rupture, as well as other conditions such as electrocution and near-drowning. Non-cardiac conditions constitute the principal cause of cardiac arrest in-hospital patients. [7,8]

Coronary heart disease (CHD) is the predominant disease process associated with sudden cardiac death in the United States and elsewhere in the developed world. The incidence of CHD in individuals who suffer sudden cardiac death is between 64 and 90%.[9]

Treatable causes:

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease of memory, these are divided into two groups of four based upon their initial letter: either H or T.[10]

They are known as the "4Hs and 4Ts". They are looked for and treated by ambulance technicians/paramedics or by medical staff at the hospital while undertaking advanced life support, protocols for which will be used alongside any specific treatments for each of the causes.[11,12]

The four H's:

1. **Hypovolemia**-A lack of circulating body fluids, principally blood volume. This is usually (though not exclusively) caused by some form of bleeding, anaphylaxis, or pregnancy with gravid uterus. Peri-arrest treatment includes giving IV fluids and blood transfusions, and controlling the source of any bleeding - by direct pressure for external bleeding, or emergency surgical techniques such as esophagogastroduodenoscopy (i.e. esophageal varices) and thoracotomy for internal bleeding. Trauma causes hypovolemia by decreasing blood volume from acute injury or primary

damage to the heart or great vessels. Cardiac arrest secondary to trauma, particularly blunt trauma, has a very poor prognosis.[11,12]

2. **Hypoxia**- A lack of oxygen to the heart, brain and other vital organs. This can be identified through a careful assessment of breath sounds and tube placement. Treatment may include providing oxygen, proper ventilation, and good CPR technique.
3. **Hyper/hypokalemia / Metabolic** (hypoglycemia,acidaemia and other metabolic disorders)- The most life threatening electrolyte derangement is **hyperkalemia** .The classic presentation is the chronic renal failure patient who has missed a dialysis appointment and presents with weakness, nausea, and broad QRS complexes on the electrocardiogram. The most important initial therapy is the administration of calcium, either with calcium gluconate or calcium chloride. Other therapies may include nebulized albuterol, sodium bicarbonate, glucose, and insulin. The diagnosis of hypokalemia can be suspected when there is a history of diarrhoea or malnutrition. Loop diuretics may also contribute.[11,12]

Hyperkalaemia is usually caused by increased potassium release from cells or impaired excretion by the kidneys. There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration higher than 5.5mmol l^{-1}, in practice, hyperkalaemia is a continuum. As the potassium concentration increases, the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration higher than 6.5 mmol l^{-1}. The first indicator of hyperkalaemia may be the presence of ECG abnormalities, arrhythmias, cardiopulmonary arrest or sudden death. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium as well as the rate of increase.

Most patients will have ECG abnormalities at a serum potassium concentration higher than 6.7 $mmoll^{-1}$.[13]

The use of a blood gas analyser that measures potassium can reduce delay in recognition.

ECG changes with hyperkalaemia are usually progressive and include:[11,12]

- First degree heart block (prolonged PR interval (>0.2 sec).
- Flattened or absent P waves.
- Tall peaked (tented) T waves (T wave larger than R wave in more than one lead).
- ST-segment depression.
- S and T wave merging.
- Widned QRS (>0.12 Sec).
- Bradycardia (sinus bradycardia or AV block).
- Ventricualr tachycardia.
- Cardiac arrest (asystole, VF/VT, PEA).

Hypokalaemia is common in hospital patients. Hypokalaemia increases the incidence of arrhythmias, particularly in patient with pre-existing heart disease. Hypokalaemia is defined as serum potassium <3.5 mmol l^{-1}. Severe hypokalaemia is defined as K+ < 2.5 mmol l^{-1} and may be associated with symptoms.

ECG features of hypokalamia are:

- U waves.
- T wave flatting.
- ST segment elevation.
- Arrythmias.
- Cardiorespiratory arrest (VF/VT, asystole, PEA).[11,12]

Metabolic (hydrogen ions, hypoglycemia). **Hydrogen ions (Acidosis)**- An abnormal pH in the body as a result of shock, DKA, renal failure, or tricyclic antidepressant overdose. This can be treated with proper ventilation, good CPR technique, and buffers like sodium bicarbonate. **Hypoglycemia or Hyperglycemia**- Low blood glucose from insulin reactions, DKA, nonketotic hyperosmolar coma. This condition can be suspected when the patient is known to be a diabetic. The treatment may include fluids, potassium, glucose (for hypoglycemia), and insulin (for hyperglycemia).[12]

Typical initial symptoms of hypoglycemia are sweating, anxiety, palpitations, hunger, and tremor, which occur as the plasma glucose

concentration falls below 60 mg/dL (3.3 mmol/L). These symptoms are an important protective response in diabetic patients, warning them that they must ingest glucose.[12]

Hypoglycemia can also cause cognitive dysfunction, which occurs in normal subjects at a plasma glucose concentration of approximately 50 to 55 mg/dL (2.8 to 3.1 mmol/L). Although the brain can adapt to hypoglycemia to some degree, the adaptation is not complete and temporary defects in memory and information processing can occur . As an example, patients may lose the ability to judge correctly when their blood glucose concentration is too low to drive a car safely.[12]

More severe neurologic symptoms occur with progressive hypoglycemia. Lethargy and obtundation begin at a plasma glucose concentration below 45 to 50 mg/dL (2.5 to 2.8 mmol/L); coma ensues at a plasma glucose concentration of about 30 mg/dL (1.7 mmol/L); convulsions at about 20 mg/dL (1.1 mmol/L); and death.[12]

4. **Hypothermia**- A low core body temperature, defined clinically as a temperature of less than 35 degrees Celsius. The patient is re-warmed either by using a cardiac bypass or by irrigation of the body cavities (such as thorax, peritoneum, bladder) with warm fluids; or warmed IV fluids. CPR only is given until the core body temperature reached 30 degrees Celsius, as defibrillation is ineffective at lower temperatures. Patients have been known to be successfully resuscitated after periods of hours in hypothermia and cardiac arrest, and this has given rise to the often-quoted medical truism, "You're not dead until you're warm and dead. "[11]

The four T's:

1. **Tablets or Toxins**- Tricyclic antidepressants, phenothiazines, beta blockers, calcium channel blockers, cocaine, digoxin, aspirin, acetominophen. This may be evidenced by items found on or around the patient, the patient's medical history (i.e. drug abuse, medication) taken from family and friends, checking the medical records to make sure no interacting drugs were prescribed, or sending blood and urine samples to the

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toxicology lab for report. Treatment may include specific antidotes, fluids for volume expansion, vasopressors, sodium bicarbonate (for tricyclic antidepressants), glucagon or calcium (for calcium channel blockers), benzodiazepines (for cocaine), or cardiopulmonary bypass.[11]

2. **Cardiac Tamponade**- Blood or other fluids building up in the pericardium can put pressure on the heart so that it is not able to beat. This condition can be recognized by the presence of a narrowing pulse pressure, muffled heart sounds, distended neck veins, electrical alternans on the electrocardiogram, or echocardiogram. This is treated in an emergency by inserting a needle into the pericardium to drain the fluid (pericardiocentesis), or if the fluid is too thick then an emergency thoracotomy is performed to cut the pericardium and release the fluid.[12]
3. **Tension pneumothorax**- The build up of air into one of the pleural cavities, which causes a mediastinal shift. When this happens, the great vessels (particularly the superior vena cava) become kinked, which limits blood return to the heart. The condition can be recognized by severe air hunger, hypoxia, jugular venous distension, hyperressonance to percussion on the affected side, and a tracheal shift away from the affected side. The tracheal shift often requires a chest x-ray to appreciate. This is relieved in an emergency by a needle thoracotomy (inserting a needle catheter) into the 2nd intercostal space at the mid-clavicular line, which relieves the pressure in the pleural cavity.[11,12]
4. **Thromboembolism**: **(Myocardial infarction)**- If the patient can be successfully resuscitated, there is a chance that the myocardial infarction can be treated, either with thrombolytic therapy or percutaneous coronary intervention.[11,12]

(Pulmonary embolism)- Usually diagnosed at autopsy. Patients in asystole or pulseless electrical activity have a poor prognosis. If this can be detected early, the patient may receive dopamine, heparin, and thrombolytics.[11,12]

In addition to the specific treatments for the causes of cardiac arrest, full resuscitation (using advanced life support protocols) is offered

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to patients as soon as possible, and continues until the patient is either declared dead or regains a pulse and stable heart rhythm.[11]

There are many causes of cardiac arrest. In the developed world most are related to ischaemic heart disease. Table 1 lists other common causes.[12]

Table (I): Causes of Cardiac Arrest.[14]

Cardiac disease	**Respiratory causes**
• Ischaemic heart disease IHD.	Hypoxia (usually causes asystole) ↓O2
• Acute circulatory obstruction	Hypercapnia ↑CO2
Fixed output states	**Metabolic changes**
Cardiomyopathies	Potassium disturbances
Myocarditis	Acute hypercalcaemia
Trauma and tamponade	Circulating catecholamines
Direct myocardial stimulation	Hypothermia
Circulatory causes	**Drug effects**
Hypovolaemia	Direct pharmacological actions
Tension pneumothorax	Secondary effects
Air or pulmonary embolism	**Miscellaneous causes**
Vagal reflex mechanisms	Electrocution
	Drowning
	Neoplasm

Diagnosis:

Cardiac Arrest is an abrupt cessation of pump function (evidenced by absence of a palpable pulse) of the heart that with prompt intervention could be reversed, but without it will lead to death.[15]

In many cases, lack of carotid pulse is the gold standard for diagnosing cardiac arrest, but lack of a pulse (particularly in the peripheral pulses) may be a result of other conditions (i.e. shock, or other conditions leading to poor circulation), or simply an error on the part of the person attempting to diagnose.[14]

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The Resuscitation Council (UK), in line with the European Resuscitation Council (ERC) recommendations and those of the American Heart Association, [16] have suggested that the diagnosis should be made only by healthcare professionals with specific training and expertise, and even then that it should be viewed in conjunction with other indicators such as agonal respiration while the current recommendation of International Liaison Committee on Resuscitation (ILCOR) is that cardiac arrest should be diagnosed in all casualties who are unconscious and not breathing normally. [16]

An ECG clarifies the heart rhythm and guides therapy, but basic life support should begin without awaiting an ECG. The ECG may reveal:[17,18]

- **Asystole** – is a state of no cardiac electrical activity, hence no contractions of the myocardium and no cardiac output or blood flow and no ventricular depolarization . ECG shows flatline.
- **Pulseless electrical activity**- The ECG shows electrical activity that could be consistent with a palpable pulse but no pulse is palpable. It may be because of electromechanical disassociation (EMD) or because the cardiac output is so poor as to not be palpable.
- **Ventricular Fibrillation**- A quivering of the ventricles.
- **Ventricular Tachycardia**- The ventricles contract so rapidly that they do not refill fully between beats, so they do not pump enough blood to maintain circulation. [19]

Pathophysiology:

Although the brain composes only 2% of body weight, it receives 15% of the body's cardiac output and accounts for 20% of the body's overall oxygen use because of its high metabolic activity. Energy expenditures include the synthesis of cellular constituents (e.g., an estimated 2000 mitochondria are reproduced each day by each cell) and neurotransmitter substances, the axoplasmic transport of those substances, and the transmembrane pumping of ions. [20]

When the brain is deprived of adequate blood flow, the resulting ischemia is characterized by a bewildering array of interrelated physiologic and cellular responses that ultimately result in neuronal cell

death. Although this complex cascade of events can be triggered by periods of ischemia lasting only a few minutes, the resulting neuronal death is usually delayed by hours or days. Furthermore, the biology of cerebral cell death after global cerebral ischemia follows (with slight variations) the pattern of delayed cerebral cell death that follows stroke, traumatic brain injury, and other forms of hypoxic or toxic brain injury.[20]

According to etiology:

- Ventricular tachycardia and fibrillation:

The most common electrophysiologic mechanisms leading to SCD are tachyarrhythmias such as ventricular fibrillation (VF) or ventricular tachycardia (VT). There are multiple factors at the organ (eg imbalance of autonomic tone), tissue (eg reentry, wave break, and action potential duration alternans), cellular (eg triggered activity, and automaticity) and subcellular (abnormal activation or deactivation of ion channels) level involved in generation of VT or VF in different conditions. An anatomical or a functional block in the course of impulse propagation may create a circuit with the wave front circling around it and resulting in VT. Other mechanisms such as wave break and collisions are involved in generating VF from VT. While at the tissue level the above-mentioned reentry and wave break mechanisms are the most important known mechanisms of VT and VF, at the cellular level increased excitation or decreased repolarization reserve of cardiomyocytes may result in ectopic activity (eg automaticity, triggered activity), contributing to VT and VF initiation. At the subcellular level, altered intracellular Ca2+ currents, altered intracellular K+ currents (especially in ischemia), or mutations resulting in dysfunction of a sodium channel (Na+ channelopathy) can increase the likelihood of VT and VF.[21]

- Bradyarrhythmia , asystole and pulseless electrical activity:

Approximately 20-30% of patients with documented sudden death events have bradyarrhythmia or asystole at the time of initial contact. Often times, it is difficult to determine with certainty the initiating event in a patient presenting with a bradyarrhythmia because asystole and pulseless electrical activity (PEA) may result from a sustained VT. Less commonly, an initial bradyarrhythmia producing myocardial ischemia may then provoke VT or VF.

- Structural abnormalities:

Most cases of SCD occur in patients with structural abnormalities of the heart. Myocardial infarction (MI) and post-MI remodeling of the heart is the most common structural abnormality in patients with SCD. In patients who survive a myocardial infarction, the presence of premature ventricular contractions (PVCs), particularly complex forms such as multiform PVCs, short coupling intervals (R-on-T phenomenon), or VT (salvos of 3 or more ectopic beats), reflect an increased risk of sudden death. However suppression of the PVCs with antiarrhythmic drugs increases mortality, owing to the proarrhythmic risk of currently available medications.Hypertrophic cardiomyopathy and dilated cardiomyopathy are associated with an increased risk of SCD. Various valvular diseases such as aortic stenosis are associated with increased risk of SCD. Acute illnesses, such as myocarditis, may provide both an initial and sustained risk of SCD due to inflammation and fibrosis of the myocardium.[21]

Less commonly, SCD happens in patients who may not have apparent structural heart disease. These conditions are usually inherited arrhythmia syndromes.

Even though many patients have anatomic and functional cardiac substrates that predispose them to develop ventricular arrhythmias, only a small percentage develop SCD. Identifying the patients at risk for SCD remains a challenge. The strongest known predictor of SCD is significant left ventricular dysfunction of any cause. The interplay between the regional ischemia, LV dysfunction, and transient inciting events (eg, worsened ischemia, acidosis, hypoxemia, wall tension, drugs, metabolic disturbances) has been proposed as being the precipitator of sudden death (see the image below).

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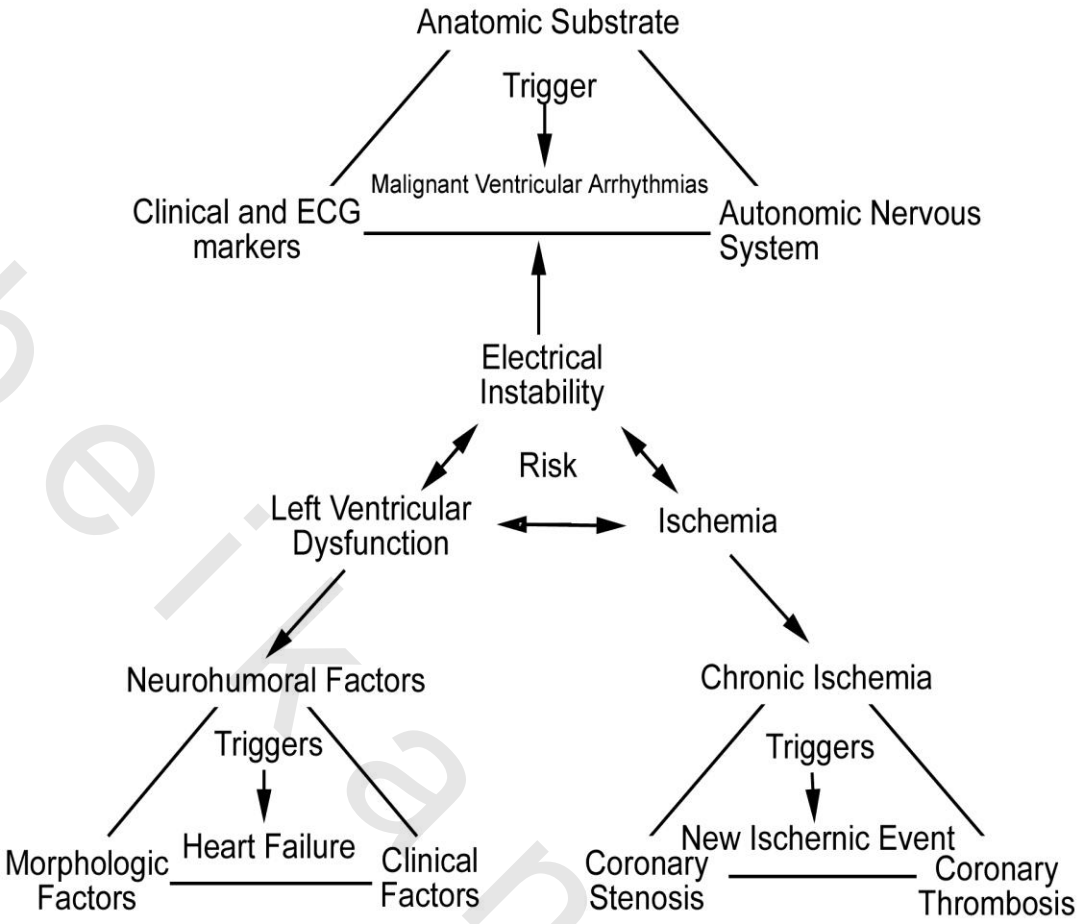

Interplay of various risk factors that can lead to SCD

Epidemiolog:

SCD accounts for approximately 325,000 deaths per year in the United States; more deaths are attributable to SCD than to lung cancer, breast cancer, or AIDS. This represents an incidence of 0.1-0.2% per year in the adult population. SCD is often the first expression of CAD and is responsible for approximately 50% of deaths from CAD. International, the frequency of SCD in Western industrialized nations is similar to that in the United States. The incidence of SCD in other countries varies as a reflection of the prevalence of coronary artery disease or other high-frequency cardiomyopathies in those populations. The trend toward increasing SCD events in developing nations of the world is thought to reflect a change in dietary and lifestyle habits in these nations. It has been

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estimated that SCD claims more than 7,000,000 lives per year worldwide.[22]

Mortality/Morbidity:

Upon emergency department (ED) presentation, the most important determinants of survival include;

(1) an unsupported systolic blood pressure (SBP) greater than 90 mm Hg, (2) a time from loss of consciousness to return of spontaneous circulation (ROSC) of less than 25 minutes, and (3) some degree of neurological responsiveness.

A major adverse outcome from a SCD event is anoxic encephalopathy, which occurs in 30-80% of cases.

Race:

Some studies suggest that a greater proportion of coronary deaths were "sudden" in blacks compared to whites and the percentage of coronary artery disease deaths occurring out of the hospital and in EDs was found to be higher in blacks than in whites. [23]

Sex:

Men have a higher incidence of SCD than women, with a ratio of 3:1. This ratio generally reflects the higher incidence of obstructive coronary artery disease in men. Recent evidence suggests that a major sex difference may exist in the mechanism of myocardial infarction. Basic and observational data point to the fact that men tend to have coronary plaque rupture, while women tend to have plaque erosion. Whether this biologic difference accounts for the male predominance of SCD is unclear.

Age:

The incidence of SCD parallels the incidence of coronary artery disease, with the peak of SCD occurring in people aged 45-75 years. The incidence of SCD increases with age in men, women, whites, and nonwhites as the prevalence of coronary artery disease increases with age. However, the proportion of deaths that are sudden from coronary artery disease decreases with age. In the Framingham study, the proportion of coronary artery disease deaths that were sudden was 62% in

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men aged 45-54 years, but this percentage fell to 58% in men aged 55-64 years and to 42% in men aged 65-74 years. [24] According to Kuller et al, 31% of deaths are sudden in people aged 20-29 years .[25]

Management of cardiac arrest:

The chain of survival:[26]

The interventions that contribute to a successful outcome after a cardiac arrest can be conceptualized as a chain, the chain of survival (Figure 2)

The chain is only as strong as its weakest link; all four links of the chain of survival must be strong. They are:

- Early recognition and call for help.
- Early Cardiopulmonary resuscitation (CPR).
- Early defibrillation.
- Post resuscitation care.

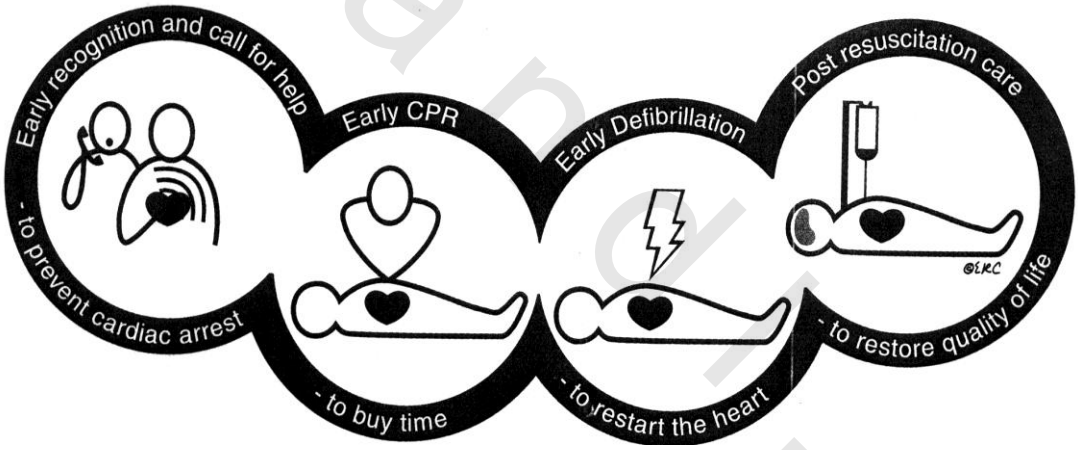

Figure (2): The chain of survival.

Early recognition and call for help:

In hospital, early recognition of the critically ill patient who is at risk of cardiac arrest and a call for the resuscitation team or medical emergency team (MET) will enable treatment to prevent cardiac arrest. A universal number for calling the resuscitation team or MET should be adopted in all hospitals.

Early CPR:

Chest compressions and ventilation of the victim's lungs will slow down the rate of deterioration of the brain and heart. After in-hospital cardiac arrest, chest compression and ventilation must be undertaken immediately. Immediate CPR can double or triple survival from VF Out-of -hospital.[27–30] Performing chest-compression only CPR is better than giving no CPR at all. [31,32]. Interruptions to chest compressions must be minimized and should occur only briefly during defibrillation attempts and rhythm checks.

Early defibrillation:

After out-of-hospital cardiac arrest, the goal is to deliver a shock (if indicated) within 5 min of the EMS receiving the call. In many areas achievement of this goal will require the introduction of public access defibrillation (PAD) programs using automated external defibrillators (AEDs). In hospitals, sufficient healthcare personnel should be trained and authorized to use a defibrillator to enable the first responder to cardiac arrest to attempt defibrillation when indicated, without delay, in virtually ever case. Following VF , cardiopulmonary resuscitation plus defibrillation within 3–5 min of collapse can produce survival rates as high as 49–75%.[33,34] Each minute of delay before defibrillation reduces the probability of survival to discharge by 10–12%.[35]

Post- resuscitation care:

Return of a spontaneous circulation (ROSC) is an important phase in the continuum of resuscitation; however, the ultimate goal is to return the patient to a state of normal cerebral function, a stable cardiac rhythm, and normal haemodynamic function, so that they can leave hospital in reasonable health at minimum risk of a further cardiac arrest. Differences in post-cardiac arrest treatment may account for some of the inter-hospital variability in outcome after cardiac arrest. [37,38]

Advanced life support algorithm:[26]

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander BLS, uninterrupted, high-quality chest compressions and early defibrillation for VF/VT. The use of adrenaline has been shown to increase ROSC, but no resuscitation drugs or advanced airway interventions have been shown to increase survival to hospital discharge aftercardiac arrest.[39,40]

Heart rhythms associated with cardiac arrest are divided into two groups: Shockable rhythms (ventricular fibrillation/ pulseless ventricular tachycardia (VF/VT) and non shockable rhythms (asystole and pulseless electrical activity (PEA). The principle difference in the management of these two group of arrhythmias is the need for attempted defibrillation in patients with VF/VT. Subsequent action, including chest compressions, airway management and ventilation, venous access, administration of adrenaline and the identification and correction of reversible factors, are common to both groups.

Shockable rhythms (VF/ VT):

In adults, the commonest rhythm at the times of cardiac arrest is VF, which may be preceded by a period of VT, by a bradyarrhthmia, or less commonly, supraventricular tachycardia (SVT). Having confirmed cardiac arrest, summon help (including a request for a defibrillator) and start CPR, beginning with chest compressions, with a compression: ventilation ratio of 30:2 as soon as the defibrillator arrives apply self-adhesive pads or paddles to the chest to diagnose the rhythm. If VF/VT is confirmed, follow the treatment step below.[26]

Treatment of shockable rhythms (VF/VT):

- Attempt defibrillation. Give one shock of 150-200 j biphasic (360J monophasic).
- Immediately resume chest compressions (30: 2) without reassessing the rhythm or feeling for a pulse.
- Continue CPR for 2 min, then pause briefly to check the monitor:
- If VF/ VT persist:
- Give a further (2nd) shock of 150-200 J biphasic (360 J monophasic).
- Resume CPR immediately and continue for 2 min.
- Pause briefly to check the monitor.
- If it still VF/VT, give a (3 rd) shock (360-J monophasic or 150–200-J biphasic). Without reassessing the rhythm or feeling for a pulse, resume CPR immediately after the shock, starting with chest compressions.
- If IV access has been obtained, give adrenaline 1mg and amiodarone 300mg once compressions have resumed.

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- Give adrenaline 1 mg IV immediately before alternate shocks (i.e., approximately every 3-5 min).
- Give further shocks after each 2 min period of CPR and after confirming that VF/ VT persist.
- If organized electrical activity compatible with a cardiac output is seen, check for pulse:
- If a pulse is present, start post-resuscitation care.
- If no pulse is present, continue CPR and switch to the non-shockable algorithm.
- If asystole is seen, continue CPR and switch to the non-shockable algorithm.
- If ROSC has not been achieved with the 3rd shock the adrenaline will improve myocardial blood flow and may increase the chance of successful defibrillation with the next shock. In animal studies, peak plasma concentrations of adrenaline occur at about 90 s after a peripheral injection.[41] If ROSC has been achieved after the 3rd shock it is possible that the bolus dose of adrenaline will cause tachycardia and hypertension and precipitate recurrence of VF. However, naturally occurring adrenaline plasma concentrations are high immediately after ROSC, and any additional harm caused by exogenous adrenaline has not been studied. [42]
- The use of up to three-stacked shocks may be considered if VF/VT occurs during cardiac catheterisation or in the early postoperative period following cardiac surgery. This three-shocks trategy may also be considered for an initial, witnessed VF/VT cardiac arrest when the patient is already connected to a manual defibrillator.[26]
- Also the role of the precordial thump is de-emphasised. [26]
- The interval between stopping compressions and delivering a shock must be minimized and certainly should not exceed 10 sec. longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation.
- Chest compressions are resumed immediately after a shock without checking the rhythm or a pulse because even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation and the delay in trying to palpate a pulse will

further compromise the myocardium if a perfusing rhythm has not been restored. If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurring. In the presence of post-shock asystole chest compressions may usefully induce VF.[26]

- When the rhythm is checked 2 min after giving a shock, if a non- shockable rhythm is present and the rhythm is organized (complexes appear regular or narrow), try to palpate a pulse. Rhythm checks must be brief, and pulse checks undertaken only if an organized rhythm is observed. If an organized rhythm is seen during a 2 min period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting return of spontaneous circulation (ROSC). If there is any doubt about the presence of a pulse in the presence of an organized rhythm, resume CPR. If the patient has ROSC, begin post resuscitation care. If the patient's rhythm changes to asystole or PEA, see non- shockable rhythms below.[26]
- There is no evidence that given any anti-arrhythmic drug routinely during human cardiac arrest increase survival to hospital discharge. In comparison with placebo and lidocaine, the use of amiodarone in shock-refractory VF improves the short term outcome of survival to hospital admission. Lidocaine 100 mg IV (or 1-1.5 mg kg-1) may be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone has been given already. Give magnesium (2g IV bolus) for shock-refractory VF if there is any possibility of hypomagnesaemia (e.g. patient treated with diuretic).[26]
- If there is doubt about whether the rhythm is asystole or very fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Very fine VF that is difficult to distinguish from asystole is unlikely to be shocked successfully into a perfusing rhthym.[26]
- Continuing good quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be very fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in

coronary blood flow. If the rhythm is clearly VF attempt defibrillation.[26]

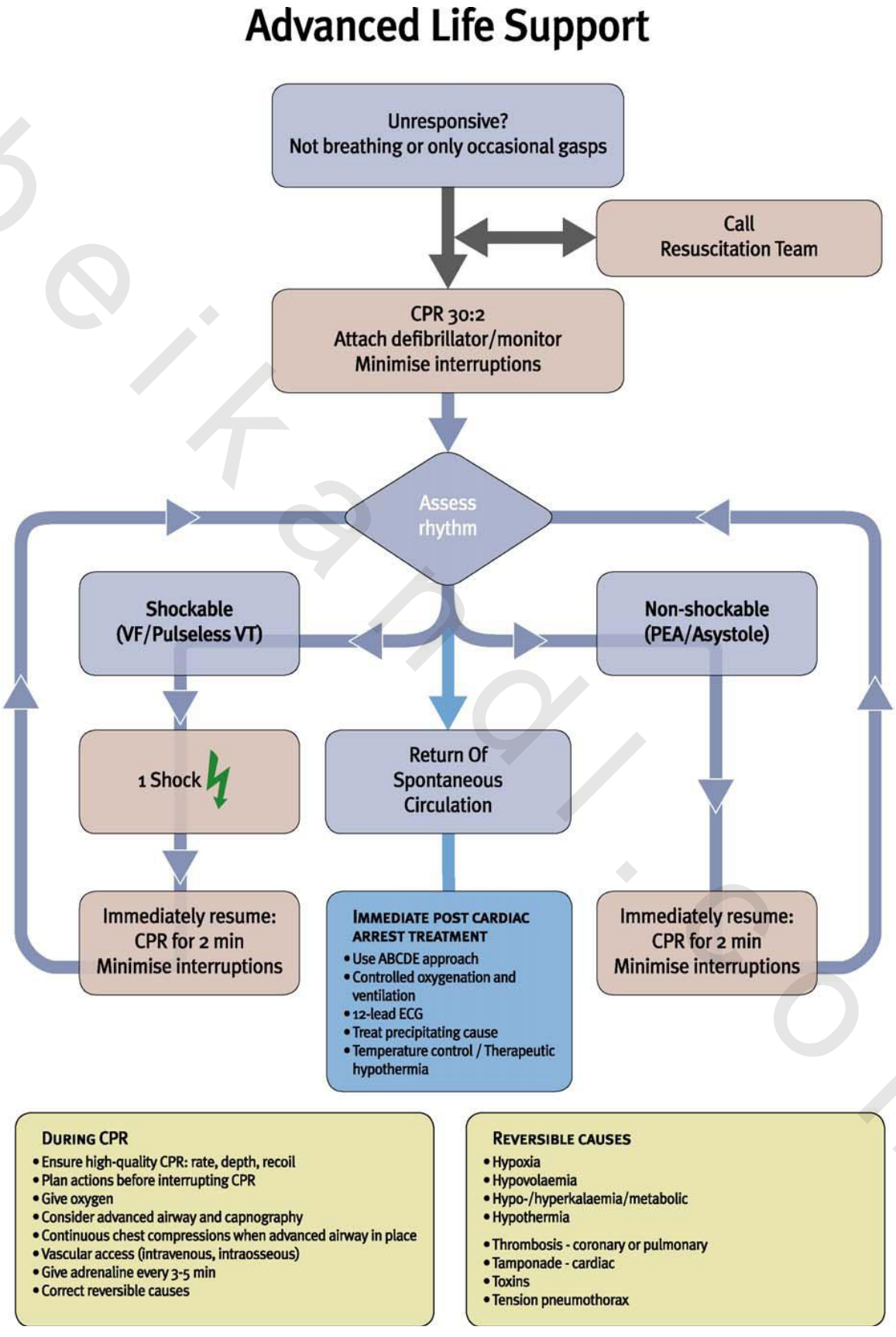

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Figure (3): ALS cardiac arrest algorithm. © 2010 ERC.[26]

Non-shockable rhythms (PEA and asystole):[26]

Pulseless electrical activity (PEA) is defined as organised cardiac electrical activity in the absence of any palpable pulses. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure. PEA may be caused by reversible conditions that can be treated. Survival following cardiac arrest with asystole or PEA is unlikely unless reversible cause can be found and treated quickly and effectively.

Treatment for PEA and asystole [26]

- Start CPR 30:2.
- Give adrenaline 1 mg IV as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured-then continue chest compressions without pausing during ventilation.
- Recheck the rhythm after 2 min:
  - If organized electrical activity is seen, check for a pulse and/or signs of life:

- If a pulse and/or signs of life are present, start post resuscitation care;
- If no pulse and/or no signs of life are present (PEA):
  - Continue CPR;
  - Recheck the rhythm after 2 min and proceed accordingly.
  - Give further adrenaline 1 mg IV every 3-5 min (alternate loops).

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- If VF/VT at rhythm check, change to shockable side of algorithm.
  - If asystole or an agonal rhythm seen at rhythm check:
  - check without stopping CPR, that the leads are attached correctly and continue CPR.
  - Recheck the rhythm after 2 min and proceed accordingly.
  - Give further adrenaline 1 mg IV every 3-5 min (alternate loops).
  - Once an advanced airway has been sited, continue chest compressions without pausing during ventilation.
  - After 2min of CPR, recheck the rhythm. If asystole is present, resume CPR immediately. If an organized rhythm is present, attempt to palpate a pulse. If no pulse is present (or if there is any doubt about the presence of a pulse), continue CPR. Give adrenaline 1mg (IV/IO) every alternate CPR cycle
  - If a pulse is present, begin post-resuscitation care.
  - if the rhythm has changed to VF, follow the algorithm for shockable rhythms.

Atropine:

Routine use for asystole or PEA is no longer recommended. Asystole during cardiac arrest is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. Several recent studies have failed to demonstrate any benefit from atropine in out-of-hospital or inhospital cardiac arrests.[43-45]

During CPR:

During the treatment of persistent VF/VT or PEA/ asystole, emphasis is placed on good quality chest compressions between defibrillation attempts, recognising and treating reversible causes (4 Hs and 4 Ts), obtaining a secure airway, and intravenous access.

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During CPR with a 30:2 ratio the underlying rhythm may be seen clearly on the monitor as compressions are paused to enable ventilation. If VF is seen during this brief pause (whether on the shockable or non-shockable side of the algorithm), do not attempt defibrillation at this stage; instead, continue with CPR until the 2 min period is completed. Knowing that the rhythm is VF, the team should be fully prepared to deliver a shock with minimal delay at the end of the 2 min period of CPR.

The quality of chest compressions and ventilations are important determinants of outcome, yet are frequently performed poorly by healthcare professionals. Providing CPR with a ratio of 30:2 is tiring. As soon as the airway is secured, continue chest compressions without pausing during ventilation. To reduce fatigue, change the individual undertaking compressions every 2 min.

Airway and ventilation:

Patients requiring resuscitation often have an obstructed airway, usually secondary to loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest.

There are three manoeuvres that may improve the patency of an airway obstructed by the tongue or other upper airway structures: head tilt, chin lift, and jaw thrust with the use of nasopharyngeal and oropharyngeal airways to maintain an open airway, particularly when resuscitation is prolonged.

Tracheal intubation provides the most reliable airway but should be attempted only if the healthcare provider is properly trained and has adequate ongoing experience with the technique. Personnel skilled intubation should attempt laryngoscopy without stopping chest compressions a brief pause in chest compressions may be required as

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the tube is passed through the vocal cords. Alternatively, to avoid any interruption in chest compressions, the intubation attempt may be deferred until return of spontaneous circulation. No intubation attempts should take longer than 30 sec: if intubation has not been achieved after this time, recommence bag-mask ventilation. After intubation, confirm correct tube position and secure the tube. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100 min-1, without pausing during ventilation. Ventilate the lungs at 10 breaths min-1. it is important not to hyperventilate the patient. A pause in the chest compressions allows the coronary perfusion pressure to fall substantially. On resuming compressions there is some delay before the original coronary perfusion pressure is restored, thus, chest compressions uninterrupted for ventilation procedure a substantially higher mean coronary perfusion pressure.

In the absence of personnel skilled in tracheal intubation, acceptable alternatives are the combitube, laryngeal mask airway (LMA), ProSeal LMA, or Laryngeal Tube.

During CPR, give oxygen whenever it is available. There are no data to indicate the optimal arterial blood oxygen saturation (SaO2) during CPR, some observational clinical data indicating an association between high SaO2 after ROSC and worse outcome. Initially, give the highest possible oxygen concentration. As soon as the arterial blood oxygen saturation can be measured reliably, by pulse oximeter (SpO2) or arterial blood gas analysis, titrate the inspired oxygen concentration to achieve an arterial blood oxygen saturation in the range of 94–98%.[46]

Intravenous access:[26]

Obtain intravenous access if this has not been done already. Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter

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compared with a peripheral cannula, insertion of a central venous catheter requires interruption of CPR and is associated with several potential complications. Peripheral venous cannulation is quicker, easier, and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid and elevation of the extremity for 10-20 sec to facilitate drug delivery to the central circulation.

Reversible causes:

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest.

- Hypoxia.
- Hypovolaemia.
- Hyperkalaemia, hypokalaemic, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders.
- Hypothermia.
- Tension pneumothorax.
- Tamponade.
- Toxins.
- Thrombosis (pulmonary embolism or coronary thrombosis).

<u>The four Hs:</u>

Minimize the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with 100% oxygen. Make sure there is adequate chest rise and bilateral breath sounds.

Check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is usually due to severe haemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Intravascular

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volume should be restored with fluid, coupled with urgent surgery to stop the haemorrhage.

Hyperkalemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history e.g. renal failure . A 12-lead ECG may show suggestive features. Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose.

Suspect hypothermia in any drowning incident , use a low reading thermometer.

The four Ts:

A tension pneumothorax may be the primary cause of PEA and may follow attempts at central venous catheter insertion. The diagnosis is made clinically. Decompress rapidly by needle thoracocentesis, and then insert a chest drain.

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Cardiac arrest after penetrating chest trauma should raise strong suspicion of tamponade-the need for needle pericardiocentesis or resuscitative thoracotomy.

In the absence of a specific history of accidental or deliberate ingestion, poisoning by therapeutic or toxic substances may be difficult to detect but in some cases may be revealed later by laboratory investigations . Where available, the appropriate antidotes should be used but most often the required treatment is supportive. The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If cardiac arrest is thought

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to be caused by pulmonary embolism consider giving a thrombolytic drug immediately.

Signs of life:

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. exhaled carbon dioxide, arterial blood pressure) appear during CPR, stop CPR briefly and check the monitor. If an organized rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care and/or treatment of peri-arrest arrhythmias if appropriate. If no pulse is present, continue CPR.

Post-resuscitation care:[47]

Introduction:

Return of a spontaneous circulation (ROSC) is an important step in the continuum of resuscitation. However, the next goals is to return the patient to a state of normal cerebral function, and to establish and maintain a stable cardiac rhythm and normal haemodynamic function.[47]

The post-cardiac arrest syndrome, which comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology, often complicates the post-resuscitation phase.[48]

It may not occur at all if the cardiac arrest is brief. Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying

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degrees of neurocognitive dysfunction and brain death. Among patients surviving to ICU admission but subsequently dying in-hospital, brain injury is the cause of death in 68% after out-of-hospital cardiac arrest and in 23% after in-hospital cardiac arrest.[49]

Air and breathing:

Aim: to ensure a clear airway, adequate oxygenation and ventilation.

Patients who have had a brief period of cardiac arrest and have responded immediately to appropriate treatment (e.g. witnessed ventricular fibrillation (VF) reverting to sinus rhythm after early defibrillation) may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation, but should be given oxygen by face mask. Hypoxia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Consider tracheal intubation, sedation and controlled ventilation for patients with obtunded cerebral function. After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia.[47]

Circulation:

Aim: the maintenance of normal sinus rhythm and a cardiac output adequate for perfusion of vital organs.

Cardiac rhythm and haemodynamic function are likely to be unstable following a cardiac arrest. Continuous monitoring of the ECG is essential. Seek evidence of poor cardiac function. Record the pulse and blood pressure and assess peripheral perfusion. Warm, pink digits with a rapid capillary refill usually imply adequate perfusion. Grossly distended neck veins when the patient is semi-upright may indicate right ventricular failure, but in appropriate cases could indicate pericardial

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tamponade. Left ventricular failure may be indicated by fine inspiratory crackles heard on auscultation of the lung fields, and the production of pink frothy sputum. Try to optimize right and left heart filling pressures: measurement of central venous pressure will guide this. Once in a high-care area, the use of non-invasive cardiac output monitoring devices may be valuable. Infusion of the fluids may be required to increase right heart filling pressures or conversely, diuretic and vasodilators may be needed to treat left ventricular failure. Early echocardiography is often helpful in guiding treatment.[47]

Disability and exposure:

Aim: to assess neurological function and ensure that cardiac arrest has not been associated with other medical or surgical conditions requiring immediate treatment.

Although cardiac arrest is frequently caused by primary cardiac disease, other precipitating conditions must be excluded, particularly in hospital patient (e.g. massive blood loss, respiratory failure). Assess the other body systems rapidly so that further resuscitation can be targeted at the patient's needs. To examine the patient properly full exposure of the body may necessary.[47]

Further assessment:

History:

Aim: to establish the patient state of health and regular drug therapy before the cardiac arrest.

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Obtain a comprehensive history as quickly as possible. Those involved in caring for the patient immediately before the cardiac arrest may be able to help (e.g. emergency medical personnel, primary community care physician, and relatives). Specifically, symptoms of cardiac disease should be sought. Consider other causes or cardiac arrest if there is little to suggest primary cardiac disease (e.g. drug overdose, subarachnoid haemorrhage). Make a note of any delay before the start of resuscitation, and the duration of the resuscitation; this may have prognostic significance.[47]

Monitoring

Aim: to enable continuous assessment of vital organ function and to identify trends.

Continuous monitoring of ECG, arterial and possibly central venous blood pressures, respiratory rate, pulse oximetry, capnography, core temperature and urinary output is essential to detect changes during the period of instability that follows resuscitation from cardiac arrest. Monitor continuously the effects of medical interventions (e.g. assisted ventilation, diuretic therapy).[50]

Investigations:

Several physiological variables may be abnormal immediately after a cardiac arrest and urgent biochemical and cardiological investigations should be undertaken.

<u>Full blood count:</u>

To exclude anaemia as contributor to myocardial ischaemia and provide baseline values.

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Biochemistry:

- To assess renal function.
- To assess electrolyte concentrations (K+, Mg+2, and Ca2+).
- To ensure normoglycaemia.
- To commence serial cardiac troponin and enzyme measurement.
- To provide baseline values.

12-Lead ECG:

- To record cardiac rhythm.
- To look for evidence of acute coronary syndrome.
- To look for evidence of old myocardial infarction.
- To provide a baseline record. [47]

Chest radiograph:

- To establish the position of a tracheal tube, a gastric tube, and / or a central venous line.
- To check for evidence of pulmonary oedema.
- To check for evidence of pulmonary aspiration.
- To exclude pneumothorax.
- To assess cardiac contour (accurate assessment of heart size requires standard PA erect radiograph not always practicable in the post-resuscitation situation). [47]

Arterial blood gas:

- To ensure adequacy of ventilation and oxygenation.
- To ensure correction of acid/base imbalance.

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Echocardiography:

- In appropriate patients.
- To identify contributing causes to cardiac arrest.
- To assess LV and RV structure and function.[47]

Patient transfer:

Aim: to transfer the patient safely between the site of resuscitation and a place of definitive care.

Evaluation of the survivor of SCD includes the following: [51-53]

- Identification and treatment of acute reversible causes.
- Evaluation for structural heart disease In patients without obvious arrhythmic triggers or cardiac structural abnormalities.
- Neurologic and psychologic assessment .
- Evaluation of family members .

Outcome of sudden cardiac death:

Outcome according to etiology:

There is an association between the mechanism of SCD and the outcome of initial resuscitation.[54-59]

Asystole — When the initial rhythm is asystole, the likelihood of successful resuscitation is low. Only 10 percent of patients with out-of-hospital arrests and initial asystole survive until hospital admission and only 0 to 2 percent until hospital discharge. The poor outcome in patients with asystole or bradycardia due to a very slow idioventricular rhythm probably reflects the prolonged duration of the cardiac arrest (usually more than four minutes) and the presence of severe, irreversible myocardial damage.[60-63]

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Pulseless electrical activity — Patients who have SCD due to pulseless electrical activity (PEA) (also called electrical-mechanical dissociation) also have a poor outcome. In one study of 150 such patients, 23 percent were resuscitated and survived to hospital admission; only 11 percent survived until hospital discharge.[61-64]

Ventricular tachyarrhythmia — The outcome is much better when the initial rhythm is a sustained ventricular tachyarrhythmia. The most frequent etiology is ventricular fibrillation (VF). Approximately 25 to 40 percent of patients with SCD caused by VF survive until hospital discharge. In the Seattle series cited above of over 12,000 EMS-treated patients with SCD, 38 percent had witnessed VF. Patients with witnessed VF had a significantly greater likelihood of surviving to hospital discharge than those with other rhythms (34 versus 6 percent).[62-66]

Acute myocardial infarction (MI) is the underlying cause of VF in many of the patients who survive to hospital discharge. In a series of 79 such patients from the Mayo Clinic, 47 percent had an acute MI, while in a series of 47 such patients from the Netherlands, 51 percent had an acute MI.[67]

Noncardiac sudden death — As many as one-third of cases of SCD are due to noncardiac causes. Trauma, nontraumatic bleeding, intoxication, near drowning, and pulmonary embolism are the most common noncardiac etiologies. In one series, 40 percent of such patients were successfully resuscitated and hospitalized; however, only 11 percent were discharged from the hospital and only 6 percent were neurologically intact or had mild disability.[68-78]

Factors related to the outcome of resuscitation:[78-97]

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Shortening the time to resuscitation — These observations constitute the rationale for attempts to provide more rapid resuscitation in patients with out-of-hospital SCD. One approach is optimizing the EMS system within a community to reduce the response interval to within eight minutes.[78-83]

Bystander CPR — An initial report from the Seattle Heart Watch program evaluated patients resuscitated at the scene by a bystander trained in CPR and compared their outcomes with those of patients who initially received CPR from EMS personnel.[84-86]

The most important reason for the improvement in survival was that earlier CPR and prompt defibrillation were associated with less damage to the central nervous system. More patients with bystander-initiated CPR were conscious at the time of hospital admission (50 versus 9 percent), and more regained consciousness by the end of hospitalization (81 versus 52 percent).[84-86]

Automatic external defibrillators — The use of automatic external defibrillators (AEDs) by early responders is another approach to more rapid resuscitation. In most but not all studies, AEDs have been found to improve survival after out of hospital cardiac arrest.[87-91]

Adequacy of CPR — End-tidal carbon dioxide levels have an excellent correlation with very low cardiac outputs when measured after at least 10 minutes of CPR, and may provide prognostic information. In one study, for example, an end-tidal carbon dioxide level 10 mmHg, measured after 20 minutes of standard CPR, identified individuals who did not survive to hospitalization discharge with a sensitivity and specificity of 100 percent. Survivors had a much higher end-tidal carbon dioxide concentration at 20 minutes of CPR than nonsurvivors (32.8

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versus 4.4 mmHg). These observations suggest that the cardiac output maintained during CPR is a determinant of outcome.[92,93]

Timing of defibrillation — The standard of care for resuscitation from ventricular fibrillation has been defibrillation as soon as possible.[93]

Induced hypothermia — The induction of mild to moderate hypothermia (target temperature 32 to 34ºC for 24 hours) may be beneficial in patients successfully resuscitated after a cardiac arrest. Improved neurologic outcome and reduced mortality has been demonstrated in series of patients with a VF arrest in whom spontaneous circulation was restored, even when the patient remains comatose after resuscitation.[94-97]

Effect of older age — The risk of SCD increases with age, and older age has been associated with a poorer survival.[97]

Effect of gender — The incidence of SCD is greater in men than women. The effect of gender on outcome was examined in a retrospective cohort study of 9651 men and women. Women were less likely than men to have VF as an initial rhythm (25 versus 43 percent) and were more likely to have pulseless electrical activity/asystole (73 versus 55 percent).[96,97]

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