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Emergency Management of Specific Cardiac Arrhythmias in Adults

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Abstract

Introduction: Resuscitation in emergency setting has been advancing for more than two centuries. In October 2020, the last update of the advanced cardiac life support (ACLS) guidelines was published online in American Heart Association (AHA) journals.

The Aim of Work: In this review, we will discuss the management of specific cardiac arrhythmia in adults. General principles of basics life support, airway management, and post-cardiac arrest management, will not be the focus of this review.

Methodology: We have reviewed the latest version of the advanced cardiac life support (ACLS) and the recent studies that aid the developing of this update.

Conclusion: Early defibrillation and excellent cardiopulmonary resuscitation (CPR) are essential for return of spontaneous circulation (ROSC) in patient with ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT). The management of asystole and Pulseless electrical activity excellent CPR and rapid reversal of underlying causes. The guidelines recommend epinephrine administration as soon as is feasible following CPR initiation. Tachycardia causing instability is best managed with immediate synchronized cardioversion. For bradycardia, the ACLS recommend no intervention unless the patient exhibits evidence of inadequate tissue perfusion related to the slow heart rate.

Keywords: *Cardiac Arrhythmias; Cardiac Life Support; Cardiac Arrest; Cardiac Resuscitation*

Introduction

Resuscitation in emergency setting has been advancing for more than two centuries [1]. Early in the 18th century, the Paris Academy of Science recommended mouth-to-mouth breathing support for drowning victims [2]. Later that century, Dr. Friedrich Maass performed the first documented chest compressions on humans [3]. In 1963, the American Heart Association (AHA) endorsed cardiopulmonary resuscitation (CPR) for the first time, and by 1966, they had adopted their standardized CPR guidelines [2]. The last update of the advanced cardiac life support (ACLS) guidelines was published in October 2020 in the journal Circulation [4,5].

In this review, we will discuss the management of specific cardiac arrhythmia in adults. General principles of basics life support, airway management, and post-cardiac arrest management, will not be the focus of this review.

Methods

We have reviewed the latest version of the advanced cardiac life support (ACLS) and the recent studies that aid the developing of this update. Additionally, a thorough search of medical literature in the last 2 decades was conducted through PubMed and google scholar search engine. The terms used in the search include cardiac arrhythmias, cardiac life support, cardiac arrest, and cardiac resuscitation.

Management of sudden cardiac arrest (SCA)

Non-perfusing rhythms emerging from the ventricles such as ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) are fatal if not identified and managed promptly. Early defibrillation and excellent cardiopulmonary resuscitation (CPR) are essential for return of spontaneous circulation (ROSC) in these patient. The most recent algorithm produced by American Heart Association (AHA) for the management of cardiac arrest can be accessed online [6]. Excellent CPR entails continuity without interruption until defibrillation is readily available and adequate spontaneous circulation is achieved. Treatable underlying causes should be identified and managed as quickly as possible [7]. Treatable causes of cardiac arrest include, but not limited to, acidosis (due Diabetes, diarrhea, drug overdose, renal dysfunction, sepsis, or shock), anemia, hypo- and hyperkalemia, and cardia tamponade.

The initial step is performing excellent chest compressions as soon as SCA is recognized and continue while the defibrillator is being attached. The compression should be continued until the defibrillator is obtained, if not immediately available. Once the defibrillator is readily available, it should be attached to the patient and charged while continuing CPR. Once charged, the compressions should be stopped to assess the rhythm and defibrillate if shockable rhythm as ventricular fibrillation or pulseless ventricular tachycardia is present. In case of asystole or pulseless electrical activity (PEA), CPR should be continued. Rescuer should resume CPR immediately after any shock is given. Successful conversion to a perfusing rhythm, and patient survival, is strongly associated with shorter time to defibrillation. Hence, monitored patient who undergo VF or pVT arrest, prompt charge and deliver of the shock should be performed before initiating CPR if a defibrillator is immediately available and pads are in place. This is based on the fact that the ten seconds or fewer of CPR are unlikely to generate adequate perfusion.

Biphasic defibrillators are more efficacious at lower energy level [8]. The guidelines recommend using the initial dose of energy recommended by the manufacturer (usually 120 to 200 J). If this dose is not known, rescuer could apply the maximal dose. Experts tend to prefer the maximal energy does cardiac arrest due to VF or pVT. Double sequential defibrillation means performing two defibrillation attempts in rapid succession. There is no sufficient evidence regarding the effectiveness of this method for refractory VT or pVT. The last update states that this approach not be used routinely [5,9].

According to the guidelines, CPR should be resumed CPR immediately after defibrillation without checking for a pulse. The stop for checking the rhythm should occur only after two minutes of CPR and after fully charging the defibrillator to be ready for discharge. After at least one attempt of defibrillation and 2 minutes of CPR, epinephrine should be used if VT or pVT persist. The dose of epinephrine is 1 mg (intravenous or intraosseous) every three to five minutes while CPR is performed [10,11]. Premature administration of epinephrine within two minutes of defibrillation has been associated with decreased survival [12]. Vasopressin is less effective than epinephrine and hence it has been removed from the treatment algorithm [10,11].

Antiarrhythmic drugs carries little benefit in refractory VF or Pvt [7,13]. The current guideline suggest the possible use in certain situations, however, without specifying the timing. Some experts suggest that they could be considered after a second unsuccessful defibrillation attempt in anticipation of a third shock. Antiarrhythmic drugs include amiodarone, lidocaine, and magnesium sulfate. Amiodarone or lidocaine may be administered in VF or pVT unresponsive to defibrillation, CPR, and epinephrine. Magnesium sulfate may be used to

treat polymorphic ventricular tachycardia consistent with torsade de pointes, however, it is not recommended for routine use in adult cardiac arrest patients.

Refractory VF or pVT may be caused by an acute coronary syndrome (ACS). In such situation, percutaneous coronary intervention can be attempted after successful resuscitation and the procedure is feasible. It is worth mentioning that the electrocardiogram (ECG) may be unreliable for ACS after cardiac arrest and specialist consultation is needed after return of spontaneous circulation (ROSC) [14]. The guideline of ACLS consistently advocate for the use of advanced airway management and treatment with specific medications in sudden cardiac arrest. However, these interventions have not been shown to improve survival in SCA. Therefore, attempts of these interventions must never be considered at the expense of performing excellent CPR and early defibrillation.

Asystole and pulseless electrical activity (PEA) are considered non-perfusing rhythms requiring the prompt initiation of excellent CPR. Asystole is defined as a complete absence of demonstrable electrical and mechanical cardiac activity. Pulseless electrical activity (PEA) is defined as any electrocardiographic rhythms without sufficient mechanical contraction of the heart to produce a palpable pulse or measurable blood pressure. The advanced cardiac life support guideline addresses asystole and PEA together because successful management for both depends on excellent CPR and rapid reversal of underlying causes [15]. The guidelines recommend epinephrine administration as soon as is feasible following CPR initiation [4,5].

Reversible causes include hypoxia, hyperkalemia, poisoning, and hemorrhage. Asystole may be the result of a primary or secondary cardiac conduction abnormality, possibly due to end-stage tissue hypoxia and metabolic acidosis, or occasionally due to excessive vagal stimulation. It is important to recognize and treat all potential secondary causes of asystole or PEA as soon as possible. Since stress pneumothorax and cardiac tamponade make CPR ineffective and are most easily reversible, the clinician should not hesitate to perform immediate needle thoracostomy or pericardiocentesis if thought necessary. Hesitation can worsen patients' outcomes while there is little chance that either intervention will make the situation worse.

After initiating CPR and considering possible reversible causes, epinephrine should be administered in a dose of 1 mg every 3 to 5 minutes intravenously as soon as feasible [5,10,11]. It is worth mentioning that data regarding the benefit of epinephrine in patients with asystole or PEA are inconsistent and further study is needed.[10,16] Neither asystole nor PEA responds to defibrillation. The guidelines no longer recommend the use of atropine for the treatment of cardiac arrest due to asystole or PEA. Cardiac pacing is ineffective for cardiac arrest and is not recommended.

Management of bradycardia

Heart rate below 60 beats per minutes is commonly define bradycardia. This definition, however, is conservative as symptomatic bradycardia generally entails rates below 50 beats per minute. The advanced cardiac life support guidelines recommend no intervention unless the patient exhibits evidence of inadequate tissue perfusion thought to result from the slow heart rate [17].

Signs and symptoms of inadequate perfusion include hypotension, altered mental status, signs of shock, persistent ischemic chest pain, and suggestive of acute pulmonary edema. Bradycardia could be caused commonly by hypoxemia of respiratory origin. Once a sign of hypoperfusion is apparent, clinician should determine oxyhemoglobin saturation using a pulse oximeter. If pulse oximetry is unavailable, patient should be assessed for signs of respiratory failure such as increased or decreased respiratory rate, diminished respiratory volume, or retractions. Bradycardia in the intubated patient should be considered die misplacement of the tube into esophageal or displacement, until proven otherwise.

Atropine is the agent of choice in cardiac bradycardia while simultaneously preparing for immediate temporary cardiac pacing and/or infusion of a chronotropic agent for significant bradycardia due to selected causes. Causes of bradycardia that benefits from atropine include high-vagal tone, drug-induced bradycardia, and high-degree AV block with a narrow QRS complex. Atropine should be avoided in

bradycardia thought to be due to a conduction disturbance at or below the Bundle of His as in wide QRS complex in complete heart block, or Mobitz type II second degree AV block. In this situation, effort should be directed to cardiac pacing and/or administration of a chronotropic agent. The initial dose of atropine is 0.5 mg IV. This dose may be repeated every 3 to 5 minutes to a total dose of 3 mg.

Transvenous temporary cardiac pacing should be initiated promptly, when possible, followed by expert consultation. If transvenous pacing cannot be initiated promptly, transcutaneous pacing could be used followed by chronotropic infusion. Before using transcutaneous pacing, clinician should ensure adequate sedation and analgesia if the patient can perceive the pain associated with this procedure. Further consultation with cardiologist is required for these patients as they may permanent pacemaker placement. For patients who remain symptomatic following atropine administration and when temporary cardiac pacing is not readily available or successful in alleviating symptoms, continuous infusion of a chronotropic agent is indicated. Dopamine or epinephrine could be used, but not both. The starting dose for dopamine infusions is 2 to 20 mcg/kg per minute, while epinephrine is started at 2 to 10 mcg per minute. Chronotropic agent should be titrated to clinical response.

Management of tachycardia

The classic definition of tachycardia entails a heart rate above 100 beats per minute. However, tachycardia is not usually symptomatic below 150 beats per minute unless patient has an underlying ventricular dysfunction [7,17]. The presence and severity of symptoms play important role in the management of tachyarrhythmia. First step in management is directed to determine stability of the patient as the presence of ongoing ischemic chest pain, acute mental status changes, hypotension, signs of shock, or signs of acute pulmonary edema. Similar to bradycardia, hypoxemia is a common cause of tachycardia. The clinician should look for signs of difficult breathing such as increased respiratory rate, retractions, paradoxical abdominal breathing and low oxygen saturation.

Tachycardia causing instability is best managed with immediate synchronized cardioversion. The exception is sinus tachycardia [18]. Some cases of supraventricular tachycardia may respond to a bolus of adenosine (6 to 12 mg IV) without the need of cardioversion. Whenever possible, determine whether the patient may feel the pain associated with cardioversion and if so, provide sufficient sedation and analgesia.

Stable patients tolerate further assessment of the nature of the arrhythmia. However, this may not be readily feasible in the urgent setting of ACLS. The assessment should provide answer for 3 main questions: whether the rhythm is sinus or not, regular or irregular, and whether the QRS complex is wide or narrow.

Sinus tachycardia and supraventricular tachycardia are the primary causes of narrow complex arrhythmia [15,17]. Sinus tachycardia is a common response to fever, anemia, shock, sepsis, pain, heart failure, or any other physiological stress. No drugs are needed to treat sinus tachycardia; care is focused on treating the underlying cause.

Supraventricular tachycardia (SVT) is a normal tachycardia most commonly triggered by a reentering process within the conduction system. The QRS complex is often narrow, but can be longer than 120 ms with concomitant bundle branch block. Vagal maneuvers as Valsalva and carotid sinus massage could be used during other therapies preparing. The mechanism is blocking conduction through the AV node that result in interruption of the reentrant circuit. Vagal maneuvers alone can convert up to 25 percent of SVTs to sinus rhythm, while Valsalva followed immediately by supine repositioning with a passive leg raise is evidenced to be even more effective. Adenosine is the agent of choice for SVT refractory to vagal maneuvers [19,20].

Owing to its extremely short half-life, adenosine (6 to 12 mg IV) is injected as quickly as possible into a broad proximal vein followed immediately by a 20 mL saline flush and an elevation of the extremity to ensure that the drug reaches the central circulation until it is metabolized. If the first dose of adenosine does not convert the rhythm, the second and third doses of 12 mg IV will be given. Larger doses may be required in patients taking theophylline or theobromine or consuming significant quantities of caffeine; smaller doses should be

given to patients taking dipyridamole or carbamazepine, patients with transplanted hearts, or with central vein injection. Patient should be counseled about the transient side effects of adenosine such as chest discomfort, dyspnea, and flushing. Clinician should reassure that these effects are very brief. Continuous ECG recording during adenosine administration is advised. Failure of conversion for sinus rhythm raises the possibility of other etiologies including atrial flutter or a non-reentrant SVT, which may become apparent on the ECG when AV nodal conduction is slowed. Rate control could be achieved with either an intravenous nondihydropyridine calcium channel blocker or a beta blocker in case conversion attempts should fail. Possible agent for rate control include diltiazem, verapamil, metoprolol, and atenolol.

Irregular narrow-complex tachycardia is caused mostly by atrial fibrillation. Other possible causes include atrial flutter with variable atrioventricular (AV) nodal conduction, multifocal atrial tachycardia (MAT), or sinus tachycardia with frequent premature atrial complexes (PACs) [7,15]. In stable patients, the goal of initial treatment is to control heart rate. To achieve this, either nondihydropyridine calcium channel blocker (CCB) or beta blocker can be used. Example of nondihydropyridine CCB include diltiazem (15 to 20 mg IV over 2 minutes, repeat at 20 to 25 mg IV after 15 minutes) and verapamil (2.5 to 5 mg IV over 2 minutes followed by 5 to 10 mg IV every 15 to 30 minutes). Metoprolol is common example of beta blocker; the regimen is 5 mg IV for 3 doses every 2 to 5 minutes; then up to 200 mg PO every 12 hours. Clinician should aware that CCB and beta-blockers may cause or worsen hypotension. Patients should be closely monitored while the drug is given. Elderly are especially prone to develop sever hypotension and often require loading doses that are below the usual range. Combination therapy with a beta blocker and calcium channel blocker increases the risk of severe heart block.

Practically, diltiazem is usually used for the management of acute atrial fibrillation with rapid ventricular response while beta blockers are more effective for chronic rate control. In addition, beta blockers are preferred in the setting of an acute coronary syndrome. For atrial fibrillation associated with hypotension, amiodarone may be used as 150 mg IV over 10 minutes followed by 1 mg/min drip for six hours, and then 0.5 mg/min. However, the possibility of conversion to sinus rhythm must be considered in this setting [21]. Atrial fibrillation associated with acute heart failure could be managed by amiodarone or digoxin for rate control. Multifocal atrial tachycardia treatment involves the correction of potential precipitants, such as hypokalemia and hypomagnesemia. The ACLS Guidelines suggest cardiologist consultation for these arrhythmias.

Cardioversion of healthy patients with abnormal narrow complex tachycardia should NOT be done without taking into account the possibility of embolic stroke. If the length of atrial fibrillation is known to be less than 48 hours, the risk of embolic stroke is low and the physician can recommend electrical or chemical cardioversion [22]. A number of medications can be used for chemical cardioversion and the best choice varies according to clinical circumstance.

Regular wide complex tachycardia generally arise from the ventricle. Less frequently, this could be caused by aberrantly conduct supraventricular tachycardia. The differentiation between ventricular tachycardia (VT) and SVT with aberrancy as a cause of regular wide complex tachycardia can be difficult. Hence, the management is based on the arbitrary assumption that VT is present. Adenosine may be used in cases of regular wide-complex monomorphic QRS for diagnosis and treatment. Adenosine should NOT be given to unstable patients, irregular wide-complex rhythm, or a polymorphic QRS complex. Adenosine is unlikely to affect ventricular tachycardia but is likely to slow or convert SVT with aberrancy. The regimen is identical to that used for SVT.

Stable patient with wide-complex tachycardia could be managed with antiarrhythmics drugs or elective synchronized cardioversion [7,15,17]. Other antiarrhythmics that may be used to treat stable patients with regular, wide-complex tachycardia include procainamide, amiodarone, and sotalol. Procainamide is given as 20 mg/min IV and continued until the arrhythmia is suppressed, the patient becomes hypotensive, the QRS widens 50 percent beyond baseline, or a maximum dose of 17 mg/kg is administered. Procainamide and sotalol should be avoided in patients with a prolonged QT interval. The regimen of amiodarone is 150 mg IV given over 10 minutes and could be repeated as needed to a total of 2.2 g IV over the first 24 hours. If pharmacologic management does not successfully control the wide-complex tachycardia, elective cardioversion may be needed. The ACLS Guidelines recommend expert consultation for all patients with wide complex tachycardia.

When the diagnosis of aberrantly conducted supra ventricular tachycardia is definite as with old ECG that demonstrates bundle branch block, the management may follow the same principles of that of narrow-complex SVT, with vagal maneuvers, adenosine, or rate control.

Irregular wide complex tachycardia could be caused by atrial fibrillation with preexcitation as in Wolf Parkinson White syndrome, atrial fibrillation with aberrancy such as with bundle branch block, or by polymorphic ventricular tachycardia (VT)/torsades de pointes [7,15,17]. The clinician must aware that attempts to block atrioventricular (AV) nodal conduction in wide complex irregular tachycardia of unclear etiology may be fatal and hence, contraindicated. This is because it may precipitate ventricular fibrillation (VF). Medication that could cause AV nodal block include beta blockers, calcium channel blockers, digoxin, and adenosine. To avoid inappropriate and possibly dangerous treatment, the advanced cardiac life support guidelines suggest that rescuer should assume that any irregular wide complex tachycardia is caused by preexcited atrial fibrillation.

The typical presentation of irregular wide-complex tachycardia caused by preexcited atrial fibrillation is extremely fast heart rates more than 200 beats per minute. The best initial step in management is prompt electric cardioversion. Antiarrhythmic agents such as procainamide, amiodarone, or sotalol may be used in cases of ineffective or unfeasible electrical cardioversion, or when atrial fibrillation recurs. The ACLS Guidelines recommend expert consultation for all patients with a wide range of complex tachycardia. The dosage for these agent is identical to the previously described. Magnesium sulfate could be given in initial dose of 2 g IV followed by a maintenance infusion to prevent polymorphic VT associated with familial or acquired prolonged QT syndrome [23].

When the diagnosis of atrial fibrillation and wide-complex QRS due to a preexisting bundle branch block is *sure*, stable patient could be managed in the same manner as a narrow-complex atrial fibrillation. The presence of preexisting bundle branch block could be ensured by record of diagnosis and/or old ECG.

Conclusion

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) are fatal if not identified and managed promptly. Early defibrillation and excellent cardiopulmonary resuscitation (CPR) are essential for return of spontaneous circulation (ROSC). Treatable underlying causes should be identified and managed as quickly as possible. Biphasic defibrillators are more efficacious at lower energy level. There is no sufficient evidence regarding the effectiveness of double sequential defibrillation for refractory VT or pVT. Antiarrhythmic drugs carries little benefit. The management of asystole and Pulseless electrical activity excellent CPR and rapid reversal of underlying causes. The guidelines recommend epinephrine administration as soon as is feasible following CPR initiation.

The presence and severity of symptoms play important role in the management of tachyarrhythmia. The assessment should provide answer for 3 main questions: whether the rhythm is sinus or not, regular or irregular, and whether the QRS complex is wide or narrow. Tachycardia causing instability is best managed with immediate synchronized cardioversion. For bradycardia, the ACLS recommend no intervention unless the patient exhibits evidence of inadequate tissue perfusion related to the slow heart rate.

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Volume 17 Issue 1 January 2021