

Interventions for Patients with Returned of Spontaneous Circulation

Student's Name

Institutional Affiliation

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Introduction

Return of spontaneous circulation (ROSC) refers to a renewal of cardiac activity related to a noteworthy respiratory effort after the occurrence of cardiac arrest. The major signs of ROSC include coughing, breathing, a measurable blood pressure, and pulse movements. Defibrillation and cardiopulmonary resuscitation increase the probability of ROSC. There is heightened recognition that consistent post-cardiac arrest care after ROSC contributes to and improves the potential of a patient's survival with a good quality of life. These assumptions rest on the publication of the outcome of randomized controlled trials alongside the description of the post-cardiac arrest symptoms. Post arrest care has a potential of reducing early mortality caused by hemodynamic instability and later mortality and morbidity caused by brain injury and multi-organ failure. These are the common issues encountered among patients initially resuscitated from cardiac arrest. In some cases, spontaneous circulation does not follow a peripheral venous or IO drug administration or defibrillation. Therefore, the health care specialist may take into consideration a placement of a central line if there are no contraindications.

The core essence of post-cardiac arrest care is to optimize vital organ perfusion and cardiopulmonary function. After an out-of-hospital cardiac arrest, one should transfer the patient to a suitable hospital comprising an all-inclusive post-cardiac arrest treatment system of care, which includes neurological and goal-directed critical care, acute coronary interventions, and hypothermia. Post-cardiac arrest care also aims at controlling body temperature to optimize neurological recovery and survival. Furthermore, its goal is also to reduce the risk of multi-organ injury and support organ function whenever required. This paper reviews literature on effective

intervention measures for patients with returned of spontaneous circulation from A-systole, pulse electrical activity (PEA), ventricular fibrillation (VF), and ventricular tachycardia (VT).

Empirical Literature Review

Cardiac arrest has remained a risky disorder despite advancement in cardiac life support. Patients who happen to undergo A-systole, VT, or electromechanical dissociation are in greater danger. Physicians have increasingly used epinephrine as an adrenergic agent for such patients. The current recommended dosage of 1mg for adults was established after conducting studies that involved dogs. However, the dosage does not permit a larger difference between humans and dogs (Lyon et al., 2009).

Interventions in the Patient with Returned of Spontaneous Circulation from A-systole

In medical perspective, A-systole is a condition of no cardiac electrical activity, and, thus, no contractions of the myocardium and no blood flow or cardiac output. A-systole is among the conditions, which a medical practitioner may utilize in certifying a legal or clinical death. A conventional treatment of A-systole involves cardiopulmonary resuscitation (CPR) which may be combined with intravenous vasopressor such as epinephrine (adrenaline). Studies have demonstrated that administration of epinephrine in such cases is quite effective. There have been claims of a favorable though not a significant impact on the use of epinephrine in the enhancement of a long-term survival for A-systole patients. When used to treat cardiac arrest and other cardiac dysrhythmias disorders such as A-systole, it leads to the elimination of cardiac output. It mainly helps to increase resistance through α_1 receptor-dependent vasoconstriction and to increase cardiac output through its binding β_1 receptors. On the other hand, epinephrine is the preferred drug in treating anaphylaxis. In this respect, practitioners use the drug to decrease the immune response to the administered allergen (Kochansky, 2010).

Hypokalemia can serve an example when, in some cases, the underlying reversible cause may be identified and treated. There are also various other recommendations for interventions for A-systole patients though considered outdated. They include defibrillation and intravenous atropine widely used before physicians started to consider them ineffective. An effective treatment for A-systole is standard 1 mg epinephrine by IV every 3-5 minutes as required. Moreover, medical staff can also prescribe vasopressin 40 units by IV every 3-5 minutes instead of the first and second doses of epinephrine. However, such treatment may not always enhance the results (Mattana & Singhal, 2003).

Dieckmann and Vardis (2005) found that high-dose epinephrine (HDE) increased the rate of spontaneous circulation. However, their analysis could not confirm that it improved survival rates. The authors further noted that the significant effect of HDE among animals, including swine and dogs, did not always have a similar effect among older diseased human beings (Dieckmann & Vardis, 2005). Therefore, it implies that doctors should not apply studies, which had relied on animal samples, to human beings.

Gueugniad et al. (2008) raised a concern in their study that HDE had an adverse impact on some groups of patients. The findings generated from animal models indicated that fibrillating heart consumes oxygen at a fast pace. In addition, HDE has a probability of increasing myocardial oxygen demand without necessarily increasing the availability of oxygen at the cellular level during the course of resuscitation. Another side effect caused by the use of HDE was contraction band necrosis, one of the quintessential features of myocardial damage normally caused by high concentration of epinephrine or catecholamine. Other unwanted effects include sustained ventricular arrhythmias and, in some cases, induces ventilation and defects of perfusion.

Some analysts have focused on the issue of HDE advanced cardiac life support (ACLS) protocols. However, these analysts have highlighted the need for more rigorous data from randomized and controlled trials. Several controlled trials have not indicated any improved rate of survival with the use of HDE. During the past decade, both controlled and experimental studies have stated that patients taking HDE have the probability of improving the outcome of cardiac arrest or anaphylaxis. Unfortunately, these are only a few studies, which have presented positive results in support of such claims. On the other hand, numerous studies have not found any significant variation in survival rates between patients treated with HDE and those taking a standard dose (SDE) (Ian, 2012).

Interventions in the Patients with Returned of Spontaneous Circulation from Pulse

Electrical Activity

PEA is a clinical disorder characterized by unresponsive and inadequate palpable pulse during the occurrence of organized cardiac electrical activity regarded as electromechanical dissociation. In most cases, the lack of ventricular electrical activity implies a deficiency of ventricular mechanical activity. However, the opposite case is not true. Stated differently, though the electrical activity may be essential, it is not an adequate condition for mechanical activity. In the context of a cardiac arrest, the expressive ventricular mechanical activity does not necessarily supplement the prevalence of organized ventricular electrical activity. The term expressive here helps to determine the degree of ventricular mechanical activity, which is sufficient to generate a palpable pulse (AHA, 2010).

The common denominator in the definition of PEA is the spontaneous cardiac electric activity in the absence of blood flow, which is adequate enough in maintaining consciousness. It is also used in reference to the swift spontaneous return of sufficient organ consciousness and

perfusion. The latter qualifier does not include transient losses of blood flow as vasovagal syncope, which has clinical implications that differ from those of true PEA.

Extensive studies have been carried out concerning the efficacy of vasoconstrictors in enhancing positive outcomes of PEA. However, there is already sufficient evidence regarding hemodynamics during clinical resuscitation. For instance, resuscitation guidelines have called for consistent doses of epinephrine during CPR whenever the treatable causes of PEA are absent. However, rescuers or practitioners have not found a reliable way of comprehending either the patient's inotropic status or the level of the vascular resistance. Vasoconstrictors enhance the flow of blood and increase survival rates among various animal models. Nonetheless, they have not contributed to improved survival rates in clinical trials, particularly when they are administered after the patients are no longer viable. In addition, vasoconstrictors have not improved the rate of survival for PEA patients because of irregular compressions of the chest and the concomitant variable amount of blood flow derived from them (Barnes, 2013).

Gueugniaud et al. (2008) conducted a research and compared HDE and SDE for cardiac arrest and PEA patients. In particular, the PEA patients' survival rate was significantly improved with the use of HDE. In contrast, the authors did not find any beneficial impact on the neurologic outcome or long-term survival. Interestingly, the mortality rate of patients who utilized HDE was higher than that of patients who took SDE. This finding differs from that of Callaham, Madsen, Barton, Saunders, and Pointer (1992) who did not find any adverse effect caused by the administration of HDE.

Moreover, Gueugniaud et al. (2008) established that HDE significantly improved the rate of effective resuscitation only when physicians utilized standard cardiopulmonary resuscitation. On the other hand, survival rates were higher among patients who received decompressed

cardiopulmonary resuscitation and SDE group. An examination of the initial cardiac rhythm pointed out that the return rate for admission to the hospital and spontaneous circulation was increased when the patients took HDE. However, such treatment works only for patients with prolonged cardiac arrests. On the contrary, SDE significantly improved the rate of effective survival in the event of the coarse VF, which represented the shortest cardiac arrests. The authors concluded that for patients who experienced VT the administration of consistent high doses, epinephrine increased the probability of initial survival after undergoing hospital cardiac arrest and the ROSC.

Similarly, Dieckmann and Vardis (2005) carried out an evaluation that underlined the usefulness of both SDE and HDE for PEA patients. In this evaluation, the authors noted that HDE appeared not to boost neurological effect and the return of organized electrical rhythm, including the ROSC. Then, they compared these findings with the results of SDE in the treatment of pediatric cardiopulmonary arrest. According to Dieckmann and Vardis (2005), an appraisal of effective resuscitation, the neurological result, hospital admission, and spontaneous circulation do not influence the choice between SDE and HDE.

Callaham et al. (2012) reinforced these findings in their assessment to determine the efficacy of HDE as compared to SDE prescribed to cardiac arrest patients in pre-hospital care. Similar to Dieckmann and Vardis' findings, these authors also established that there was no significant indication that HDE was more effective in the treatment of cardiac arrest patients when compared to SDE. However, there were no complications associated with HDE, including a longer stay in the hospital.

Interventions in the Patient with Returned of Spontaneous Circulation from Ventricular Fibrillation

Due to the implication of early defibrillation, pre-hospital care is significant for cardiac arrest patients owing to the VF that, in most instances, occur outside the hospital. Interventions that influence the result and survival resuscitation comprise witnesses of an arrest, application of an automated external defibrillator, early recognition, and activation of emergency services. In addition, resuscitation of VF includes early access to trained EMS practitioners possessing the potential of performing defibrillation, CPR, and ACLS (Brugada P., Brugada J, Mont, Smeets, & Andries, 2007).

Conventionally, CPR consists of chest compressions and artificial respirations. There has been evidence, which indicates that high chest compressions are significant actions, which provide some cardiac perfusion in the course of CPR and that artificial respirations have less significance. Interruption of chest compression in performing artificial respiration by a single resuscitator leads to a loss of cardiac perfusion pressure. Furthermore, after restarting compressions, it could take quite some time before the restoration of the previously obtained perfusion pressure (Brugada et al., 2007).

Other concerns relating to VF treatment include the recommendation for consistent artificial ventilation. There is a potential for rescuers to be prone to hyperventilate the victim, which could result in intrathoracic pressure and subsequently lead to decreased coronary survival and perfusion. There is also a likelihood for bystanders to undertake CPR, which only involves chest compression with no artificial respirations. The existing courses of action by the American Heart Association (2010) offer recommendations for the immediate treatment with more than 30 chest compressions before performing any artificial ventilation. Lay rescuers who have not gone adequate training are only required to offer chest compression by emphasizing on the principle “push hard and fast” (American Heart Association, 2010). This process ought to continue until

the arrival of health care providers with automated external defibrillator who are then required to take over (Rassi A. Jr, Rassi A., & Rassi, G., 2007).

On the other hand, trained rescuers are required to provide two artificial breaths and 30 compressions. They also should perform cycles of 30 chest compressions and two ventilation until advanced airways are placed. Thereafter, chest compressions should be consistently performed alongside the provision of one breath for every 6 to 8 seconds. The guidelines of the American Heart Association (2010) reflect on the developments in the ongoing research areas. They include the "cardiocerebral resuscitation" approach, which emphasizes minimized interruptions to consistent chest compressions for those who had been victims of cardiac arrest (AHA, 2010).

Application of an automated external defibrillator and the defibrillation by the trained practitioners in the field has transformed the pre-hospital VF management because it significantly reduced the time for defibrillation. It is achieved through having the units prepositioned in places where there is a likelihood of cardiac arrest occurring. They include but not limited to jails, casinos, airports, malls, industrial parks, and stadiums. This approach is important in eliminating the necessity of training and increasing the number of trained practitioners (Rassi et al., 2007).

Interventions in the Patient with Returned of Spontaneous Circulation from Pulseless Ventricular Tachycardia

Several studies have affirmed that sustained VT is the cause of the hemodynamic collapse. Apparently, such patients need an urgent conversion to sinus rhythm. The approach for conversion is dependent upon whether the patient is hemodynamically unstable or stable. Unstable patients have symptoms and signs of inadequate delivery of oxygen into important

organs because of tachycardia. These manifestations include but not limited to hypotension, dyspnea, chest pain, and altered consciousness level. In the event of a work-up, such a context ought to be differentiated from clinical manifestations of an underlying medical disorder, which could be causing secondary tachycardia (Rassi et al., 2007).

McNally et al. (2010) explained that unstable patients who have no monomorphic VT ought to be immediately treated by use of synchronized direct current cardioversion mostly at a starting energy dose of 100 J. Immediate defibrillation has also been found effective in treating unstable polymorphic VT. Nonetheless, there may be some difficulty for the defibrillator to recognize the divergent QRS complexes, thus hindering synchronization of shocks. On the other hand, stable patients have sufficient perfusion of the vital end organ and, therefore, could not experience symptoms or signs of hemodynamic compromise. The kind of treatment that may effectively treat this condition is dependent upon whether VT is polymorphic and monomorphic and whether left ventricular function is impaired or normal.

In stable patients with normal left ventricular functions and monomorphic VT, restoration of sinus rhythm is typically realized with intravenous sotalol or procainamide. When left ventricular function is impaired, lidocaine or amiodarone are preferred over procainamide for pharmacological conversion since the latter can lead to the heart failure. Nonetheless, a study by McNally et al. (2010) points out that amiodarone ought to be the first-line antiarrhythmic for stable VT since its impact on myocardial refractoriness and conduction are gradual in onset. In the event that medical therapy proves to be unsuccessful, synchronized cardioversion (50-200 J) prior to sedation is effective. For stable patients, polymorphic VT typically terminates on its own. However, there is a tendency for it to recur. When the sinus rhythm returns, electrocardiogram ought to be analyzed to determine whether the QT interval has been

prolonged or normal. Polymorphic VT in patients with a regular GT interval is treated in a similar manner as the monomorphic VT.

Summary and Conclusion

This literature review has noted that cardiac arrest has remained a risky disorder despite advancements in cardiac life support. Patients who experienced A-systole, VF, VT, or PEA are in greater danger. Consequently, physicians used epinephrine as an adrenergic agent to treat such patients.

A widespread treatment of A-systole and PeA involves CPR in a combination with intravenous vasopressor such as epinephrine. Studies analyzed have demonstrated that the administration of epinephrine in such cases is quite effective. There is an opinion that the use of epinephrine in providing a long-term survival has a favorable although not significant influence on of A-systole and PEA patients. While treating cardiac arrest and other cardiac dysrhythmias disorders such as A-systole and PEA, such treatment leads to the elimination of cardiac output. It helps patients by increasing resistance through α_1 receptor-dependent vasoconstriction and increasing cardiac output through β_1 receptors. Therefore, this medicine is used to decrease the immune response to the administered allergen.

However, the findings from studies state that the consistent use of HDE in treating A-systole or PEA has no significant benefits, but rather adverse effects. However, SDE has a significant impact on the rate of successful resuscitation and an increased rate of spontaneous circulation. Therefore, it should still be recommended in the management of cardiac arrest, PEA, A-systole, and VF. Nevertheless, there are a number of benefits associated with using HDE, including better rates of resuscitation and increased rates of spontaneous circulation.

In case of VF, interventions that influence the result and survival resuscitation comprise witnesses of an arrest, application of automated external defibrillator, early recognition, and activation of emergency services. In addition, resuscitation of VF includes early access to trained EMS practitioners possessing the potential of performing defibrillation, CPR, and ACLS. Another concern related to VF treatment is continuous artificial ventilation. Other studies reviewed have also provided recommendations for the immediate treatment with more than 30 chest compressions before performing any artificial ventilation. Common citizens can also rescue patients by performing chest compressions, following the principle “push hard and fast”.

Common intervention approaches for patients with VT include the use of synchronized direct current cardioversion mostly at a starting energy dose of 100 J. Furthermore, immediate defibrillation is effective in treating unstable polymorphic VT. Nonetheless, the kind of treatment that may effectively treat this condition depends on whether VT is polymorphic or monomorphic and whether left ventricular function is impaired or normal. When left ventricular function is impaired, lidocaine or amiodarone are preferred over procainamide for pharmacological conversion since the latter can result in heart failure. Nonetheless, other studies have noted that amiodarone ought to be the first-line antiarrhythmic for stable VT since its impact on myocardial refractoriness and conduction are gradual in onset. In the event that medical therapy proves to be unsuccessful, synchronized cardioversion (50-200 J) prior to sedation is effective.

Potential Research Questions

It is critical to note that the arguments presented by the authors above are applicable to the use of artificial respirations for initial resuscitations for VF circulatory arrest. There is a necessity for future studies to confirm the significance of the artificial respirations for drowning as well as respiratory, traumatic, and other causes of cardiac arrest. From these studies, it is clear

that the optimal treatment for such conditions with grossly divergent etiologies will be different and that it is not possible for one approach to fit all the issues addressed.

Epinephrine commonly identified as adrenaline has been widely employed in pre-hospital cardiopulmonary resuscitation for patients with cardiac arrest and other disorders, including Asystole and PEA. According to the literature reviewed, there are clinicians who are increasingly using HDE in an attempt to improve the rate of survival. However, the evidence regarding its routine has failed to show the positive impact on survival rates. The use and efficacy of this medication, particularly in out of hospital cases have continued to remain controversial for a long time. Therefore, there is a need for more studies on the exact doses needed to address these conditions. Furthermore, there is a need for more research to identify the impact of improper doses on an individual's health.

Therefore, studies need to come up with an effective therapy that can address the problem irrespective of the circumstances or features.

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