Cardiac Arrest Arrhythmias

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Abstract

Cardiac arrest (CA) is a clinical condition defined by the absence of an effective circulatory blood flow due to the inability of the heart to provide consistent hemodynamic support.

Two main characteristics challenge the treatment of CA: its unpredictable onset and the very short time in which to intervene in order to avoid irreversible and dramatic organ damage.

The cardiology community has attempted to address the difficulties related to the management of CA through the development of standardized protocols aimed at treating CA. The current guidelines place strong consideration on the initial rhythm and the restoration of spontaneous circulation with resuscitation. What is not sufficiently emphasized in the current approach to CA is the dynamic transition of different cardiac rhythms during the acute event. In this chapter, the temporal sequence of different cardiac arrhythmias during CA and their prognostic significance are described. The proarrhythmic effects of pharmacological and electrical treatment utilized during the resuscitation are also discussed. In recent years, therapeutic hypothermia has been introduced for the treatment of survivors of CA in an effort to reduce neurological damage. Electrocardiographic abnormalities associated with therapeutic hypothermia are briefly described.

Keywords

Cardiac arrest • Sudden cardiac death • Cardiac rhythm transition • Ventricular arrhythmias

Cardiac arrest (CA) is a clinical condition defined by the absence of an effective circulatory blood flow due to the inability of the heart to provide consistent hemodynamic support. CA represents the most severe complication of any cardiac disease and can also constitute the final evolution of a wide spectrum of noncardiac illnesses.

The main challenges in the management of cardiac arrest stem from its unpredictable onset in addition to the very short time in which to intervene in order to avoid irreversible organ damage. A lack of understanding of the mechanisms of cardiac arrhythmias makes this clinical entity extremely difficult to treat.

Cardiopulmonary Resuscitation

The cardiology community has attempted to address the difficulties surrounding the management of CA by developing standardized treatment protocols. This standardized

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approach, termed cardiopulmonary resuscitation (CPR) [1], provides guidelines for the treatment of cardiac rhythm abnormalities in an effort to preserve oxygen supply to vital organs.

Because of the sudden nature of the event, often occurring in a nonmedical environment and involving family members or persons with no medical training, a simplified approach has been developed, emphasizing the importance of cardiac rhythm analysis during the first 2 min of the CA, followed by treatment as dictated by standardized algorithms. This oversimplified approach has resulted in two misconceptions: one which identifies success as only the restoration of spontaneous circulation and one which identifies CA according to only the initial arrhythmia without consideration for the possibility of degeneration into a different arrhythmia.

Regarding the first misconception, the success of resuscitation should not simply be defined as the return of spontaneous circulation (ROSC), but should be measured by a patient's survival to discharge from hospital. Descriptive analysis shows that sustained ROSC occurs in approximately half of the cases of CA. In the remaining cases, ROSC is merely a temporary condition. The significance of this distinction is evident in the disparity between published rates of on-scene ROSC versus rates of survival of CA upon hospital discharge. For instance, a meta-analysis of published studies reporting outcomes after out-of-hospital CA indicates that the median rate of survival to hospital discharge is only 6.4 % [2]. However, the reported incidence of on-scene ROSC of out-of-hospital CA, while variable, is significantly higher, ranging from 35 to 61 % [3, 4]. There are several reasons accounting for this discrepancy, including the neurologic response to circulatory arrest, complications arising during hospitalization, and repetitive spontaneous arrests prior to arrival to hospital. However, the most critical of all variables is the initial presenting cardiac rhythm during which appropriate therapy can have a very relevant impact on the survival to hospital discharge.

The second misconception as a result of the oversimplification of management algorithms suggests that the CA is due to, and should be solely treated as per the initial presenting rhythm. However, if the phases preceding the sudden onset of CA have been extensively studied without conclusive insight, much less attention has been paid to the rhythm transition after CA. Moreover, few data are available on the effect of medication and electrical therapy in the dynamic sequence of cardiac rhythms during CA.

The phases following CA can present many more complex challenges in terms of rhythm diagnosis and treatment than the onset. External thoracic compressions as well as the precordial blow can produce artifactual rhythms that may simulate the return of cardiac activity, thereby triggering an incorrect cessation of treatment by nonmedical persons. Electrocardiographic modifications can be more difficult to diagnose after the resuscitation maneuver, and some of the drug administration protocols, for example, high doses of epinephrine, can generate a return of a tachyarrhythmia. It is therefore useful to separate post-CA arrhythmias into three different categories:

1. Arrhythmias causing cardiac arrest
2. Arrhythmias generated by the administered treatment for cardiac arrest
3. Artifactual postresuscitation arrhythmias – induced by specific CPR maneuvers (precordial blow, thoracic compressions, and repeated defibrillation)

In this manner, we are able to differentiate between two important categories of rhythms: arrhythmias that begin prior to CA and arrhythmias that are generated during CPR maneuvers which may or may not promote successful return of circulation.

Pathophysiology of Cardiac Arrest

The pathophysiology of CA is believed to require the interaction between a transient event and an underlying substrate. These factors may culminate in electric instability and lethal ventricular arrhythmias followed by hemodynamic collapse. The challenge remains to predict when such interactions prove harmful. Physical activity, psychosocial stress, and air pollution have been proposed as risk factors in timing of onset of CA [5]. The presence of prior cardiac disease plays a critical role in the dynamic process that characterizes CA. Arrhythmias in patients with structurally abnormal hearts with depleted functional reserves have less chance of successful resuscitation as compared to patients with no prior cardiac history [5]. Risk factors for CA include advanced age, male sex, cigarette smoking, hypertension, diabetes mellitus, hypercholesterolemia, obesity, and a family history of coronary artery disease. Not surprisingly, these risk factors are also predictors of coronary heart disease–related death and all-cause mortality. Coronary heart disease is the most common substrate underlying CA in the Western world, being responsible for 75 % of CA [5]. Cardiomyopathies (dilated, hypertrophic, and right ventricular arrhythmogenic dysplasia), and primary electrical disorders due to underlying channelopathies (Brugada syndrome, Long QT syndrome, short QT syndrome, and catecholaminergic polymorphic ventricular tachycardia) account for most of the remainder. Channelopathies are inherited arrhythmia disorders characterized by ion channel defects in structurally normal hearts capable of causing electrical disturbances leading to ventricular fibrillation. Finally, CA may also occur in the context of drowning, hanging, electrocution, and severe trauma.

Existing energy reserves and the underlying functional integrity of the heart muscle can ensure positive results from CPR. In contrast, CAs that present in patients with structurally or electrically abnormal hearts are often associated with

poor CPR outcomes [5]. The pattern of arrhythmias initiating a CA is also likely to be influenced by the underlying heart disease.

Cardiac Rhythms Associated with Cardiac Arrest

Four main electrocardiographic rhythms are associated with CA: ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), asystole, and pulseless electrical activity (PEA). VF can be defined as a spatiotemporal electric turbulence compromising the heart's ability to pump blood efficiently and is the mechanism underlying most sudden CA episodes. By contrast, VT might not initially present with hemodynamic impairment. However, if left untreated, VT may progress to pulseless CA with subsequent degeneration into VF. Because clinical "pulseless VT" is an organized rhythm that may be associated with some degree of effective cardiac output, it is thought that outcomes from VT are more favorable than those from VF. Asystole is characterized by the absence of electrical cardiac activity. In contrast, PEA is, by definition, characterized by the presence of electrical cardiac activity, but without effective systemic perfusion. There may, however, be some degree of systemic perfusion despite the lack of palpable pulses. Not surprisingly, PEA has been associated with better outcomes than asystole during in-hospital and out-of-hospital cardiac arrest [5]. VF or VT has been reported to be the first presenting rhythm in 75–84 % of cases of CA, while asystole and PEA account for the remainder [5]. All of these arrhythmias necessitate rapid initiation of CPR maneuvers to ensure patient recovery. The return and persistence of electrical cardiac activity, associated with an effective systemic circulation (i.e., ROSC), is the goal of resuscitation maneuvers.

CA rhythms can be separated into two categories with respect to management: arrhythmias that respond to electrical defibrillation or cardioversion (also called shockable rhythms), namely, VF and VT, and arrhythmias that do not respond to defibrillation (nonshockable rhythms), namely, asystole and PEA. Both groups are managed by advanced cardiac life support (ACLS) maneuvers. Evidently, patients with CA presenting with nonshockable rhythms (asystole or PEA) typically have poorer survival rates than those presenting with shockable rhythms (VF or VT) [6, 7]. For this reason, VF and VT have traditionally been considered "good" cardiac arrest rhythms (Table 21.1).

Table 21.1 Association between first presenting rhythm, rhythm transition, and prognosis following resuscitation of cardiac arrest

First detected rhythm during cardiac arrest	Prognosis[a]
Shockable rhythm (VF or VT)	+++++
Nonshockable rhythm (asystole or PEA)	++
Rhythm transition during ongoing cardiac arrest	
Recurrent shockable rhythm (VT/VF)	++
Transition from shockable to nonshockable	+
Transition from nonshockable to shockable	+

[a]The number of + symbols, ranging from 1 to 5, indicates the relative prognosis ranging from poorest to best. Transition from a nonshockable to a shockable rhythm is not associated with an improvement in prognosis (see text for explanation)

Cardiac Rhythm Transitions During Cardiac Arrest

The reported incidence of cardiac re-arrest (defined by the occurrence of any pulseless cardiac rhythm after a ROSC) ranges from 61 to 79 % in different studies [8–10], and the transition from a shockable rhythm to a nonshockable rhythm commonly occurs during the CA (Table 21.1). Skogvoll et al. [11], analyzed defibrillator event recordings of 304 cases of CA among adult populations in Norway, Sweden, and the UK. They reported a median of five (range 1–39) rhythm transition states among the four main rhythms of CA during an acute episode, with an increased number of rhythm transitions when the initial rhythm was VF or VT rather than asystole or PEA. Importantly, in all 304 defibrillator recordings, 35 % of patients regained a ROSC during CPR and only 21 % went on to achieve sustained ROSC. In patients initially presenting with VF, the reappearance of VF and/or the appearance of asystole are major risk factors for poorer outcome. PEA is a particularly negative risk factor for resuscitations of longer duration. Knowledge of the transition and timing of different rhythms during CA can provide important information in directing management decisions during CA (Fig. 21.1).

Comparable data was demonstrated by Meaney et al. [12], who analyzed 51,919 cases of patients having undergone in-hospital CA occurring in 411 different centers. They found that it was both the presence of a shockable rhythm as well as the temporal sequence in the succeeding shockable and nonshockable rhythms that determined the eventual outcome. Importantly, patients with nonshockable rhythms, such as asystole or PEA, followed by a subsequent shockable rhythm during the resuscitative efforts, had much worse outcomes than those who remained in asystole of PEA. Similarly, but in a cohort of patients with out-of-hospital CA, Hallstrom et al. [13] reported a poorer outcome in patients whose initial rhythm was asystole or PEA that subsequently converted to VF or VT versus patients who remained in asystole or PEA.

A recent study by Sacedo et al. [14], on the incidence of re-arrest in a cohort of out-of-hospital CA, demonstrated that the identification of cardiac re-arrest in the acute setting was a few minutes longer than the time it took a competent observer to make the correct diagnosis from the same data. A clear understanding of the dynamic evolution of CA is of paramount importance in prompting well-timed therapeutic intervention.

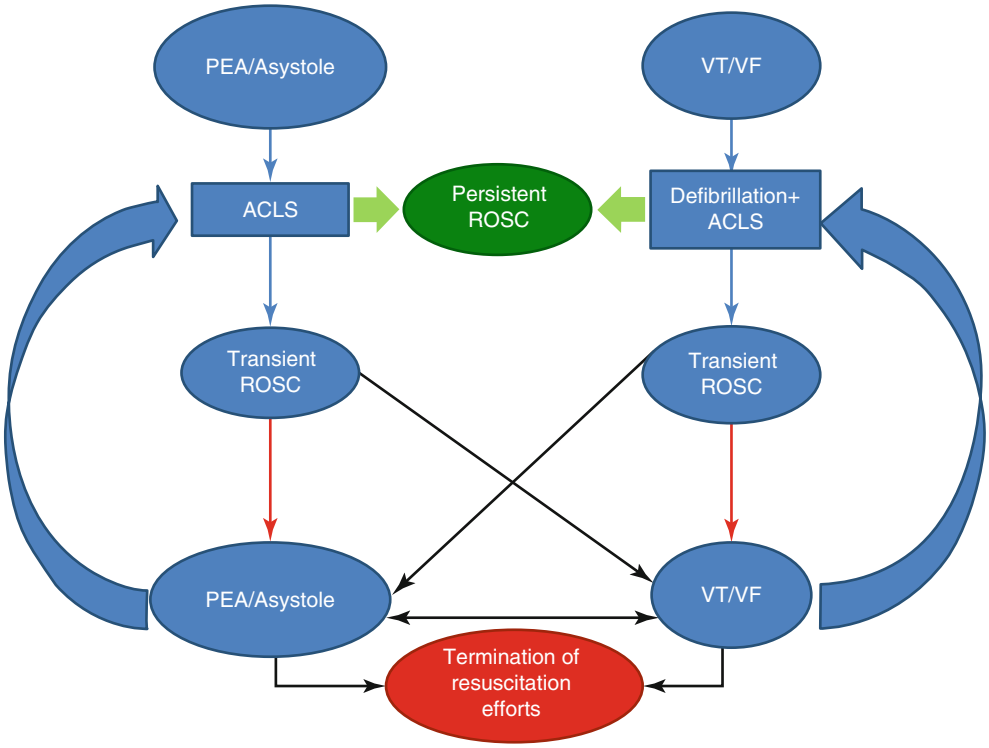

Fig. 21.1 Flow diagram of the potential sequence of arrhythmias and resuscitation efforts during cardiac arrest. The temporal sequence of different cardiac arrhythmias has prognostic significance. *Black arrows* indicate a worse prognosis than *red lines*; *red arrows* indicate a worse prognosis than *blue arrows*. *Abbreviations*: *PEA* pulseless electrical activity, *ACLS* advanced cardiac life support, *ROSC* return of spontaneous circulation

The Impact of Pharmacological Therapy on Arrhythmias During Cardiac Arrest

It has been hypothesized that in the dynamic transition between different cardiac arrhythmias during CA, the administered pharmacological therapy could have detrimental effects. Clinical observations suggest that adrenaline, a cornerstone therapy in the current treatment algorithms, can make a cardiac rhythm during CA more dynamic and unstable. Recently, Neset et al. [15] published an analysis of a trial conducted on 223 cases of out-of-hospital CA. Patients were randomized to ACLS with or without the administration of intravenous drugs. Patients receiving intravenous adrenaline presented with more frequent rhythm transitions than patients not receiving intravenous adrenaline. A trend toward shock-resistant VF or VT in the adrenaline group was detected. Relapse from ROSC to VF or VT in the no-adrenaline group tended to occur during the first 20 min, whereas the adrenaline group experienced the relapse after more than 20 min. The transition from nonshockable rhythm to VF or VT occurred more frequently in the adrenaline group than in the no-adrenaline group. However, as discussed earlier, this rhythm transition does not necessarily correspond to an improvement in prognosis.

Evidence concerning the effect of anti-arrhythmic drugs is incomplete. It has been suggested that amiodarone may play a role in the treatment of CA to prevent VF or VT recurrence, but this effect has not been thoroughly studied and its contribution to rhythm transition has not yet been assessed. Beta-blockers may have a benefit during CA from VT due to their effect on decreasing oxygen demand. Improvements in long-term prognosis of patients treated with beta-blockers has been shown [16]. However, concerns have arisen regarding beta-blocker effects on reduction of myocardial contractility which causes hypotension in patients with CA.

Cardiac Rhythm Preceding Sudden Death in Patients with Implantable Cardioverter Defibrillators and Impact of Electrical Therapy

Over the last decade, important new insights into the mechanisms of cardiac rhythm transition during CA have emerged as a result of follow-up of patients with implantable cardioverter defibrillators (ICDs). Approximately 25 % of patients with ICDs present with sudden cardiac death despite the presence of an ICD [17–20]. Incessant VF or VT as well as shock failure account for 26 and 18 %, respectively, of sudden cardiac death mechanisms in patients with functioning ICDs.

Mitchell et al. [21] interrogated more than 317 patients ICDs and described an electromechanical dissociation after repeated defibrillator discharge as the mechanism responsible for sudden cardiac death in up to 29 %. In these patients, two or more shocks were required to convert the VF or VT, but this was followed by cardiac mechanical deterioration leading to death. The short temporal sequence between

shock and mechanical dysfunction excludes necrosis as the instigating mechanism, but supports a potential deleterious effect of the shock on the molecular and electrical functioning of the heart, which could last from minutes to hours. These functional changes have only thus far been demonstrated in animal models [22, 23]; therefore, the clinical impact is still to be determined. Recently, Tsuji et al. [24, 25] have demonstrated in an animal model of electrical storm that repeated defibrillation can affect the activity of membrane protein handling calcium cell homeostasis (mainly calcium/calmodulin-protein kinase II). The upregulation of this protein can lead to intracellular calcium overload which may be responsible for mechanical dysfunction and arrhythmia recurrence.

Finally, even the electrical therapy used for cardioversion at the time of CA can be proarrhythmic [26, 27]. In some circumstances, when the energy delivered by the shock is not adequate and the myocardium is more vulnerable, delivery of a shock can result in re-initiation of VF instead of halting its propagation.

Impact of Cooling on Arrhythmias After Cardiac Arrest

In recent years, therapeutic hypothermia has been introduced as a treatment to reduce neurological damage in survivors of CA due to an initial shockable rhythm [28]. The rationale underlying this therapy is to reduce cerebral metabolic demands and minimize cellular brain damage produced by reperfusion. Due to the significantly better neurological outcomes shown in trials [29, 30], therapeutic hypothermia has largely become adopted for patients resuscitated from CA. The patient is sedated, paralyzed, and rapidly cooled to a temperature of 32–34 °C for 12–24 h. Following the adoption of hypothermia therapy, a wide spectrum of electrocardiographic abnormalities have been detected among patients monitored in the intensive care unit. An alteration commonly described is a prolongation of the QTc interval from 47 to 80.3 ms longer than the baseline QTc interval [31–33]. The incidence of ventricular arrhythmias is very rare, and if present, is associated with more severe hypothermia (below 32 °C) [33]. Cases of torsades de pointes [34], idioventricular rhythms [35], and VF [36] after QT prolongation have been described. The pharmacological management of cardiac effects during therapeutic hypothermia is currently under debate. The effect of hypothermia on defibrillation threshold in patients with ICDs is also unclear [37].

Conclusion

In the management of CA, great attention has been paid to the initial presenting rhythm and to ROSC after resuscitation. This is a consequence of the efforts of the cardiology community to attempt to address the challenge posed by CA. However, what is not sufficiently emphasized in the current approach to CA is the dynamic evolution of different cardiac rhythms during the event. The temporal sequence of different cardiac arrhythmias has prognostic significance. Moreover, the pharmacologic and electrical management during the resuscitation process may themselves result in secondary adverse cardiac arrhythmias. An appropriate knowledge of this dynamic evolution of cardiac rhythm constitutes a challenge to the current management of CA.

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