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Simultaneous Transthoracic Defibrillation With Two Defibrillators for Refractory Ventricular Fibrillation
A Literature Review

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Abstract
Ventricular fibrillation and pulseless ventricular tachycardia are the presenting rhythm in half of sudden cardiac deaths and have a higher successful resuscitation rate than asystole and pulseless electrical activity. The goal of defibrillation is to stun the myocardium and allow normal cardiac pacemakers to take over. Current American Heart Association guidelines for the treatment of ventricular fibrillation may not recommend enough energy to terminate prolonged ventricular fibrillation. Several reports have looked at simultaneous cardioversion with two defibrillators for atrial fibrillation refractory to treatment guidelines and have a high success rate. Because atrial and ventricular fibrillation have many common features, it is reasonable to extrapolate that simultaneous defibrillation for ventricular fibrillation refractory to current guidelines may be beneficial in certain patient populations. The literature contains a few cases of simultaneous defibrillation with two defibrillators for refractory ventricular fibrillation; the technique should be considered for patients not responding to standard treatment. **Key words:** defibrillation, refractory ventricular fibrillation

IN 2010, THE LEADING CAUSE OF DEATH in the United States was heart disease (Murphy, Xu, & Koanek, 2013). Sudden cardiac death occurs in an estimated 180,000 to 450,000 people annually and is believed to account for up to 50% of deaths from cardiovascular disease (Kong et al., 2011; Stecker et al., 2014). Ventricular fibrillation (VF) and ventricular tachycardia are the presenting rhythm in approximately 50% of all sudden cardiac death patients (vs. pulseless electrical activity and asystole) and have a much higher successful resuscitation rate (~40%) versus pulseless electrical activity (less than 6%; Teodorescu et al., 2010). Given that VF is a potentially treatable arrhythmia, it is important for health care providers to have current knowledge about the treatment modalities of proven or possible benefit.

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DEFINITION OF VF

Fibrillation is defined as "turbulent cardiac electrical activity whereby propagation of electrical waves through the heart is severely disrupted, with consequent inability of the myocardium to contract" (Vaquero, Calvo, & Jalife, 2008, p. 872). There are three phases of VF. The first phase is the electrical phase,

which occurs during the first 5 min. During this time, the myocardium still has energy reserves and significant cellular damage has not yet occurred (Ewy, 2010). The second phase is the circulatory phase and lasts between 5 and 15 min. In this period, there are decreased myocardial energy stores due to continued uncoordinated contractions, and toxic metabolites accumulate (Ewy, 2010). The third phase is the metabolic phase, when ischemic and reperfusion injuries result in endothelial damage and the release of inflammatory mediators into the circulation results in systemic injury (Weisfeldt & Becker, 2002).

Ventricular fibrillation is the result of disordered electrical excitation of the ventricles. The mechanism behind VF is explained by the theory of wave breaks and rotors. Loss of coordinated ventricular contraction is caused by repetitive reentrant depolarization and/or rapid focal discharges that begin in larger waves that take indirect routes before degenerating into smaller reentry wavelets with changing pathways (Ewy, 2010). As the duration of VF continues, the number of wavelets increases, whereas their size decreases (Gradaus et al., 2002). Waves are formed by spinning rotors, which are the organizational source of fibrillation. Rotors are initiated in two ways: when a stimulus is applied during the refractory phase of the first wave front leading to wave break or by a block occurring in the electrical pathway caused by changes in electrical excitability and repolarization (Pandit & Jalife, 2013).

Ventricular fibrillation is typically short-lived secondary to lack of myocardial blood flow but can be prolonged with adequate chest compressions administered during cardiopulmonary resuscitation (CPR; Ewy, 2010). During this time, wave breaks decrease in the ischemic area and increase around the ischemic border to maintain fibrillatory activity (Vaquero et al., 2008). It is thought that prolonged VF may be maintained by different means than short duration VF, and the mechanism behind defibrillation and its efficacy may differ among the phases of VF because of different wavelet patterns and varying defibrillation thresholds (Jin et al., 2013). Refibrillation is also common after successful defibrillation of long duration VF (Jin et al., 2013).

DEFIBRILLATION

The goal of defibrillation is to stun the myocardium, terminate fibrillatory activity, and allow normal cardiac pacemakers to take over and establish an organized rhythm. The thought behind the mechanism of defibrillation is that the myocardial transmembrane electrical potentials are altered, making the cells temporarily unexcitable, which allows the normal electrical conduction with associated contraction to return (Monteleone, Borek, & Althoff, 2012).

Lerman and Deale (1990) postulated two theories behind defibrillation; the critical mass theory and the upper limit of vulnerability theory. The critical mass theory states that defibrillation is achieved by simultaneously depolarizing a large enough quantity of excitable cells, thereby extinguishing wavelets and rotors within the myocardium. The upper limit of vulnerability theory states that defibrillation occurs by delivering a depolarizing stimulus that exceeds the upper threshold of both fully excitable cells and cells that are in their relative refractory period.

There are two types of defibrillators currently available. Monophasic defibrillators deliver unidirectional energy flow from one paddle or pad to the other, whereas in biphasic models, energy flows from one paddle or pad to the other and then reverses to flow in the opposite direction. There is a growing popularity of biphasic defibrillators secondary to general consensus that a lower level of energy is needed for successful defibrillation and that they are generally more effective in cardioversion and defibrillation. Monophasic defibrillators are able to deliver up to 360 joules (J). Biphasic defibrillators are available in both low energy (maximum of 200 J) and high energy (maximum of 360 J) models.

FACTORS AFFECTING DEFIBRILLATION

The factors affecting successful defibrillation are transthoracic impedance and

defibrillation threshold. Transthoracic impedance is the resistance between the defibrillator and the patient's heart and is a measure of opposition to electric current. It is created by both the electrical circuit and the patient's body (Monteleone et al., 2012). Defibrillation is more likely to be successful with lower impedance (Ewy, 2010). Patients with higher impedance will require more voltage for defibrillation to be effective (Finamore & Turris, 2008).

There are many different factors contributing to transthoracic impedance. Nonmodifiable patient-related factors include a history of hypertension, chronic obstructive pulmonary disease/lung hyperinflation/pulmonary air trapping, large body mass/obesity, tissue scarring, edema of the thoracic cage, and plural effusion (Finamore & Turris, 2008; L'Italien, 2013; Monteleone et al., 2012; Reiffel, 2009). Contributing patient factors that providers can modify are excessive hair present on the chest, moisture present on skin, and hyperthermia (Finamore & Turris, 2008; Monteleone et al., 2012).

Defibrillator factors that increase transthoracic impedance include improper pad placement and energy being delivered at full inspiration (Ewy, 2010; Finamore & Turris, 2008). Higher impedance is noted with greater distance between electrodes, and self-adhesive electrodes have higher impedance than paddles due to less force being applied during shock delivery (Ewy, 2010; Jones & Lode, 2007). Impedance is noted to decrease with an increase in the number of shocks delivered (Bjerregaard, El-Shafei, Janosik, Schiller, & Quattromani, 1999; Ewy, 2010), and an increase in electrode diameter may result in decreased impedance (Ewy, 2010).

Defibrillation threshold is the level at which shock strength is delivered to defibrillate the myocardium. Delivery of energy must achieve sufficient current through the myocardium to exceed the threshold necessary in order for defibrillation to be successful, irrespective of the electrophysiologic state (Deakin & Ambler, 2006). Factors that increase the defibrillation threshold include cardiac ischemia, electrolyte imbalance, chronic lung disease, and obesity (Cohen et al., 1993; Hoch et al., 1994; Walcott, Killingsworth, Smith, & Ideker, 2002). Antiarrhythmic drugs (specifically Class I and Class III antiarrhythmics) can either increase or decrease the defibrillation threshold (Reiffel, 2009). The position and polarity of electrodes can also have an effect on the defibrillation threshold, as well as the presence of implanted internal defibrillator patches, especially if the patches are placed parallel to the transthoracic electrode axis (Hoch et al., 1994).

CURRENT TREATMENT RECOMMENDATIONS

Current American Heart Association (AHA) recommendations for the treatment of a witnessed cardiac arrest are to defibrillate a shockable rhythm as soon as the defibrillator is ready. In the event of an unwitnessed arrest, some studies advocate for a cycle of CPR prior to defibrillation; however, there is insufficient evidence to determine whether this has any proven benefit to patient outcomes. It is advised in all cases that CPR be performed while the defibrillator is being prepared. Recommendations are to use an energy dose of 360 J for all attempts when using a monophasic defibrillator and an energy dose of 120 to 360 J (following the manufacturer's guidelines) with subsequent attempts being of equal or greater energy when using a biphasic defibrillator (Link et al., 2010).

HIGH-ENERGY DEFIBRILLATION

Historically, the use of high-energy defibrillation has been necessary to overcome refractory ventricular and atrial fibrillation (AF). The underlying premise is that only a fraction of the current delivered actually reaches the heart, and the rest is shunted around the heart to the muscles of the chest wall and lungs (Finamore & Turris, 2008; Monteleone et al., 2012). Lerman and Deale (1990) showed that only approximately 4% of transthoracic current delivered reaches cardiac tissue. The longer the duration of VF, the more the electrophysiological state of the myocardium

deteriorates, leading to a less organized rhythm, which is more difficult to abolish, and requires higher-energy levels for successful defibrillation (Deakin & Ambler, 2006; L'Italien, 2013). Higher energy levels are also needed for subsequent shocks after insufficient doses have been delivered (Jones & Lode, 2007).

Guidelines for defibrillation energy doses come from testing done by electrically induced, short duration VF on nonischemic canine and porcine hearts. Recommendations based on this testing may not be appropriate for treating VF caused by ischemic myocardium (Walcott et al., 2002), because it is possible that the two types of fibrillation are maintained by different mechanisms. Walcott et al. (2002) found that VF secondary to ischemia required much higher energy doses to defibrillate than electrically induced VF in two different animal species. Higher energy was needed to defibrillate both ischemic and reperfusion arrhythmias. They hypothesized that spontaneous VF is harder to defibrillate than electrically induced VF because the shock needs to stop the original initiator of the fibrillatory activity and most or all VF wave fronts as well as not restart VF (Walcott et al., 2002).

A few studies have looked at using high-dose energy for termination of VF, the most recent being the BIPHASIC trial in 2007. The results showed that although half of the patients responded to a single shock at 150 J, the other half (who presented with persistent or recurrent VF) required additional shocks. Patients who received escalating energy doses were more likely, than those who received fixed energy shocks at 150 J, to terminate VF and convert to an organized rhythm (Stiell et al., 2007).

Walcott et al. (2002) also found two categories of VF, one that was successfully defibrillated with energy levels from 150 to 200 J and another that required 400 J or was unsuccessful. They hypothesized that VF that was successfully terminated with low levels of energy were initiated by reentrant mechanisms and similar to electrically induced VF commonly used in laboratory defibrillator testing. Higher energy doses are required for VF that is initiated by focal mechanisms and VF that is secondary to ischemic myocardium (Walcott et al., 2002).

A major concern with using high-dose energy for defibrillation is myocardial function after resuscitation. Several studies have measured cardiac function after defibrillation with high-dose energy. Walcott, Melnick, Killingsworth, and Ideker (2010) showed in animals that there was no difference among survivors with regard to blood pressure, echocardiographic measurements, and troponin levels in animals defibrillated with 150 J and animals defibrillated with 360 J (biphasic model used).

A second animal study showed that the dose to convert VF in 50% of study population was 1.5 J/kg, myocardial damage occurred in 50% of population at 30 J/kg, and the lethal dose for 50% of the population being 470 J/kg (Jones & Lode, 2007). Translating that data to a 70-kg (154 lb) individual, around 150 J would be needed to defibrillate 50% of the population. Myocardial damage would occur in 50% of the population with delivery of 2,100 J, and a lethal dose of energy for 50% of the population would be almost 33,000 J. These results suggest that the threshold for myocardial damage from electrical current is well above any dose that would be administered in the clinical setting.

To overcome transthoracic impedance and defibrillation threshold in patients refractory to standard treatment, some providers have begun using two defibrillators attached to the patient and simultaneously discharging them, effectively doubling the energy delivered and increasing the area of the myocardium that is within the current's path.

SIMULTANEOUS CARDIOVERSION WITH TWO DEFIBRILLATORS

Very few studies have looked at the use of two defibrillators for treatment of arrhythmias refractory to standard treatment; however, more data are available to support simultaneous use of two defibrillators for cardioversion of refractory AF. Reiffel (2009) used the

technique for morbidly obese patients in his study population. Chang, Lent, and Grinberg (2008) reported a case in which the patient failed cardioversion at 100, 200, and 360 J. Simultaneous cardioversion was then employed. The technique initially failed at 600 J and then succeeded on the second attempt, with two defibrillators administering a combined total of 720 J. The patient exhibited an elevated creatinine kinase; however, there were three subsequent negative troponin levels (less than 0.03) and no other complications were noted secondary to cardioversion. In a report by Rodriguez et al. (2005), patients with AF refractory to two to three monophasic shocks of up to 360 J underwent double sequential shocks with 720 J. Cardioversion was achieved in 90% of the patients, with the largest predictor of unsuccessful cardioversion being patient weight. They reported no elevation in troponin levels.

Alaeddini et al. (2005) used simultaneous cardioversion with two defibrillators in patients with AF who failed to convert with 360 J delivered over two different pad positions. Two monophasic defibrillators were discharged simultaneously and the technique was repeated once if the patient failed to convert on the first attempt. They reported an 81% success rate. Complications noted included transient sinus bradycardia, sinus pause lasting up to 3 s, and skin burns similar to those reported with traditional cardioversion methods. None of the patients experienced a thromboembolic event, hypotension, or congestive heart failure secondary to the increased energy dose, and they concluded that simultaneous cardioversion using two defibrillators does not cause any more adverse effects than conventional cardioversion (Alaeddini et al., 2005).

Kabukcu, Demircioglu, Yanik, Minareci, and Ersel-Tuzuner (2004) and Marrouche, Bardy, Frielitz, Gunther, and Brachmann (2001) used the technique on patients who had failed two cardioversions at 200 and 360 J. Simultaneous cardioversion was employed using two defibrillators delivering 720 J. They reported an 87% and 74% (respectively) success rate. Kabukcu et al. (2004) reported no elevation in creatinine kinase MB, no resulting VF or ventricular tachycardia, no transient ST elevation, as well as no development of "significant hemodynamic compromise, congestive heart failure, higher AV block, stroke or transient ischemic attack" (p. 932). A few patients had multifocal premature ventricular contractions or transient bradycardia that responded to atropine. Marrouche et al. (2001) reported no significant differences in creatinine kinase, or changes in troponin levels from those patients who were cardioverted using standard energy, as well as no thromboembolic complications and no significant ST segment elevation. They noted postcardioversion bradycardia lasting less than 15 s in several patients and a few complaints of generalized cramps.

Bjerregaard et al. (1999) performed cardioversion using simultaneous energy delivery using two defibrillators in patients who had failed three attempts at 200 and 360 J in two different pad positions, with a 67% success rate. They noted an increase in creatinine kinase levels in patients who received a greater number of shocks, and skin burns, but reported no evidence of increased pain with simultaneous cardioversion and no worsening left ventricular function.

Saliba et al. (1999) used the technique in patients who failed cardioversion at 360 J after two attempts. They reported an 84% conversion rate with the application of 720 J. No hemodynamic compromise, congestive heart failure, stroke, or transient ischemic attacks were reported. Complications included transient bradycardia and transient right bundle branch block with sinus brady-induced torsades. Patient reports of pain or skin burns were similar to that observed with standard cardioversion.

SIMILARITIES BETWEEN AF AND VF

There are several similarities between AF and VF, suggesting that we can extrapolate knowledge from one area to apply to the other. Despite having different structure and ion channel distributions, both atria and ventricles have similar mechanics by which they produce electrical current (Pandit & Jalife,

2013; Vaquero et al., 2008). Both AF and VF are characterized by wave break leading to wavelet formation and rotor initiation and have a left-to-right gradient of defibrillation threshold (Vaquero et al., 2008). Both AF and VF fibrillate as a three-dimensional structure with similar cycle lengths and both successfully respond to shocks on a "probability of success curve" (Ideker, Cooper, & Walcott, 1994, p. 1039).

SIMULTANEOUS DEFIBRILLATION WITH TWO DEFIBRILLATORS

Simultaneous use of two defibrillators for refractory VF has only recently been implemented in the clinical setting. There are three hypotheses about the effectiveness postulated by Erich (2011). One possible mechanism is that the use of four pads creates a larger energy vector than two. Another is that a prolonged shock is delivered by the two defibrillators, as exact simultaneous discharge is difficult to achieve. The third hypothesis behind the mechanism of action is that the increased energy delivered is responsible for its effectiveness.

Hoch et al. (1994) was the first to report using two transthoracic defibrillators for defibrillation of refractory VF. The technique was used on patients who failed defibrillation at 200 J shocks, followed by multiple shocks at 360 J (7–20 attempts per patient), using two different pad configurations. Pads from two defibrillators were applied, both defibrillators were set to 360 J and they were discharged 0.5–4.5 s apart. They reported a 100% success rate. Although the longer duration between shock administrations is consistent with that of the "stacked shocks" technique, those shocks that were delivered closer together are similar to what happens during simultaneous energy delivery using two defibrillators, as exact synchrony of defibrillators is difficult to achieve.

Fender, Tripuraneni, and Henrikson (2013) reported a case of a patient with a left ventricular assist device, who received 10 internal cardiac defibrillator shocks, followed by a failed attempt at biphasic defibrillation with 200 J. Delivery of 400 J via two simultaneous biphasic shocks yielded successful defibrillation. Leacock (2013) reported a patient who was successfully defibrillated with 400 J via two simultaneous biphasic shocks on the first attempt, followed by an episode of ventricular tachycardia that responded to a second attempt with 400 J. This occurred after five unsuccessful attempts with traditional defibrillation methods. The patient made a full recovery with no neurologic impairment. Gerstein, Shah, and Jorgensen (2014) successfully defibrillated a patient via two simultaneous biphasic shocks on the second attempt after 15 unsuccessful attempts at 200 J. The patient subsequently expired several hours later secondary to complications of prolonged resuscitation efforts. New Orleans Emergency Medical Services adopted the technique into its protocol for refractory VF several years ago and reports use of the technique 16 times in 1 year with 25% of patients achieving return of spontaneous circulation and one patient returning home neurologically intact (Erich, 2011). Although the actual data were not reported, Fort Worth MedStar Emergency Medical System stated that approximately 50% of the patients who underwent simultaneous defibrillation had return of spontaneous circulation, with one report of a patient returning home neurologically intact (Erich, 2011).

IMPLEMENTATION OF SIMULTANEOUS DEFIBRILLATION

Based on the current literature, indicators for simultaneous defibrillation with two defibrillators for refractory VF include patient history of cardiac disease such as structural (dilated or hypertrophic cardiomyopathy, atrial septal defect) or valvular heart disease, coronary artery disease, aortic aneurysm, congestive heart failure, and right ventricular dysfunction (Alaeddini et al., 2005; Chang et al., 2008; Hoch et al., 1994; Kabukcu et al., 2004; Marrouche et al., 2001; Saliba et al., 1999). In addition, it should be considered in patients with chronic obstructive pulmonary disease, body mass index greater than 36–40, hypertension, diabetes mellitus, hyperlipidemia,

and treated hyperthyroidism, or patient use of antiarrhythmic drugs (Alaeddini et al., 2005; Chang et al., 2008; Hoch et al., 1994; Kabukcu et al., 2004; Marrouche et al., 2001; Saliba et al., 1999; see Table 1).

Protocols that currently exist for implementation of simultaneous defibrillation are from prehospital services. MedStar Emergency Medical Services initiates simultaneous defibrillation as early as the second defibrillation attempt if the first attempt using 200 J is unsuccessful. New Orleans Emergency Medical Services applies the second defibrillator after four unsuccessful shocks at standard energy levels. Wake County Emergency Medical Services initiates its protocol after five unsuccessful shocks in at least two different pad positions.

Current AHA guidelines state that the four different pad positions (anterolateral, anteroposterior, anterior–left infrascapular, and anterior–right infrascapular) have equivalent shock success rate (defined as "termination of fibrillation for at least 5 s following the shock" [Link et al., 2010, p. S708]) when treating both atrial and ventricular arrhythmias but acknowledge that no studies currently exist examining pad position in relation to return of spontaneous circulation (Link et al., 2010). All four pad positions have been used in varying accounts of use of two defibrillators for treatment of either refractory VF or AF with similar success rates. Pads were placed either adjacent to each other in the anterolateral or anteroposterior position or in opposing positions with electrical fields crossed, creating a new vector of energy.

LIMITATIONS AND RECOMMENDATIONS

No prospective or control group studies have been done because of the nature of the problem, and all existing recommendations come from sources with low levels of evidence (case reports and expert opinion). Formal studies should be conducted to better understand the mechanisms behind, and potential complications of, simultaneous transthoracic defibrillation. The AHA should review all the available evidence and consider including the technique in the Advanced Cardiac Life Support algorithm as an alternative in patients who present with persistent VF refractory to conventional treatment.

CONCLUSION

Based on the literature, it is safe and effective to initiate simultaneous transthoracic defibrillation with two defibrillators in patients who

Table 1. Indicators for simultaneous defibrillation with two defibrillators for refractory VF

History	Specific examples
Cardiac disease	Structural heart disease
	Valvular heart disease
	Coronary artery disease
	Aortic aneurysm
	Congestive heart failure
	Right ventricular dysfunction
	Use of antiarrhythmic drugs
Noncardiac diseases	Chronic obstructive pulmonary disease
	Hypertension
	Diabetes mellitus
	Hyperlipidemia
	Treated hyperthyroidism
	Obesity (body mass index greater than 36–40)

have failed traditional defibrillation methods. Current evidence suggests that this technique should be included in guidelines for treatment of VF and appears to be most effective if employed after three to five unsuccessful attempts at defibrillation using traditional protocol. No specific recommendation can be made at this time from available data as to which pad position is the most effective, because multiple positions have proven successful.

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