Direct current cardioversion: Indications, techniques, and recent advances

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Direct current cardioversion/defibrillation is an important part of the intensivist's armamentarium. Emergent application may be lifesaving. Elective cardioversion should be used cautiously, with attention to patient selection and proper techniques. Repetitive, futile attempts at direct current cardioversion should be avoided. Reducing or eliminating arrhythmia precipitants may be safer and more effective than this more dramatic intervention. (Crit Care Med 2000; 28[Suppl.]:N170–N173)

KEY WORDS: atrial fibrillation; ventricular tachycardia; ventricular fibrillation; cardioversion; defibrillation

Closed-chest defibrillation was introduced in the 1950s (1, 2). The technique was popularized in the 1960s when Lown and colleagues (3) introduced transthoracic direct current cardioversion for atrial fibrillation (AF). Although direct current cardioversion remained the most effective means to terminate tachycardias and rapidly restore sinus rhythm, the approach to this procedure remained rather static until the 1990s. Significant technical advances are now available for treatment of both atrial and ventricular tachyarrhythmias.

MECHANISM OF DEFIBRILLATION

The exact mechanism of effective defibrillation is unknown. Zipes and colleagues (4) suggest that a critical mass of myocardium is depolarized and the remaining muscle mass is insufficient to maintain the reentrant tachycardia. Another group believes that defibrillating shocks prolong refractoriness in enough myocardium to terminate ventricular fibrillation (VF) (5). Shibata and associates (6) have postulated that shocks must achieve sufficient current density throughout the ventricular myocardium to prevent reinitiation of VF.

It is uncertain whether mechanisms of VF termination are applicable to AF. The success of the MAZE operation in controlling AF suggests that a critical mass of atrial tissue may be required to sustain atrial fibrillation (7). However, this surgery also isolates the pulmonary veins (the most frequent source of atrial fibrillation triggers) (8).

Atrial flutter and ventricular tachycardia are more organized tachycardias involving specific reentrant circuits. It is likely that these tachycardias can be terminated by regional depolarization in the path of their circulating wavefronts.

INDICATIONS

The most common indication for elective electrical cardioversion is persistent atrial fibrillation or flutter. Most cardiologists/electrophysiologists believe that all patients with AF deserve at least one attempt at restoration of sinus rhythm.

Emergent direct current (DC) cardioversion/defibrillation is indicated for any hemodynamically unstable sustained ventricular tachyarrhythmia. Hemodynamically stable sustained ventricular tachycardia should be treated via DC shock, if sinus rhythm is not restored promptly after intravenous antiarrhythmic drug therapy. Urgent electrical cardioversion should be contemplated for sustained supraventricular tachycardias that precipitate angina, heart failure, or hypotension.

Serial DC shocks are not appropriate for recurrent (within hours or days) paroxysms (self-terminating episodes) of AF. This scenario is relatively common in intensive care units or after cardiac surgery. Appropriate management should include treatment (or removal) of potential precipitants and institution of specific antiarrhythmic therapy (intravenous procainamide, intravenous or oral amiodarone, oral sotalol) to prevent additional episodes.

DC cardioversion is ineffective at terminating automatic tachycardias. Multifocal atrial tachycardia may superficially resemble atrial fibrillation. Careful scrutiny of the 12-lead electrocardiogram should avoid inappropriate shock delivery. Electrical cardioversion is, likewise, contraindicated in the presence of digitalis toxicity. Refractory ventricular fibrillation may ensue.

Special Considerations for the Intensivist. Patients in an intensive care setting frequently have active precipitants for reinitiation of atrial (Table 1) or ventricular arrhythmias. The include hypoxemia, excess circulating (endogenous and exogenous) catecholamines, congestive heart failure, fever (sepsis), pulmonary emboli, etc. Many of these conditions have overlapping features as well.

Administration of concomitant antiarrhythmic therapy may be difficult when renal insufficiency is present. Digoxin, procainamide, and sotalol are renally excreted and must be carefully managed (or avoided) to prevent complications.

Drugs administered orally may not be well absorbed, and intravenous agents (procainamide and amiodarone) may cause hypotension. These factors con-

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Table 1. Causes of atrial fibrillation

Atrial pressure evaluation, atrium, secondary to:
Mitral or tricuspid valve disease
Myocardial disease (primary or secondary, leading to systolic or diastolic dysfunction)
Semilunar valvular abnormalities (causing ventricular hypertrophy)
Intracardiac tumors or thrombi
Atrial ischemia
Coronary artery disease
Inflammatory or infiltrative atrial disease
Pericarditis
Amyloidosis
Myocarditis
Age-induced atrial fibrotic changes
Intoxicants
Alcohol
Carbon monoxide
Poison gas
Increased sympathetic activity
Hyperthyroidism
Pheochromocytoma
Anxiety
Alcohol
Caffeine
Drugs
Increased parasympathetic activity
Primary or metastatic disease in or adjacent to the atrial wall
Postoperative
Cardiac and pulmonary surgery
Overhydration
Pericarditis
Cardiac trauma
Hypoxia
Pneumonia
Congenital heart disease
Particularly atrial septal defect
Neurogenic
Subarachnoid hemorrhage
? Nonhemorrhagic, major stroke
Idiopathic

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spire to make prophylaxis against recurrence difficult.

The intensivist must balance complicated issues before undertaking direct current cardioversion. Strong effort should be focused on avoidance of low-yield attempts. Repeated doses of anesthesia and multiple shocks will ultimately result in further deterioration of critically ill patients. Optimal management of precipitants, careful selection and monitoring of antiarrhythmic drug therapy, as well as a solid understanding of technique will maximize success.

ANTICOAGULATION

Anticoagulation status is generally not an issue during cardioversion of ventricular arrhythmias. The goal is prompt elimination of (or protection against) life-threatening hemodynamic deterioration.

Anticoagulation plays a pivotal role in minimizing the risk of emboli and strokes during elective cardioversion of atrial fibrillation (9). Classic recommendations for AF $\geq$48 hrs in duration include 3 wks of therapeutic warfarin (prothrombin time/international normalized ratio $\geq$2.0) before DC shock and at least another 4 wks of warfarin after the procedure. Although emboli may be less frequent with atrial flutter (9), it is clear that they occur (10),and the recommendations are the same as for atrial fibrillation.

Special Considerations for the Intensivist. The intensivist rarely sees ideal candidates for classic anticoagulation. Outpatient preparation for an elective cardioversion would be an exception rather than the rule. Likewise, the intensivist sees many patients with recent or active bleeding (gastrointestinal, intracerebral, etc.) and a variety of coagulopathies that make anticoagulation absolutely or relatively contraindicated.

Short-term anticoagulation (48 hrs) with heparin before cardioversion (followed by warfarin in the usual manner) combined with transesophageal echocardiography (TEE) has gained acceptance as a alternative approach (11). Data presented at the recent annual scientific sessions (3/2000) of the American College of Cardiology from the ACUTE (Assessment of Cardioversion Using Transesophageal Echocardiography) trial suggested similar embolic rates (0.5% vs. 0.8%) comparing conventional and TEE guided approaches.

TEE is useful for detecting left atrial thrombi. It provides an excellent, minimally invasive, view of the left atrial appendage. Patients with obvious thrombi should be anticoagulated for 4–6 wks and have demonstrable resolution of clot before attempting cardioversion.

The intensivist must carefully weigh the risks and benefits of direct current cardioversion for each individual patient. Difficult decisions about the safety of both short-and long-term anticoagulation may be compounded by concomitant disease processes. At times, TEE may be the only possible assurance against emboli. However, a negative TEE is not a guarantee against emboli, and the temptation to routinely substitute a TEE for adequate anticoagulation should be strongly avoided.

TECHNICAL ASPECTS

When the capacitors of a defibrillator charge, it becomes capable of energy delivery (measured in watt-secs or joules). The energy is composed of voltage and current. Transthoracic current flow is partially determined by electrode placement. A variety of configurations have been used. We have favored an anteroposterior (parasternal and left infrascapular) pathway. This configuration provides the best vectors for energy delivery to the atria (Fig. 1) (12). We also found it to be optimal for patients with implantable cardioverter defibrillators and epicardial patches (13).

Electrode size is an important determinant of transthoracic impedance. Cur-

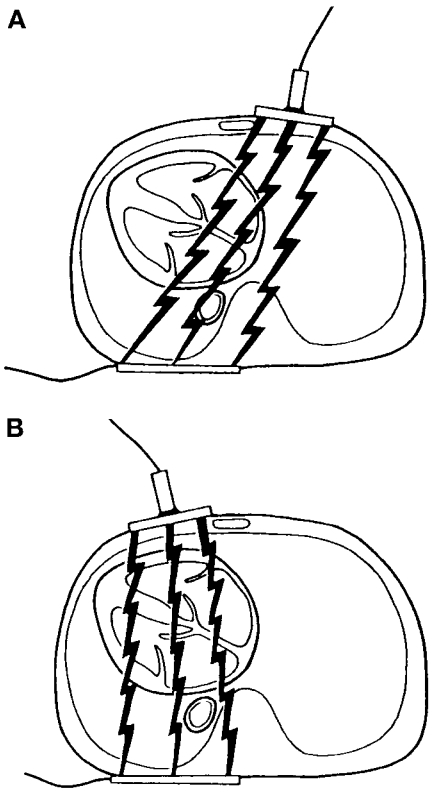

Figure 1. *Top*, right parasternal anteroposterior electrode placement. *Bottom*, left parasternal anteroposterior electrode placement. In each instance, the current vector traverses a critical mass of atrial myocardium. The right parasternal position has more of the right atrium between the electrodes and may be advantageous in patients with biatrial pathology. The left parasternal position has a smaller interelectrode distance and less lung between electrodes. It has been advocated in patients with left atrial enlargement. Adapted from *Clinical Cardiology* (1994; 17:79–84) with permission from Clinical Cardiology Publishing Co. Inc., and/or the Foundation for Advances in Medicine and Science, Inc.

rent flow is inversely related to impedance. Optimal paddle size ranges from 8–12 cm. A couplant or conductive paste/gel (a variety of effective agents are available, Hewlett-Packard Redux paste [Parker Laboratories, Fairfield, NJ] is reportedly one of the best) must be used between the metal electrodes and the chest wall (12). Smearing of gel between paddles may deflect energy away from the heart (14).

Self-adhesives pads are commonly used in high-risk patients. They are easy to position precisely. Transthoracic impedance may be higher with these pads compared with metal electrodes (12).

DC shocks should never be administered to a conscious patient. Even low-energy shocks (≥1 joule) may be painful. A high energy shock delivered to an awake patient may result in lifelong emotional trauma and has appropriately been termed a "calamity" (15).

Controversy exists over whether an anesthesiologist must be present for the procedure. Individual hospitals have different policies (16). Our electrophysiology group has had extensive experience administering anesthesia for *elective* cardioversion. We believe that the agent of choice is methohexital delivered as an intravenous bolus in a dose of 0.6 mg/kg. The incidence of respiratory compromise requiring intubation is extremely low. Most of this experience has taken place in the electrophysiology laboratory without the presence of an anesthesiologist (17). We have safely administered methohexital in various intensive care units without precipitating respiratory crises (unpublished observations); however, caution should be used in extrapolating results from the electrophysiology laboratory to the critical care setting. Regardless of who administers the anesthesia, expert ability to manage the airway must be immediately available. We recommend that an anesthesiologist be present for very high-risk patients.

Energy Selection. Although it has been common to recommend an initial energy of 100 joules for atrial fibrillation (with initial success rates of 50%), we agree with Ewy (12) and begin with 200 joules. A recent report advocates an initial energy of 360 J for atrial fibrillation of >48 hrs duration (17a). We recently reassessed optimal energy delivery for cardioversion of atrial flutter. Starting with 100 joules is as safe as 50 and appears to be more efficient. (18) Monomorphic ventricular tachycardia should be converted with 100–200 joules (much lower energies may be effective) whereas polymorphic ventricular tachycardia or VF should be initially defibrillated with 200 joules (19). Energies are titrated upward as rapidly as required.

Table 2. Determinants of transthoracic or transchest resistance to DC defibrillator or cardioverter capacitor discharge

Stored energy
Electrode size
Electrode composition
Interface between the electrode and the skin
Distance between electrodes
Number of previous shocks
Time interval between previous and present shock
Pressure on the electrodes
The phase of ventilation
Patients' body build
Recent sternotomy

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R-wave synchronization should be assured during cardioversion of arrhythmias with well-defined QRS complexes. Failure to do so may lead to shock delivery within the "vulnerable period" of the T-wave and induction of VF.

Determinants of Short and Long-Term Success. It has already been noted that electrode size and placement, as well as transthoracic impedance, influence current flow and procedural outcome. Impedance, in turn, is influenced by a variety of factors (in addition to those previously described). These include (Table 2) the phase of ventilation (expiration has lower impedance than inspiration), distance between electrodes, pressure on electrodes (air does not conduct well), effect of previous discharges (decreased impedance), time between discharges (waiting as long as 3 mins may provide continued decreases in impedance), and patient body habitus (high weights or increased body mass index decrease success) (12, 20).

Poor long-term success in atrial fibrillation relates to arrhythmia duration (>1 yr) and large left atrial diameter (>5 cm). Untreated hyperthyroidism, mitral stenosis, or congestive heart failure increases the likelihood of recurrence. Use of concomitant antiarrhythmic drugs (especially amiodarone) may help maintain sinus rhythm. Atrial flutter recurrences are hard to prevent even with pharmacotherapy. Recurrences of typical atrial flutter should be eliminated with radiofrequency catheter ablation.

Patients with structural heart disease and sustained ventricular tachycardia or VF may be at risk for sudden cardiac death. If reversible causes cannot be identified, they should be considered candidates for an implantable cardioverter defibrillator. Patients with sustained ventricular tachyarrhythmias in the presence of multiple system organ failure may have very high in-hospital mortality rates despite aggressive antiarrhythmic therapy (21). Long-term management should be individualized, based, in part, on their overall recovery.

OPTIONS FOR REFRACTORY PATIENTS

The intensivist must weigh the risk and benefits of repeated attempts at DC cardioversion. Care should be taken to avoid serial futile shocks. Nevertheless, an awareness of available options is essential. Most hospitals have defibrillators that deliver up to 360 joules. Standard waveforms have been monophasic (damped sinusoidal or truncated exponential). A switch from self-adhesive pads to paddles with pressure is a simple method to increase procedural success.

Internal cardioversion has been used as an emergent option for VF during electrophysiologic testing and electively for atrial fibrillation (22, 23). The most successful application of internal cardioversion/defibrillation has been the implantable cardioverter defibrillator. Device implantation was dramatically advanced by the development of biphasic shocking waveforms that permitted routine transvenous (nonthoracotomy) lead placement, and generator size reduction (via advances in capacitor technology) allowing subcutaneous pectoral pocket location.

Internal cardioversion for AF is effective in ~70% to 80% of patients refractory to 360 joules. Transvenous placement of both right atrial and coronary sinus multipolar catheters and biphasic shock waveforms markedly reduced energy required for successful cardioversion. Unfortunately, catheter placement may require reduction or discontinuation of anticoagulation increasing the risk of emboli. Despite very low energy requirements (as low as 2–3 joules with biphasic waveforms), the procedure remains painful and requires sedation or anesthesia (23). Implantable permanent atrioverters

have not gained widespread acceptance because the shocks are painful.

In 1997, Trohman and associates (24) reported the use of higher energy transthoracic shocks (720 joules) in large patients with AF refractory to 360 joules. Success rates were similar to those seen with internal cardioversion. These results were confirmed in a larger series (20). Despite early reports of myocardial necrosis with energies >400 joules, hemodynamic compromise was not seen in our patients. This technique has gained some degree of clinical acceptance (25).

We did not envision using 720-joule shocks as a long-term technical solution. Our goal was to demonstrate that higher success rates could be achieved transthoracically. We hoped this technique would be a "stepping stone" and would spur development of R-wave synchronized transthoracic biphasic cardioversion. The automatic external defibrillator had demonstrated that transthoracic biphasic waveforms defibrillated VF with lower energies than monophasic devices (26). However, these devices did not permit R-wave synchronization and were impractical for organized tachyarrhythmias.

Recent investigations have confirmed that rectilinear biphasic waveforms are indeed more effective for cardioversion of atrial fibrillation (27). Higher success rates with lower energy requirements have been observed. Biphasic defibrillators became commercially available in 2000. It seems inevitable that biphasic external cardioverter defibrillators will become the standard of care within a relatively short period of time.

Direct current cardioversion/defibrillation is a useful tool in the intensivist's armamentarium. When used emergently, it may be lifesaving. Elective cardioversion requires a correct tachyarrhythmia diagnosis, careful patient selection and optimization, an understanding of proper technique and energy selection, assessment of and protection from embolic risk, optimal anesthesia, expert airway protection, and prophylaxis against recurrence.

Although this procedure seems simple and is often taken for granted, improper attention to detail can make it futile or deleterious. There is no excuse for failure to consider or check the patient's anticoagulation status. Shocks delivered to an awake patient are completely inexcusable. Failure to synchronize to the R-wave may result in VF. Inappropriate shocks delivered for sinus or automatic tachycardias can be avoided via careful electrocardiographic analysis. A hemodynamically stable patient should not be shocked based solely on a one-lead rhythm strip. Futile repeat shocks in the setting of near-certain recurrence are unnecessary and foolhardy. Treating underlying precipitants may be far more important than rapid establishment of a "clean" arrhythmia-free electrocardiogram in determining the patient's ultimate recovery.

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