CARDIOVERSION

(Roberts: Clinical Procedures in Emergency Medicine, 4th ed. 2004)

Cardioversion is the application of direct electrical current across the chest or directly across the ventricle to normalize the conduction pattern of a rapidly beating heart. Defibrillation refers to application of electrical energy during the nonvulnerable period to restore a fibrillating ventricle to normal sinus rhythm.

The patient with a significant tachycardia may be asymptomatic or may complain of chest pain or discomfort, lightheadedness, or shortness of breath. These symptoms are the result of altered cardiovascular physiology. Rapid cardiac rhythms allow less time for ventricular filling, resulting in reduced preload and hypotension. The reduced preload as well as the increased ventricular work caused by the rapid heart rate may also result in ventricular ischemia. Pulmonary capillary wedge pressures may also rise despite shortened filling time, due to reduced ventricular compliance secondary to ventricular ischemia. Elevated pulmonary capillary wedge pressures can then lead to pulmonary edema.

Termination of rapid rhythms to alleviate or prevent these symptoms must occur quickly to prevent further deterioration. Persistently poor cardiac output due to rapid heart rate results in development of a lactic acidosis that further compromises cardiac function and makes cessation of the dysrhythmia even more difficult. Unchecked myocardial ischemia may lead to infarction with its attendant sequel. Drug therapy, rapid cardiac pacing, and cardioversion are the methods available to terminate tachydysrhythmias.

In many cases, direct-current cardioversion has specific advantages over drug therapy. The speed and simplicity of electrical cardioversion enhance its usefulness in the ED setting. Cardioversion is effective almost immediately, has few side effects, and is often more successful than drug therapy in terminating dysrhythmias. In addition, the effective dose of many antidysrhythmic medications is variable, and there is often a small margin between therapeutic and toxic dosages. Although they can often suppress an undesirable rhythm, drugs may also suppress a normal sinus mechanism or may create toxic manifestations that are more severe than the dysrhythmia being treated.

In the clinical setting of hypotension or acute cardiopulmonary collapse, cardioversion may be life saving. The key concepts in the use of this procedure include understanding the indications for its use, the equipment involved, the importance of adequate sedation, and the concerns for health-worker safety.

BACKGROUND

The first successful defibrillation of the human heart was performed in 1947 by Beck. By the 1960s, electrical energy was being used to treat dysrhythmias other than VF. AC remained in vogue until 1962, when Lown and colleagues advocated DC countershock as the method of choice for terminating atrial fibrillation.[175] The use of DC significantly decreased the incidence of VF following countershock.

A brief burst of electrical current momentarily causes depolarization of the majority of cardiac cells and allows the sinus node to resume normal pacemaker function. In reentrant dysrhythmias, such as paroxysmal supraventricular tachycardia (SVT) and VT, cardioversion restores sinus rhythm by interrupting a self-perpetuating circuit. Cardioversion is much less effective in terminating tachycardias resulting from augmented automaticity, such as digitalis-induced dysrhythmias.

Monophasic versus Biphasic Wave Form Defibrillators

From the pioneering days of defibrillation technology in the 1950s until the start of the 20th century, traditional defibrillators generated a monophasic damped sine wave form to deliver the energy needed to convert tachydysrhythmias. Starting in the 1990s, biphasic technology was introduced for AEDs and automatic implantable cardiac defibrillators (AICD). The biphasic defibrillator is now positioned to replace the monophasic waveform defibrillator in all hospital-based defibrillators.

In contrast to the monophasic defibrillator, the biphasic wave generator delivers current in two directions. During the first phase, the biphasic current travels from one paddle to the other, similar to the monophasic wave form. However, during the second phase, the current reverses direction. This distinctive feature has been proven to lower the electrical threshold for successful defibrillation and cardioversion.
INDICATIONS AND CONTRAINDICATIONS

Cardioversion is indicated whenever a reentrant tachycardia causes unstable vital signs, ischemic chest pain, or otherwise significantly compromises cardiovascular or respiratory function. No specific parameters exist and the decision to perform cardioversion is best made on a case by case basis by the clinician at the bedside. It is also indicated on an elective basis when medical therapy has failed. In any situation where a prolonged rapid heartbeat can be anticipated to cause complications related to cardiac ischemia or dysfunction, early intervention with cardioversion may also be considered.

A reentrant tachyarrhythmia should be suspected when a sudden change in the heart rate occurs within a few beats. Unless the dysrhythmia is noted while the patient is being monitored, it can only be inferred from the patient's history of sudden onset of symptoms. In the unusual case of sinus node reentrant tachycardia, rapid onset and offset may be the only clue. Other clues to the presence of a reentrant dysrhythmia are a history of Wolff-Parkinson-White syndrome or another known accessory pathway syndrome. Ventricular rates in excess of those predicted for age strongly suggest an accessory pathway.

Dysrhythmias due to enhanced automaticity will not be terminated by uniformly depolarizing myocardial tissue because a homogeneous depolarization state already exists. Enhanced automaticity is the cause of most cases of digitalis toxicity-induced dysrhythmia, sinus tachycardia, and, probably, multifocal atrial tachycardia. Enhanced automaticity means that the threshold for phase 4 depolarization has been lowered or that the rate of ion leak during phase 4 has been accelerated. This effect on phase 4 depolarization is caused by alterations in the metabolic or chemical environment or on the cell membrane, causing pacemaker cells to fire more rapidly. Although cardioversion will not be successful in these cases, medications that suppress automaticity, including potassium and magnesium, may be useful.

In digoxin toxicity, cardioversion is not only ineffective, but it is also associated with a higher incidence of postshock VT and VF. However, for a patient with a therapeutic digoxin level, the risk of cardioversion is no different from that of other patients. Digoxin is still generally withheld for 24 hours prior to cardioversion as a precaution against inadvertently elevated levels. Pregnancy at any stage is not a contraindication to cardioversion.

Supraventricular Tachycardia with Aberrancy versus Ventricular Tachycardia

Determining the rhythm is critical for the clinician to make the appropriate clinical/pharmacologic intervention. However, at times SVT may manifest patterns on the ECG that look very similar to VT. An incorrect assessment of the ECG can prompt the clinician to implement a pharmacologic/therapeutic intervention which may result in cardiovascular collapse. Although a comprehensive discussion of SVT versus VT is beyond the scope of this chapter, some salient features to look for are presented herein to assist in the emergent decisionmaking process of the clinician.

A few caveats must be kept in the forefront when facing the task of discriminating between SVT and VT. A wide-complex tachycardia refers to a dysrhythmia where the ventricles beat at more than 100 beats/minute and the QRS duration is 0.12 seconds or more. The originating foci for these wide-complex tachycardias can be either supraventricular or ventricular. For a supraventricular focus to produce a wide-complex tachycardia, a preexisting or new onset intraventricular conduction block must be present, resulting in increased time of depolarization. Increased heart rate or ischemia can also precipitate the appearance of a wide-complex tachycardia when it is supraventricular in origin. If the focus of the tachycardia is below the AV node, the tachycardia is considered ventricular in origin.

Criteria to facilitate the process of discriminating between SVT and VT were compiled by Wellens and Brugada. The Wellens criteria use several clinical data points to help determine if the tachycardia is ventricular or supraventricular in origin. The Brugada criteria extend the Wellens criteria and add a four-step decision-tree approach to the process. Although the methods are not without pitfalls, a careful scrutiny of the ECG in light of the aforementioned criteria will usually lead to the appropriate diagnosis.

Following are characteristics of SVT versus VT. Although the guidelines and criteria appear clear-cut, there are times when exceptions occur in the clinical setting (Table 12-4, Table 12-5, Table 12-6, and Table 12-7).
TABLE 12-4. Standard Criteria to Differentiate SVT from VT

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate:</td>
<td>Too much overlap to make rate a useful criterion.</td>
</tr>
<tr>
<td>Regularity:</td>
<td>Grossly irregular complexes are likely to represent one of three conductions: (1) AF with aberrancy, (2) AF with conduction through an accessory pathway, or (3) irregular form of VT. Rates greater than 200 bpm with a wide-QRS-complex are highly suggestive of AF with aberrancy or AF with accessory pathway conduction.</td>
</tr>
<tr>
<td>AV Dissociation, Fusion Beats: (Table 12-6)</td>
<td>AV dissociation during tachycardia with a wide-complex is highly suggestive of VT.</td>
</tr>
<tr>
<td>QRS Axis:</td>
<td>Preservation of normal QRS axis in a wide-complex tachycardia favors a diagnosis of SVT with aberrancy. Change in axis or extreme left or right axis deviation is often seen in VT. Abrupt change in QRS morphology is frequently seen in VT but not in SVT.</td>
</tr>
<tr>
<td>QRS Duration:</td>
<td>QRS duration greater than 140 ms occurs more frequently in VT compared to SVT.</td>
</tr>
<tr>
<td>QRS Concordance:</td>
<td>Concordance in the precordial leads is rarely seen in SVT with aberrancy.</td>
</tr>
</tbody>
</table>
| QRS Morphology:                          | **RBBB-shaped complex**: To distinguish RBBB aberration in SVT from VT, the presence of a triphasic pattern in V1 or a qRs pattern favors aberrancy. While a monophasic or biphasic QRS in V1 or an rS or QS complex in V6 favors VT. \  
**LBBB-shaped complex**: SVT is suggested if the LBBB pattern has a small initial r wave with a steeply down sloping S wave. An initial r wave (>30 ms) and a notched, broad (>70 ms) down slope is favorable for VT. QS complex or a qR in V6 suggests VT. |

AF, atrial fibrillation; AV, atrioventricular; LBBB, left bundle-branch block; QRS, QRS Interval; RBBB, right bundle-branch block; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

TABLE 12-5. Brugada Algorithm

First reported in 1991, this algorithm was designed to aid the clinician in diagnosing lethal VT from the less urgent SVT with aberrancy conduction. Taking the standard 8- to 10-point criteria used by cardiology at the time, Brugada et al. focused on four ECG criteria to aid in diagnosing VT versus SVT with aberrancy. To differentiate VT from SVT with aberrancy, the Brugada Algorithm uses the following ECG criteria. First, examine the ECG. Is the rhythm regular? An irregular rhythm highly suggests atrial fibrillation with aberrancy. Does the purpose dysrhythmia fit the clinical picture ascertained by the history? Then, ask the following questions:

- Absence of an RS complex in all precordial chest leads?
- R to S interval > 100 ms in one precordial lead? (measured from beginning of R to deepest part of S wave)
- Atrioventricular dissociation? (Table 12-4)
- Morphology criteria for VT present in precordial chest leads V1-2 and V6? (Table 12-4)

(A single "yes" response suggests VT  
Only when none of the VT criteria are affirmed is SVT diagnosed  
Adapted from Brugada et al., 1991).

Wellens Criteria

In a landmark study published in 1978, Wellens et al. determined that the findings suggestive for a ventricular origin of tachycardia were the following:

- QRS width over 0.14 sec
- Left axis deviation
- AV dissociation (Table 12-4)
- Certain configurational characteristics of the QRS morphology (Table 12-4)

AV, atrioventricular; ECG, electrocardiogram; QRS, QRS Interval; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

*Wellens et al., 1978*
TABLE 12-6. Electrocardiogram (ECG) Evidence of Atrio-Ventricular Dissociation

1. Dissociated P waves at a slower rate than the ventricular rate
2. Presence of Fusion or Capture beats
3. Irregular changes in ST-T waves suggesting presence of dissociated P waves
4. P wave and QRS complexes at different rates on adjunct ECG recording (esophageal or nasogastric approach)


TABLE 12-7. Characteristics of Wide QRS Tachycardia Suggesting Ventricular Tachycardia

1. AV Dissociation, Fusion or Capture Beats
2. Positive or Negative Concordance in V1–V6
3. QRS Configuration
   RBBB-shaped:
   V1: Monophasic or biphasic QRS complex
   V6: R:S ratio < 1
   LBBB-shaped:
   V1: r > 30 ms, S > 70 ms, notched S
   V6: qR pattern

AV, atrioventricular; LBBB, left bundle-branch block; QRS, QRS Interval; RBBB, right bundle-branch block.


Special Considerations: Wide-QRS-Complex Tachycardias

Wide-complex tachycardias (wide-complex SVT) are diagnostic challenges in clinical medicine. The criteria often used to define wide-complex SVT (WCSVT) is a tachycardia with a QRS duration of >0.12 seconds. It is important to differentiate the rhythm as one of the following: VT, SVT with aberrancy (LBBB or RBBB), or an accessory AV pathway (“pre-excitation”). The need for a proper diagnosis is obvious. Incorrect diagnosis and inappropriate treatment can be life threatening. This is especially true in misdiagnosing VT as SVT. Studies have shown that VT is often misdiagnosed, even with the ready availability of clinical and ECG criteria. In one study, VT was correctly diagnosed in 32% of patients presenting with wide-complex tachycardias.[184]

Etiology

Normally, ventricular depolarization is initiated when the His bundle depolarizes both ventricles simultaneously through the bundle branches and Purkinje fibers. Normal depolarization takes place within 80 to 120 ms. Prolongation of the QRS duration happens if (1) the ventricles are activated sequentially rather than simultaneously (this is the case in VT, bundle-branch blocks, or accessory pathway ventricular activation [WPW, LGL]), or (2) when His-Purkinje-myocardium conduction is slowed from ischemia, drugs, or electrolyte disturbances.

Classification

Wide-complex tachycardias fall into three classifications based on mechanism: (1) VT, (2) SVT with aberration, and (3) pre-excitation tachycardia. VT is the most common cause of wide-QRS-complex tachycardias. It is defined as 3 or more consecutive ventricular beats at a rate of 100 beats/minute. VT is further classified as nonsustained (tachycardia lasting <30 seconds) or sustained (tachycardia that lasts >30 seconds). Sustained tachycardia usually results in hypotension or syncope and requires termination intervention. SVT is a tachydyrsrhythmia using the normal AV conduction system for ventricular activation. This tachycardia originates in the SA or AV node. To sustain propagation, the AV node is recruited. Aberrancy refers to the existence of an aberrant or nontraditional conduction mechanism, resulting in a longer depolarization phase. SVTs with aberrancy by definition must result in wide-QRS-complex tachycardias. The two forms of aberration are fixed (i.e., a permanent bundle-branch block or functional block that is a rate-dependent bundle-branch block). The most common areas of the His-Purkinje functional block are in the left or right bundle-branches. Sudden acceleration is often the initiating cause of the SVT with aberrancy. The aberrancy is maintained by a continuous, concealed, retrograde conduction pathway, which leads back into the blocked area.
For preexcitation wide-QRS-complex tachycardias, AV conduction occurs over two circuits: (1) normal AV nodal conduction or (2) through an accessory pathway. These two pathways create a reentry circuit. Aberration appears because of the presence of intraventricular conduction block. Preexcited tachycardia is any tachycardia where the ventricles are antegrade activated over an accessory pathway. The most common preexcited tachycardia is atrial fibrillation with ventricular activation over an accessory pathway.

**Clinical Diagnosis**

The directed history on presentation may provide the most valuable clues to the diagnosis of tachydysrhythmia. The on and off occurrence of tachydysrhythmia in the past, the age of the patient, and the age of past occurrence all are important indicators of preexcitation rhythms usually found in young patients. Sudden onset of tachydysrhythmia in the older coronary-prone patient or patient with structural heart disease points more toward ventricular tachycardias. Symptoms associated with the tachydysrhythmia are important clues in diagnosis. The young patient often has few if any symptoms when experiencing the wide-complex or narrow-complex SVTs. The older patient may experience the entire range of cardiac symptoms. Tachydysrhythmia present for long periods often defines VT. Patients with SVT often have recurrent tachycardias from their childhood or early adulthood. Attention must be paid to the medications the patient is using. Antiarrhythmic medications have a use-dependency property. Conduction velocity is slowed as rates increase.\footnote{187}

**Electrocardiographic Criteria for Differentiating Ventricular Tachycardia from Wide-Complex Supraventricular Tachycardia**

The clinician should not attempt the differential diagnosis of wide-QRS-complex tachycardias without the use of the 12-lead ECG and extended rhythm strip. A common error is to attempt to determine the cause of a tachycardia based only on the rhythm strip. A comparison of past ECGs is often helpful. Examination of the ECG should focus on the following areas: rate, regularity, AV dissociation, QRS axis, QRS duration, QRS concordance, and QRS morphology (see Table 12-4).

**Treatment**

Therapy is dictated by the specific wide-complex tachycardia and the patient's clinical presentation. The emergency clinician's initial approach must always be led, and modified if necessary, by the patient's presentation and subsequent changes. It is recommended in all cases of wide-complex tachycardias, and narrow-complex tachycardias, which are producing hemodynamic instability, that the clinician should immediately consider use of cardioversion. Synchronized monophasic or biphasic cardioversion is the appropriate first choice of treatment for these cases.

In patients with wide-complex tachycardias who are cardiovascularly stable, the therapeutic options are more diverse. Stable, wide-complex tachycardia can always be considered VT and treated according to current VT protocols.\footnote{188} Verapamil should never be used in unknown etiology wide-complex tachycardia. A reasonable treatment protocol for stable patients may be the use of adenosine, procainamide, lidocaine, and finally cardioversion. Amiodarone is effective for most SVTs and its use in stable unknown wide-complex SVT is both appropriate and safe.\footnote{189}

**EQUIPMENT AND SETUP FOR CARDIOVERSION**

The critical components of preparation for cardioversion are IV access, airway management equipment, drugs for sedation, monitoring equipment, and DC delivery equipment (cardioverter).

Secure IV access is essential for delivery of sedatives, antidyssrhythmics, fluids, and, possibly, paralytic agents. Although many of these drugs are not used routinely, if they are needed, timing is likely to be critical. A saline lock catheter of at least 20 ga should be inserted and secured.

A significant and preventable complication of procedures involving sedation is hypoventilation leading to hypoxia. Airway management equipment includes working suction with tonsil-tipped device attached, bag-valve-mask apparatus, oxygen, and an appropriately sized laryngoscope and endotracheal tube. A pulse oximeter is generally recommended for patients undergoing conscious sedation. Another adjunct is continuous pCO\textsubscript{2} monitoring. A rising pCO\textsubscript{2} level will be an earlier clue to hypoventilation due to sedation than pulse oximetry because the O\textsubscript{2} saturation may remain normal for several minutes, especially if the patient has been preoxygenated.
Sedative medications should be ready for use in labeled syringes, with a prefilled saline flush. Antidysrhythmic medications for ventricular dysrhythmias (e.g., lidocaine and amiodarone) and for unexpected bradycardia (e.g., atropine) should be readily accessible.

The cardioverter device consists of 5 components (see Fig. 12-7 (Figure Not Available) ): (1) a DC depolarizer, which provides varying amounts of electrical current; (2) an oscilloscope screen for monitoring heart rate and rhythm; (3) access to a continuous ECG readout to document the patient’s course and response to treatment; (4) 2 removable electrode paddles that can be applied easily to the patient's chest wall; and (5) a synchronizer, permitting discharge of energy outside the vulnerable period of the cardiac cycle. The synchronizer permits triggering of the electrical discharge by the R or S wave of the ECG (see Fig. 12-3).

![Figure 12-3. Phases of vulnerability for atrium and ventricle. Note that an alternating current shock of 0.20 seconds may end at the T wave even when synchronized with the R wave of the electrocardiogram. (From Resnekov L: Theory and practice of electroversion in cardiac dysrhythmias. Med Clin North Am 60:325, 1976. Reproduced by permission.)](image)

Paddles must be large enough to depolarize the majority of heart fibers simultaneously; therefore, most conventional paddles have an electrode diameter of at least 4 inches. Larger paddles also limit the risk of myocardial injury by decreasing the density of current passing through the myocardium.

**TECHNIQUE**

If time permits, metabolic abnormalities such as hypokalemia and hypomagnesemia should be corrected before attempting cardioversion. Hypoxia should be corrected with supplemental \( O_2 \). If a patient has a metabolic acidosis, compensatory hyperventilation after endotracheal intubation may be indicated prior to cardioversion. Respiratory acidosis should always be treated prior to the use of sedative drugs.

**Sedation and Anxiolysis**

Cardioversion may be extremely painful or terrifying, and **patients must be adequately sedated prior to its use**. Patients who are not adequately sedated may experience extreme anxieties and fear. Several IV medications are available for sedation of patients prior to cardioversion. These include etomidate (0.15 mg/kg), midazolam (0.05–0.1 mg/kg), methohexital (1 mg/kg), propofol (0.5–0.8 mg/kg over 3–5 minutes), and thiopental (3 mg/kg). In addition, fentanyl (1–2 mcg/kg), a synthetic opioid analgesic, is sometimes administered 3 minutes prior to induction. The above doses are commonly used, but the agents should be titrated to effect as outlined in Chapter 34.
Midazolam (Versed) is probably the most commonly used agent, with deep sedation occurring about 2 minutes after IV injection. Although induction with midazolam takes slightly longer than the other medications, it has the advantage that a commercial antagonist, flumazenil, is available for reversal, if necessary. Small additional doses of fentanyl (1 to 1.5 mcg/kg) may be added for more profound sedation. Fentanyl can cause respiratory depression, but it is reversed with naloxone. Methohexital has the advantage of quick onset and somewhat shorter duration than midazolam, but it has a rare association with laryngospasm. All the drugs except etomidate cause a small drop in blood pressure.

In elderly patients the pharmacodynamics and kinetics of medications used for sedation/anxiolysis are altered by coexisting illness and polypharmacy, rather than by any intrinsic effect of old age. Older patients with medical conditions such as congestive heart failure, renal failure, cancer, or malnutrition will therefore experience deeper, prolonged sedation with increased respiratory depression. Drug dose should be adjusted accordingly in these patients.

Cardioverter Use

Selection of synchronized or nonsynchronized mode is the next critical step. In “synchronized” mode, the cardioverter searches for a large positive or negative deflection, which it interprets as the R or S wave. It then automatically discharges an electric current that lasts <4 msec, avoiding the “vulnerable” period (see Fig. 12-3) during repolarization when VF may be induced. When the cardioverter is set to “synchronize,” a brief delay will occur after the buttons are pushed for discharge, as the machine searches for an R wave. This delay may be disconcerting to the unaware operator.

If concern exists about whether the R wave is large enough to trigger the electrical discharge, the clinician can place the lubricated paddles together and press the discharge button. Firing should occur after a brief delay. When the R- or S-wave deflection is too small to trigger firing, change the lead that the monitor is reading or move the arm leads closer to the chest.

If there is no R or S wave to sense, as in VF, then the cardioverter will not fire. Always turn off "synchronization" if VF is noted.

Electrode Contact

A number of substances can be used to ensure good contact between the paddle and the skin, it is necessary that they be nonflammable and have a low electrical resistance. Conductive gel or paste is most commonly used, but waxy conductive pads are also available. Generous use of conductive gel on the underside and especially along the edges of the electrode paddles is essential, both to reduce TTI and to prevent skin burns. Paste should be applied liberally but must not run onto the skin between the paddles, because the paste may divert current over the skin surface and away from the heart. Even under ideal circumstances, only 10% to 30% of the total current passes through the heart, so diversion over the skin may significantly reduce the effectiveness of an electric discharge. Saline-soaked pads are therefore generally not desirable. Pregelled adhesive electrode pads are useful if available.

Wet Gel Electrodes

The tendency for solid gel electrode pads to burn patients during cardioversion has led to the re-introduction of wet gel electrodes. The return of the wet gel electrodes was centered on conductivity and skin coupling features. On both issues, wet gel electrodes performed better than solid gel electrodes.

Electrode Position

Electrode paddles may be positioned in 2 ways on the chest wall: (1) the anterolateral (or base and apex) position, with one paddle placed in the left fourth to fifth intercostal space, midaxillary line, and the other just to the right of the sternal margin in the second to third intercostal space (see Fig. 12-10); or (2) the anteroposterior position, with one paddle placed anteriorly over the sternum and the other on the back between the scapulae (see Fig. 12-11). The anterolateral position is used for emergent cardioversion, when placement of an electrode on the patient's back may not be feasible. Paddles should be pressed firmly against the skin to avoid arcing or skin burns.
Safety is a key concern in the performance of cardioversion. Any staff member acting as a ground for the electrical discharge can be seriously injured. The operator must announce "all clear" and give staff a chance to move away from the bed before discharging the paddles. Care must be taken to clean up spills of saline or water, because they may create a conductive path to a staff person at the bedside.

Energy Requirements

The amount of energy required for cardioversion varies with the type of dysrhythmia, the degree of metabolic derangement, and the configuration and thickness of the chest wall (see Table 12-2). Obese patients may require a higher energy level for cardioversion; the anteroposterior paddle position is sometimes more effective in these patients. If patients are shocked while in the expiratory phase of their respiratory cycle, energy requirements may also be lower.

Ventricular tachycardia in a hemodynamically stable patient should be treated with lidocaine followed by procainamide, and amiodarone if necessary. If these drugs are unsuccessful, cardioversion is then used. Cardioversion with 10 to 20 J is successful in converting VT in more than 80% of cases. Cardioversion will be accomplished with 50 J in 90% of cases, and conversion should be initially attempted at this energy level.194 Cardioversion should be synchronized unless the T wave is large and could be misread as the R wave by the cardioverter. If initial attempts of electrical cardioversion are unsuccessful, the energy level should be doubled, and doubled again if necessary, until a perfusing rhythm is restored. Immediately following conversion of VT, antidysrhythmic medication should be given to prevent recurrence.

Patients with pulseless VT should be initially shocked with 200 J, followed by 300 J if the first shock is not successful. Reentrant SVTs generally respond to low-energy levels. Atrial flutter, for example, usually requires <50 J for conversion.26 Cardioversion of atrial flutter in the ED is indicated when the ventricular rate is not slowing in response to pharmacologically enhanced AV-node blockade, or if the patient is unable to tolerate the aberrant rhythm.

The majority of patients with paroxysmal atrial tachycardia (PAT) respond to adenosine. If they do not, or if urgent conversion is needed due to high ventricular rate, electric countershock should be administered in the synchronized mode at 50 J, and doubled if necessary.

In atrial fibrillation, the response to cardioversion is dependent on the duration of atrial fibrillation and the underlying cause. Cardioversion is successful in 90% of cases secondary to hyperthyroidism but in only 25% of cases secondary to severe mitral regurgitation.195 However, 50% of cases revert within 6 months, especially those with longstanding atrial fibrillation.196 [197]

Most patients with atrial fibrillation do not require cardioversion in the ED unless their ventricular response is excessively rapid due to a bypass tract, as in Wolff-Parkinson-White syndrome. Atrial fibrillation may also require cardioversion when sequelae of rapid ventricular contraction are present or anticipated and the ventricular rate is not responding to drug therapy aimed at slowing AV node conduction. Conversion of atrial fibrillation generally requires more energy than the reentrant SVTs (about 100 J in most cases).198

### Pediatric Cardioversion

Pediatric cardioversion is similar to adult cardioversion. As previously described, the purpose of the procedure is to depolarize the myocytes completely at the most opportune time, during the peak of the R wave, so as not to precipitate VF, and allow a slower perfusing rhythm to resume. However, the energy levels for pediatric cardioversion are different from the adult. In the pediatric procedure the initial recommended energy dose is 0.5 to 1 J/kg, while the defibrillator is in the synchronized mode. If needed, a repeated cardioversion may be attempted at 2 J/kg, again while the defibrillator is in the synchronized mode! Remember to re-synchronize the defibrillator after each cardioversion attempt and look for the appropriate markers on the monitor to ensure that the current is delivered at the appropriate phase of the cardiac cycle (see Fig. 12-3).

<table>
<thead>
<tr>
<th>Determinant</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inter-electrode distance (chest size)</td>
<td>Larger distance → higher impedance</td>
</tr>
<tr>
<td>Energy selected</td>
<td>Higher energy → lower impedance</td>
</tr>
<tr>
<td>Electrode size</td>
<td>Larger electrode → lower impedance</td>
</tr>
<tr>
<td>Electrode-skin couplant</td>
<td>Failure to use a couplant → very high impedance</td>
</tr>
<tr>
<td>Previous shocks</td>
<td>Previous shock → lower impedance, especially after first shock</td>
</tr>
<tr>
<td>Phase of respiration</td>
<td>Inspiration → higher impedance</td>
</tr>
<tr>
<td>Electrode-chest contact pressure (handheld paddles)</td>
<td>Firm pressure → lower impedance</td>
</tr>
</tbody>
</table>

COMPLICATIONS OF CARDIOVERSION

Complications of cardioversion may affect the patient, particularly the patient with a cardiac pacemaker, as well as health care personnel at the bedside. Patient complications are dose-related and may involve the airway, heart, or chest wall, or they may be psychological.

Injuries to health care personnel with cardioversion/defibrillation include mild shock and burns. There is a reported incidence of 1 injury per 1700 shocks for paramedics in the field. Of the 13 injuries reported in this study, 15% were due to equipment failures, 23% were due to injuries during testing or demonstrations, and 23% were due to arcing of the electric shock from the paddle to an electrode on the patient's chest, whereas the remainder were due to direct contact with the patient or with the stretcher.

Hypoxia may result if sedation is excessive or if the airway becomes compromised. With proper preparations and precautions, airway complications can be minimized. Respirations may also be depressed by any of the anesthetic agents, and adequacy of tidal volume must be continually assessed by either direct observation or end-tidal CO\textsubscript{2} monitoring. An assistant should be placed in charge of monitoring the patient's airway cardiac monitor, and pulse oximetry. Routine supplemental O\textsubscript{2} is suggested for all patients undergoing sedation.

Chest wall burns resulting from electrical arcing are generally superficial partial-thickness burns, although deep partial-thickness burns have occurred. These are preventable by adequate application of conductive gel and firm pressure on the paddles. Paddles should not be placed over medication patches or ointments, especially those containing nitroglycerin, because electrical discharge may cause ignition resulting in chest burns.

Cardiac complications following cardioversion are proportionate to the energy dose delivered. In the moderate energy levels used most commonly, the hemodynamic effects are small. At higher energy levels, however, complications include dysrhythmias, hypotension, and, rarely, pulmonary edema, which may occur several hours after the countershock. A transient failure of myocardial O\textsubscript{2} extraction due to a direct effect on cellular mitochondria has been proposed as an explanation for some of these cardiac complications.

The dysrhythmias following high-dose (approximately 200 J) direct-current shocks include VT and VF, bradycardia, and AV block, in addition to transient and sustained asystole. Sustained VT or VF was reported following 7 of 99 shocks in a study of patients undergoing electrophysiologic study and requiring cardioversion for VT, VF, or atrial fibrillation. These episodes occurred only in the patients with prior VT or VF. Patients with ischemia or known coronary artery disease appear to be at much higher risk for significant postshock bradycardia, with rate support pacing required after 13 of 99 shocks in the study. Asystole requiring pacing occurred only once in 99 countershocks. Therefore, the proclivity for dysrhythmias is greater in high-dose cardioversion of an ischemic heart.

Two types of VF following cardioversion have been described. The first variety occurs immediately after countershock and is easily reversed by a second, nonsynchronized shock. This type of VF results from improper synchronization, with discharge of current occurring during the vulnerable period. The second variety, which is more ominous, occurs approximately 30 seconds to a few minutes following attempted cardioversion. This dysrhythmia is characteristically preceded by the development of PAT with block or a junctional rhythm. In affected patients, it may be very difficult to convert the dysrhythmia to a sinus rhythm. This phenomenon occurs in patients who have been taking digitalis glycosides and is presumably a manifestation of digitalis toxicity.

In the event of VF following cardioversion, the equipment and manpower should be present for immediate defibrillation. If postcardioversion VF occurs, switch the cardioverter to "nonsynchronized" before attempting defibrillation. Electrical discharge will not occur in the "synchronized" mode, because the machine will be searching for a nonexistent R wave.

VF is much more likely to occur if depolarization occurs on the T wave. If a patient has large T waves in the lead selected for cardioverter sensing, the electric shock may discharge during the vulnerable period of the cardiac cycle, resulting in VF. Always examine the complexes on the cardioverter monitor carefully for large T waves and, if necessary, change the sensing lead. A randomly firing pacemaker can also be sensed by the cardioverter, resulting in countershock during the vulnerable period.

Transient and intermittent ST-segment elevation has also been reported to occur (though rarely) after cardioversion, with myocardial injury or coronary vasospasm offered as possible explanations.
An increase in serum enzyme levels (creatine kinase, lactate dehydrogenase, aspartate aminotransferase) may also occur following cardioversion, and the incidence has been reported to be between 10% and 70%. The enzyme rise is usually a consequence of skeletal muscle injury rather than myocardial damage. Cardioversion does not alter the enzyme profile of patients with myocardial infarction. [209]

CONCLUSIONS

Cardioversion is a safe and effective method of quickly terminating reentrant tachycardia. Complications related to psychological trauma, respiratory depression, and unintentional health-worker shock can be avoided with proper precautions. Adequate sedation is essential. Synchronized shock should be administered after close scrutiny of the lead used for sensing, to be sure that the R or S wave is significantly larger than the T wave. Be prepared for postshock VT or VF, and if VF occurs, switch the cardioverter to "nonsynchronized" and defibrillate. Atropine and temporary pacing equipment should be available to treat postshock bradycardia, especially in patients with myocardial ischemia or infarction.

Referencias