Implantable cardioverter defibrillators (ICDs) have achieved a preeminent role in the treatment of life-threatening ventricular arrhythmias. Because most ICD recipients have structural heart disease, they receive care from cardiologists who are not electrophysiologists but are increasingly involved in the follow-up of ICD patients. The most common arrhythmia-related complaint in ICD patients is “shocks.” This review describes the diagnosis and acute management of single, multiple, or repetitive shocks delivered by ventricular ICDs. It supplements comprehensive reviews of ICD troubleshooting. Management of shocks from atrial ICDs, which cardiovert atrial fibrillation with a slow ventricular rate, are beyond the scope of this review.

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Implantable cardioverter defibrillators deliver shocks in response to electrical signals that satisfy programmed criteria for detection of VT or VF. The first step in diagnosis of inappropriate shocks in patients with ICDs is to determine if the shock was delivered in response to a true tachyarrhythmia by inspecting data stored in the ICD. Shocks occur in the absence of tachyarrhythmias because nonarrhythmic physiologic or nonphysiologic signals are oversensed by the ICD and detected as arrhythmias. Diagnosis and causes of oversensing are reviewed. The second step in diagnosis is to determine if the tachyarrhythmia stored in the VT/VF episode log is VT/VF or SVT by analyzing stored electrograms. Frequent or repetitive shocks constitute an electrophysiologic emergency. The approach to this problem is reviewed.

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Key words: Implantable cardioverter defibrillator • Ventricular tachycardia • Inappropriate shocks
To deliver appropriate and effective therapy, an ICD must sense cardiac signals, detect tachyarrhythmias, and discriminate ventricular tachycardia (VT) from supraventricular tachycardia (SVT). Sensing is the process by which an ICD determines the timing of electrogram signals caused by cardiac depolarization. Detection is the algorithm by which an ICD processes a series of electrogram signals to classify the cardiac rhythm and determine if treatment should be delivered. The most basic detection algorithms measure and count RR intervals to detect any tachyarrhythmia that fulfills ventricular rate and duration criteria. Present detection algorithms include programmable SVT-VT discriminators (enhancements) that process either single-chamber ventricular information or both atrial and ventricular information to discriminate VT from SVT. Antitachycardia pacing or shocks must be withheld continuously for SVT and confounding nonarrhythmic signals but delivered without delay for life-threatening VT or ventricular fibrillation (VF).

**Basics of ICD Troubleshooting**

**Identifying the ICD.** Diagnosing the cause of shocks involves determining the ICD’s programming, evaluating stored data, and performing specific troubleshooting. These activities require identification of the ICD generator and electrodes from the patient’s identification card, medical alert bracelet, records from the implanting hospital or physician, or chest radiograph. ICD generators from different manufacturers have unique radiographic markers. Because all ICDs are registered at implantation, they can be identified by a phone call to the manufacturer’s 24-hour toll-free number (Table 1). Each manufacturer provides technical assistance 24 hours per day.

**ICD interrogation.** Basic interrogation using the ICD’s programmer can be performed without special training (Table 1). After the ICD is interrogated, the initial settings and all stored data must be documented by printing them or saving them electronically. These include bradycardia pacing parameters; rate, duration, and SVT-VT discrimination criteria used to detect VT and VF; programmed therapies for VT and VF; measurements of lead integrity; and data from each device-detected tachyarrhythmia episode.

**Interpretation of stored ICD-detected tachyarrhythmia episodes.** Stored electrograms of VT and VF episodes provide the most important data for interpreting the causes and results of ICD shocks. Physicians can interpret stored electrograms using skills learned from reading surface ECGs. Single-chamber ICDs store one or more ventricular electrograms; dual-chamber ICDs store atrial and one or more ventricular electrograms. ICDs use a closely spaced, near-field (“rate”) dipole for sensing. On transvenous leads, this dipole’s signal is recorded between the right ventricular tip electrode and either a sensing ring electrode or the right ventricular coil. A widely spaced, far-field (“shock”) dipole may be preferred for discrimination of
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VT from SVT by analysis of electrogram morphology. Marker channels indicate ICD classification of atrial and ventricular events and device-measured intervals (P-P and R-R).

Tachyarrhythmia detection and classification by ICDs have a significant error rate. Episodes stored as ICD-detected SVT or VT/VF may not represent a true arrhythmia. If an episode represents a true arrhythmia, the ICD may not accurately discriminate SVT from VT or VF. Thus, true SVT episodes may be stored in the ICD's VT or VF log; instances of true VTs may be stored in the ICD's SVT log. The physician must review all stored tachyarrhythmia episodes for accuracy of classification.

Diagnosing the Cause of Shocks

Figure 1 summarizes the approach to patients who present with ICD shocks. Rarely, a patient may complain of a shock but no shock is recorded in the ICD episode log. ICDs record delivered shocks so reliably that the absence of a recorded shock indicates that the patient’s symptom is not caused by ICD shock(s). The physician must evaluate the cause of “phantom shocks.” Patients may distinguish between “little” and “big” shocks. Patients almost never report ICD shocks as “little.” Little shocks may be caused by pacing-induced pectoral, diaphragmatic, or intercostal muscle stimulation.

Shocks in the absence of arrhythmias (oversensing). As indicated in Figure 1, the first step in diagnosing the cause of shocks is to determine if a tachyarrhythmia occurred. Because ICDs utilize feedback mechanisms (automatic gain control or auto-adjusting sensitivity) to ensure reliable sensing of low- and variable-amplitude ventricular electrograms during VF, they may sense electrical signals they were not

![Diagram of Approach to Patients with Shocks](image)

**Figure 1. Approach to the patient with shocks:** Top panel: flow diagram for one or infrequent shocks. Bottom panel: diagram for multiple or repetitive shocks.

- **Intracardiac signals**
  - Physiologic
    - T wave (low-amplitude R wave, prolonged QT)
    - P wave (consider ventricular lead dislodgment)
    - Double-counted R waves
  - Nonphysiologic
    - Separate electronic pacemaker
    - Mechanical contact of sensing lead and abandoned lead fragment

- **Extracardiac signals**
  - Physiologic (myopotentials)
    - Lead insulation failure
    - Diaphragmatic myopotentials (bradycardia, positional)
  - Nonphysiologic
    - Signals originating in ICD system
      - “Chatter” in active fixation lead
      - Lead fracture
      - Loose set screw or adapter
  - Electromagnetic interference
    - Surgical electrocautery
    - Electronic article surveillance equipment
    - Magnetic resonance imaging
    - Lithotripsy

Table 2. Major Causes of inappropriate Shocks Due to Oversensing
Shocks occur in the absence of tachyarrhythmias because nonarrhythmic physiologic or non-physiologic signals are oversensed by the ICD and detected as arrhythmias. Common causes of oversensing that results in shocks are summarized in Table 2. Clinical history is of limited value in diagnosing oversensing except for specific identifiable causes of external electromagnetic interference. The presence of antecedent arrhythmic symptoms—palpitations, syncope, or near syncope—suggests a true arrhythmia, but most shocks, appropriate or inappropriate, are not preceded by symptoms. A pattern of consistent postural changes or muscle activity preceding shocks suggests electrode-related oversensing. Audible tones from the ICD may be an alarm that indicates a loss of electrical integrity in a lead.

**Physiologic intracardiac signals.** Patients with low-amplitude R waves may require higher sensitivity settings to ensure reliable sensing of VF. This may result in T-wave oversensing, which can cause inappropriate inhibition of bradycardia pacing, antitachycardia pacing at the wrong rate, or inappropriate detection of VT (Figure 2). P-wave oversensing can occur if the ventricular sensing dipole is near the tricuspid valve. Except in the case of lead dislodgment, this is rare. R-wave double sensing occurs if the duration of the sensing electrogram exceeds the ventricular refractory period of 120-140 ms. It may be exacerbated by sodium-channel-blocking antiarrhythmic drugs, particularly at high heart rates as use-dependent sodium-channel blockade increases.

In some cases, ICDs may oversense intracardiac electronic signals from independent, implanted pacemakers. Pacemaker-ICD interactions have been described comprehensively.

Oversensing of intracardiac electrograms can be recognized by characteristic alternation of intervals and electrogram morphology (P-R vs R-P, T-Q vs Q-T, and short R-R' vs long R'-R separated by isoelectric baselines. In contrast, the hallmark of oversensing of nonphysiologic signals or extracardiac physiologic signals (myopotentials) is the replacement of the isoelectric baseline with high-frequency noise (Figure 3). Oversensing of physiologic cardiac signals may be detected inappropriately as VT or VF, but nonphysiologic signals are often oversensed as R-R intervals close to the ICD’s refractory period (120-140 ms) and are almost always detected as VF.

**Myopotential oversensing.** Sensing lead insulation failures in the pocket are a common cause of inappropriate oversensing of pectoral or abdominal myopotential signals. A low pacing lead impedance (<200 Ω) indicates the presence of a parallel conductor pathway and supports the diagnosis of insulation failure. The risk of lead failure depends on the lead type and the pulse generator location. Failure is common in epicardial leads and transvenous leads with abdominally implanted generators. With pectorally implanted generators, failure is more common in coaxial leads than in multilumen leads. Oversensing of diaphragmatic myopotentials may occur in ICD systems with integrated bipolar electrodes in the right ventricular apex. It may be bradycardia dependent or positional and may cause both inappropriate inhibition of bradycardia pacing and inappropriate detection of VT/VF.

**Extracardiac nonphysiologic signals.** A lead fracture, loose set screw, or adapter malfunction may result in oversensing of nonphysiologic signals. A high pacing lead impedance (>2000 Ω) indicates complete or partial interruption of the electrical circuit and supports this diagnosis.
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Except for medical electrocautery, external sources of electromagnetic interference are rare causes of oversensing. They have been summarized in recent reviews. Diagnosis of suspected oversensing may be confirmed by real-time telemetry of electrograms and marker channels. Postural changes and deep breathing may be necessary to confirm oversensing of diaphragmatic myopotentials. Postural changes, isometric exercises, and pocket manipulation may facilitate diagnosis of lead or connector-related oversensing.

ICD algorithms for discrimination of SVT from VT. If stored electrograms indicate that a shock was delivered in response to a true tachyarrhythmia, the second step in diagnosis is to determine if the rhythm stored in the VT or VF episode log is VT or SVT. Clinical history is of limited value. A history of rapidly conducted atrial
Implantable Cardioverter Defibrillator Shocks

Continued

fibrillation suggests inappropriate therapy for atrial fibrillation. A shock during vigorous exercise suggests inappropriate therapy for sinus tachycardia. Inappropriate shocks for SVT do not occur in pacemaker-dependent patients.

Three single-chamber SVT-VT discriminators have been implemented in ICDs13-19: stability, which discriminates monomorphic VT from atrial fibrillation based on regularity of the R-R interval; onset, which discriminates VT from sinus tachycardia based on suddenness of onset, enabling therapy to be withheld from tachycardias in which the rate increases gradually; and morphology, which discriminates VT from any SVT based on morphologic differences between electrograms in sinus rhythm and tachycardia. Dual-chamber algorithms may use atrial and ventricular rates, chamber of origin, P:R pattern, and atrioventricular association (P-R interval stability) in addition to the single-chamber criteria.

Interpreting the specific actions of multiple, complex detection enhancements may be daunting. Fortunately, this is largely unnecessary for acute evaluation of patients who present with shocks. Thus, the nonelectrophysiologist should focus on applying established principles of surface ECG analysis to intracardiac electrograms of ICD-detected VT or VF. The diagrams in Figure 4 show methods for analyzing single-chamber and dual-chamber electrograms, as discussed below.

Physician analysis of single-chamber data for SVT-VT discrimination. Morphology. In discriminating SVT from VT using the surface ECG, the first step is to

Figure 4. SVT-VT discrimination by analysis of stored EGMs. **Top panel:** method for single-chamber stored EGMs. **Bottom panel:** method for dual-chamber stored EGMs.

Figure 5. Rate-related bundle branch block in atrial tachycardia. Dual-chamber EGMs show abrupt onset of regular tachycardia in the atrium with 1:1 atrioventricular conduction. The ventricular EGM morphology in SVT is distinct from the morphology during sinus rhythm. If only a single-chamber ventricular EGM were available, the rhythm would be misdiagnosed as VT based on different ventricular EGM morphology, abrupt onset, and regular rhythm. Atip-Aring = atrial EGM; RV Tip-Coil, right ventricular integrated bipolar sensing EGM.
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determine if the QRS morphology is ventricular or supraventricular. Analogously, analysis of ventricular electrogram morphology is the cornerstone of SVT-VT discrimination based on visual inspection of stored episodes in single-chamber ICDs. Whenever possible, electrogram morphology should be analyzed from the signals of far-field dipoles. Visual inspection does not permit unequivocal discrimination of VT from sinus morphologies from near-field electrograms in 5% to 10% of VTs. A real-time reference sinus electrogram should be recorded with the patient in the same posture in which the episode occurred.

Tachyarrhythmias are classified as SVT if the electrogram morphology is uniform and identical to the sinus morphology. Tachyarrhythmias are classified as VT if the electrogram morphology is uniform and distinctly different from the sinus morphology. This approach necessarily classifies rate-related bundle branch block during SVT as VT (Figure 5).

Onset. If electrogram morphology is variable or differs only minimally from sinus morphology, ancillary criteria must be applied: abruptness of onset, R-R interval stability, and response to therapy. Sinus tachycardia has a gradual onset and is usually detected at the sinus-VT rate boundary (Figure 6). In contrast, the onset of VT is usually abrupt. Exceptions include VT that originates during sinus tachycardia or other SVTs and VT that starts abruptly but at an initial rate below the programmed VT detection rate. In the latter case, the ICD will store the “onset” of the arrhythmia as the gradual acceleration across the sinus-VT rate boundary.

Stability. An irregularly irregular rhythm is characteristic of atrial fibrillation. Subtle beat-to-beat variation in electrogram morphology, the intracardiac correlate of rate-related aberrancy, is common in rapidly conducted atrial fibrillation. Atrial fibrillation is usually detected inappropriately during an ongoing atrial fibrillation episode when the ventricular rate exceeds the programmed rate criterion. Thus, stored intervals are irregularly irregular prior to and during detection. However, interval stability has 2 important limitations as a criterion: (1) Because the ventricular rate in atrial fibrillation is more regular at faster ventricular rates, stability cannot reliably discriminate atrial fibrillation from VT at rates above 170/min. (2) Amiodarone or type IC antiarrhythmic drugs may cause monomorphic VT to become markedly irregular or polymorphic VT to slow, causing underdetection of VT.

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Response to therapy. Termination of a tachyarrhythmia by a single trial of ventricular antitachycardia pacing favors a diagnosis of VT. This is not helpful in the analysis of shocks, but termination of multiple other episodes with similar morphology and rate by a single trial of anti-tachycardia pacing favors the conclusion that shocks are due to VT. The vast majority of VTs are terminated by 1 or 2 shocks. Thus, failure of multiple shocks to terminate a regular tachycardia suggests VT, particularly sinus tachycardia.

Physician analysis of dual-chamber data for SVT-VT discrimination. Discrimination of VT from SVT by analysis of the surface ECG is often hampered by uncertainty regarding the atrial rhythm diagnosis. Dual-chamber electrograms permit accurate diagnosis of the atrial rhythm if the atrial lead is functioning. Analyses of atrial and ventricular rates and atrioventricular relationships are the cornerstones of dual-chamber SVT-VT discrimination. If the ventricular rate is faster than the atrial rate in an ICD patient, VT should be diagnosed.

1:1 A/V relationship. If the atrial and ventricular rates are equal,
the physician must discriminate between VT with 1:1 ventriculoatrial conduction and SVT with 1:1 atrioventricular conduction. Sinus morphology strongly favors a diagnosis of SVT. Sinus tachycardia has a characteristic gradual atrial acceleration, usually with a stable PR interval. Analysis of the chamber of acceleration (where acceleration originates) may allow discrimination of atrial tachycardia from VT: Atrial tachycardia begins with a short P-P interval followed by a short R-R interval, whereas VT usually begins with a short R-R interval and a few beats of atrioventricular dissociation until 1:1 ventriculoatrial conduction stabilizes (Figure 7). Atrial electrogram morphology in sinus rhythm differs from the morphology of retrograde atrial electrograms in VT and from that of ectopic atrial electrograms during atrial tachycardia; however, these differences may be subtle, and their absence should not be considered as confirmatory of sinus P waves.

VT during SVT. If the atrial rate exceeds the ventricular rate, the physician must distinguish between conducted atrial fibrillation, or atrial flutter (Figure 8) and VT during atrial
rapid atrial electrograms during atrial fibrillation and atrial flutter are easily identified by the physician but may be underdetected by the ICD due to atrial blanking periods. However, the physician must not misinterpret far-field R waves on the atrial channel as atrial electrograms (Figure 9). Such misinterpretation is a possible cause of incorrect classification of VT as SVT by dual-chamber ICDs. In stored electrograms, abnormal ventricular morphology and regular ventricular rate are the best diagnostic criteria for VT during atrial fibrillation (Figure 10). Conducted atrial flutter may be diagnosed in the presence of abnormal ventricular morphology if consistent 2:1 atrioventricular association or Mobitz 1 atrioventricular block is present.

Even when all criteria for VT-VT discrimination have been applied, experts are unable to diagnose some arrhythmias with certainty. It is better to consider the diagnosis uncertain than to make important therapeutic decisions based on unreliable data.

**Clinical Approach to the Patient with Shocks**

**Single or infrequent shocks.** Single shocks or a few shocks separated by days do not constitute an emergency. The ICD should be interrogated within 24 to 48 hr, and the logic in Figure 1 should be applied.

If oversensing is caused by external electromagnetic interference, the treatment is to remove the offending source. Oversensing of physiologic signals may require reprogramming, lead revision, or insertion of a new sensing lead.

Inappropriate therapy of SVT may be corrected by reprogramming rate zones or SVT-VT discriminators.

### Table 3: Drug Effects in Patient with ICD Shocks

<table>
<thead>
<tr>
<th>Frequency of VT/VF</th>
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<tr>
<td>Proarrhythmic²⁶,²⁷</td>
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<tr>
<td>Antiarrhythmic drugs</td>
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<tr>
<td>Drugs with proarrhythmic side effects</td>
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<tr>
<td>Drugs that interact with proarrhythmic drugs</td>
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<tr>
<td>Antiarrhythmic</td>
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<tr>
<th>Detection of VT/VF</th>
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<tr>
<td>Programmed Rate Boundaries</td>
</tr>
<tr>
<td>VT rate (decrease–IC, amiodarone)</td>
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<tr>
<td>SVT ventricular rate</td>
</tr>
<tr>
<td>Decrease (beta blockers, amiodarone, sotalol)</td>
</tr>
<tr>
<td>Increase (1:1 conduction of atrial flutter on IC drugs)</td>
</tr>
<tr>
<td>ICD VT-VT discrimination algorithms</td>
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<tr>
<td>VT interval stability (more irregular on IC, amiodarone)</td>
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<tr>
<td>Electrogram morphology</td>
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<tr>
<th>Therapy for VT/VF</th>
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<tbody>
<tr>
<td>Defibrillation energy requirement</td>
</tr>
<tr>
<td>Increase (IA, IB, IC, amiodarone)</td>
</tr>
<tr>
<td>Decrease (III except amiodarone)</td>
</tr>
<tr>
<td>Antitachycardia pacing</td>
</tr>
<tr>
<td>Efficacy: variable</td>
</tr>
<tr>
<td>Use-dependent increase in pacing threshold (IC)</td>
</tr>
<tr>
<td>Heart failure exacerbation</td>
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<tr>
<td>(disopyramide, beta blockers, sotalol, IC, calcium antagonists)</td>
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### Table 4: Causes of Repetitive Sustained VT (“Storm”)

<table>
<thead>
<tr>
<th>Acute ischemia</th>
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<tr>
<td>Exacerbation of heart failure</td>
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<tr>
<td>Metabolic abnormalities</td>
</tr>
<tr>
<td>Hypokalemia</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
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<tr>
<td>Hyperthyroldism (amiodarone-induced)</td>
</tr>
<tr>
<td>Drug</td>
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<tr>
<td>Proarrhythmia</td>
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<tr>
<td>Change in antiarrhythmic drug</td>
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<tr>
<td>Change in prescribed drug</td>
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<tr>
<td>Change in preparation of prescribed drug</td>
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<tr>
<td>Noncompliance</td>
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</tbody>
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*Implantable Cardioverter Defibrillator Shocks continued*
Alternatively, SVT may be treated with drugs or ablation. Reprogramming is usually preferable for sinus atrial tachycardia, whereas pathologic arrhythmias may require both approaches. The physician should consider the effect of reprogramming and drugs on detection of VT and on drugs efficacy of VT therapy. Comprehensive reviews of drug-ICD interactions have been published. Relevant points are summarized in Table 3.

Single or infrequent appropriate shocks for VT or VF do not require a change in therapy. Fast monomorphic VT with rates up to 240/min may be terminated by painless antitachycardia pacing without increasing the risk of syncope. Thus, one attempt at antitachycardia pacing should be programmed to improve patient acceptance of ICD therapy. If antitachycardia pacing is unreliable, an antiarrhythmic drug may be required to decrease the frequency of VT or permit successful antitachycardia pacing. Drug-ICD interactions should be considered (Table 3).

Frequent or repetitive shocks. More than 3 shocks in 1 day or repetitive shocks constitute an electrophysiologic emergency (Figure 1). These may cause release of troponin I or severe psychological complications. However, the logical approach is similar to that for infrequent shocks. The diagnosis of repetitive shocks caused by oversensing is clear when the patient receives shocks during electrocardiographically documented sinus rhythm. Emergency treatment consists of suspending or disabling VT/VF detection by placing a magnet over the ICD. Tachyarrhythmia detection and/or therapy should then be turned off with a programmer. The specific steps are summarized in Table 1. The specific cause of oversensing can be determined by analysis of real-time electrograms with therapies programmed off. Once the ICD is deactivated, the patient will not receive appropriate ICD therapy if VT or VF occurs; external shock will be required.

Repetitive shocks caused by inappropriate detection of SVT can also be addressed on an emergent basis by suspending detection with a magnet. The VT detection rate can then be programmed to be above the SVT rate. The definitive approach is the same as that for single inappropriate shocks for SVT.

Repetitive shocks for VT may be caused by recurring episodes of VT after successful shock termination of VT (‘VT storm’/’cluster’ shocks) or by multiple unsuccessful shocks for a single episode. Therapeutic approaches differ.

VT storm. Known causes are summarized in Table 4. Frequently, the cause is unknown. Diagnosis of acute coronary syndromes during VT storm is problematic, since multiple (appropriate or inappropriate) shocks can cause repolarization changes as well as elevations in troponin I up to 15 ng/mL. Therapy may include reversing the precipitating cause, antiarrhythmic drugs (usually amiodarone, sotalol, and/or a beta blocker), and catheter ablation.

Rarely, ICDs deliver unnecessary repetitive shocks for recurring self-terminating episodes of VT. Shocks may be prevented in such cases by programming the ICD to a noncommitted mode or increasing the number of beats required for detection of VT or VF.
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Failed shocks. Because defibrillation is a statistical process, occasional shock failures occur randomly, but failure of ≥2 maximum-output shocks should not occur. Shocks from chronically implanted ICD systems may fail to terminate VT or VF because of patient-related or ICD system-related reasons. Pneumothorax and pleural effusion decrease transmyocardial current, the former by adding intrathoracic resistance and the latter by providing a low-resistance parallel current path.

Misclassification of shock efficacy. ICD shocks may terminate VT, but the ICD may not detect the termination, resulting in incorrect classification of the shocks as unsuccessful. This happens if VT recurs before the ICD can detect post-shock sinus rhythm because of immediate post-shock recurrence of nonsustained or sustained VT. The latter should be treated as repetitive sustained VT. Misclassification of both may be corrected by decreasing the number of beats for detection of sinus rhythm; misclassification of the former may be corrected by increasing the number of beats for detection of VT.

ICDs may also misclassify shocks as ineffective if the post-shock rhythm is SVT in the VT rate zone (catecholamine-induced sinus tachycardia or shock-induced atrial fibrillation). This should be treated as inappropriate detection of VT. It may be corrected by applying SVT-VT discrimination algorithms to detection of VT, increasing the VT detection rate to above the rate of post-shock SVT, or increasing shock strength to prevent shock-induced atrial fibrillation. Pharmacologic approaches include beta blockers to slow post-shock SVTs.

References