Amiodarone Is Poorly Effective for the Acute Termination of Ventricular Tachycardia

Keith A. Marill, MD
Ian S. deSouza, MD
Daniel K. Nishijima, MD
Thomas O. Stair, MD
Gary S. Setnik, MD
Jeremy N. Ruskin, MD

From the Massachusetts General Hospital, Boston, MA (Marill, Ruskin); State University of New York, Downstate Medical Center, Brooklyn, NY (deSouza, Nishijima); Brigham and Women's Hospital, Boston, MA (Stair); and Mount Auburn Hospital, Cambridge, MA (Setnik).

Study objective: It is hypothesized that intravenous (IV) amiodarone is poorly effective for the acute termination of sustained monomorphic ventricular tachycardia because of the relatively slow onset of its Vaughn-Williams class III effect to prolong myocardial depolarization and the refractory period. This study is designed to determine the effectiveness and safety of IV amiodarone for the termination of sustained monomorphic ventricular tachycardia.

Methods: A retrospective case series was collected at 4 urban university-affiliated hospitals from September 1996 to April 2005 after institutional review board approval with waiver of informed consent. Emergency department (ED) patients treated with IV amiodarone for ventricular tachycardia were identified by ED treatment and hospital pharmacy billing records, *International Classification of Diseases, Ninth Revision* discharge codes, and ECG characteristics. All consecutive patients who received at least 150 mg amiodarone in 15 minutes or less for spontaneous sustained monomorphic ventricular tachycardia were eligible for inclusion. Sustained monomorphic ventricular tachycardia was defined as a tachycardia with uninterrupted duration or rapid recurrence despite automatic internal cardiac defibrillator therapy for at least 5 minutes before amiodarone treatment, monomorphic morphology, rate greater than 120 beats/min, QRS duration greater than 120 ms, and subsequently determined to be ventricular tachycardia by ECG criteria (eg, atrioventricular dissociation), implanted device interrogation, or formal electrophysiology study. Measured outcomes included sustained termination of ventricular tachycardia within 20 minutes of initiation of amiodarone infusion and any documented adverse effects. Rates of successful termination and adverse effects and their 95% confidence intervals (CIs) were calculated. The presence or average values of potentially confounding predictors in patients with and without ventricular tachycardia termination after amiodarone were also calculated and compared.

Results: Thirty-three patients were identified and included. Five patients received electrical therapy within 20 minutes of initiation of amiodarone infusion, and the response to amiodarone was unknown. Twenty-seven of the remaining 28 patients received 150 mg amiodarone, and the rate of successful ventricular tachycardia termination was 8 of 28, 29% (95% CI 13 to 49). Two of 33 patients, 6% (95% CI 1 to 20), required direct current cardioversion for presyncope or hypotension temporally associated with amiodarone treatment.

Conclusion: IV amiodarone, as currently administered, is relatively safe but ineffective for the acute termination of sustained ventricular tachycardia. [Ann Emerg Med. 2006;47:217-224.]

0196-0644/$-see front matter

doi:10.1016/j.annemergmed.2005.08.022

SEE EDITORIAL, P. 227.

INTRODUCTION

Stable ventricular tachycardia is an uncommon but dangerous medical condition because of the risk of hemodynamic deterioration. The most effective treatment is immediate direct current cardioversion[1]; however, this procedure is painful and requires sedation. The American Heart Association recommends that patients with stable ventricular tachycardia may be treated medically with intravenous (IV)

Editor's Capsule Summary

What is already known on this topic

The advanced cardiac life support guidelines preferentially recommend amiodarone for termination of "stable" ventricular tachycardia.

What question this study addressed

This retrospective study addressed the percentage of patients with stable monomorphic ventricular tachycardia who convert within 20 minutes after a single dose of amiodarone.

What this study adds to our knowledge

Despite being a first-line recommendation of the advanced cardiac life support guidelines, amiodarone converted only a minority of the 28 patients with stable monomorphic ventricular tachycardia.

How this might change clinical practice

Knowledge of the poor response to amiodarone may result in increased use of other agents or electrical cardioversion without a prolonged trial of pharmacologic management.

amiodarone or lidocaine or, if the ejection fraction is estimated to be greater than 40%, procainamide or sotalol.[2]

There is a large body of indirect evidence suggesting that IV amiodarone may be useful to terminate ventricular tachycardia. The administration of IV amiodarone to suppress recurrent destabilizing ventricular tachycardia has been extensively studied.[3-11] It has generally been found to suppress ventricular tachycardia effectively during prolonged IV infusion for hours or days, often in patients with severe myocardial disease and after multiple other antidysrhythmics have failed.

There is limited direct evidence supporting the use of IV amiodarone for the rapid termination of sustained stable ventricular tachycardia. After administration of a 5 mg/kg bolus during 20 minutes, followed by a continuous infusion, Schutzenberger et al[12] noted termination of 8 episodes of ventricular tachycardia in 19 patients (42%; 95% confidence interval [CI] 20 to 67) within 31 minutes. Benaim and Uzan[13] primarily used a dosing regimen of 300 mg infused during 30 seconds and found termination of ventricular tachycardia in 6 of 12 cases (50%; 95% CI 21 to 79). In combination with continued electrical therapy, a new aqueous formulation of amiodarone has also been associated with a high rate of ventricular tachycardia termination in patients initially refractory to electroshock.[14]

There are reasons to believe, however, that amiodarone may be neither effective nor safe for the treatment of sustained monomorphic ventricular tachycardia. Amiodarone is often classified primarily as a Vaughan-Williams class III antidysrhythmic, and when administered chronically, it lengthens the duration of repolarization (QT interval corrected for pulse rate) and refractory period in most cardiac tissue.[15,16] It is the lengthening of the refractory period by antidysrhythmics that is thought to be responsible for breaking the reentrant conduction pathway present in the majority of monomorphic ventricular tachycardia episodes and terminating the tachydysrhythmia. Episodic IV amiodarone administration differs in that it causes little prolongation of myocardial repolarization and the effective refractory period in ventricular myocardial tissue.[17,18] Hemodynamically, IV amiodarone usually causes a decrease in systemic vascular resistance with coronary and peripheral vasodilatation and variable depressant effects on cardiac contractility.[19-22] This report details our observations of the efficacy and safety of IV amiodarone in terminating sustained monomorphic ventricular tachycardia.

MATERIALS AND METHODS

Study Design

This preliminary report describes a retrospective case series of consecutive patients with sustained monomorphic ventricular tachycardia treated with IV amiodarone. The case series is part of a larger multicenter cohort study in progress comparing the safety and efficacy of IV amiodarone and procainamide.

Study Design and Setting

This was a multicenter study at 4 urban hospitals in 2 cities. Patients were enrolled from the emergency departments (EDs) of all 4 facilities (Table 1). Patients treated by emergency medical services en route to the ED were included. Expedited institutional review board approval with waiver of informed consent was obtained from all participating institutions.

Selection of Participants

All cases in which a patient presented with wide QRS complex tachycardia that was stable and sustained or recurrent, requiring multiple antitachycardia pacing or shocks from an implanted automatic internal cardiac defibrillator, and received at least 150 mg amiodarone IV infusion during 15 minutes or fewer were eligible for inclusion. Wide QRS complex tachycardia was defined as a regular heart rhythm with rate greater than or equal to 120 beats/min and QRS duration greater than or equal to 120 ms. The rhythm was considered sustained if it existed either as an uninterrupted rhythm or as a rapidly recurring rhythm after automatic internal cardiac defibrillator treatment for at least 5 minutes before amiodarone infusion. The rhythm was considered stable if the treating physician elected to use pharmacologic therapy as the initial treatment. The wide complex tachycardia was diagnosed as ventricular tachycardia and the case was included based on criteria defined in the measurements section. Patients excluded from the study included those with polymorphic ventricular tachycardia morphology and all inpatients, including those receiving pressor infusions or in the surgical ICU, recovering from open heart surgery.

Table 1. Enrollment information.

Hospital	ED Volume	Beginning Search Date	Ending Search Date	Search Criteria	Number of Patients Enrolled
Massachusetts General Hospital	75,000	1/1/1998	10/31/2003	Hospital admission or primary discharge diagnosis ''paroxysmal ventricular tachycardia,'' *ICD-9* 427.1, with or without stored ECG with wide QRS complex tachycardia	2
		10/1/2001	9/16/2004	Pharmacy charge for IV amiodarone or primary diagnosis ventricular tachycardia in the ED	13
Brigham and Women's Hospital	55,000	5/1/2000	9/16/2004	Pharmacy charge for IV amiodarone or primary diagnosis ventricular tachycardia in the ED	4
Mount Auburn Hospital	30,000	9/1/1996	4/30/2005	Digital ED physician record search for ''amio,'' ''vent tach,'' or ''VT''	10
SUNY Downstate Medical Center	45,000	7/1/1999	2/28/2005	Principal hospital discharge diagnosis code ''paroxysmal ventricular tachycardia,'' *ICD-9* 427.1	4

ICD-9, International Classification of Diseases, Ninth Revision.

Patients were identified for enrollment using computerized searches based on the databases available at each facility (Table 1). This search included pharmacy records of all patients billed for IV amiodarone, text search of the treatment section of digital emergency physician records for the terms ''amio,'' ''vent tach,'' or ''VT,'' patients with the diagnosis code ''paroxysmal ventricular tachycardia,'' (*International Classification of Diseases, Ninth Revision* code 427.1), and patients with a wide QRS complex tachycardic ECG stored in the hospital ECG database (GE Marquette Muse System, Milwaukee, WI). When available, multiple modalities were used to search for patients to maximize capture of all potential cases. Each patient was enrolled only once using the first presentation that satisfied the enrollment criteria.

Methods of Measurement

Three unblinded investigators, including 2 emergency medicine–trained attending physicians and 1 emergency medicine resident, used a standardized abstraction form to collect data. Each abstractor was trained to use the form, and the principal investigator reviewed all of the forms of the other 2 abstractors in periodic meetings. For variables noted multiple times on the chart, the first documented value was recorded. In particular, the medical history and physical examination findings documented by the emergency physician were recorded whenever available. Information not available on the record was documented as missing. Study data were collected from each record by a single abstractor, and there was no measure of interrater reliability.

Patients' demographics, including age and sex, were recorded. Hospital and hospital ward or unit were noted. History of cardiac disease, including congestive heart failure, coronary artery disease, myocardial infarction, and ventricular tachycardia or other dysrhythmias, was obtained from the recorded medical history and physical examinations performed by the treating physicians. Left ventricular ejection fracture was recorded from cardiac imaging studies performed most closely temporally to the wide QRS complex tachycardia episode. Necessary supporting evidence to confirm a history of myocardial infarction included Q waves on the ECG, localized wall motion abnormalities on a cardiac imaging study, or documentation of the myocardial infarction event in the clinical record. Medications at presentation, including antidysrhythmics and digoxin, were recorded.

The characteristics of the wide QRS complex tachycardia were recorded in detail, which included the duration of the dysrhythmia before treatment, if known, and the heart rate and blood pressure before, during, and after treatment. The dose, duration, and timing of any treatment before or after amiodarone infusion were recorded. Symptoms of chest pain or shortness of breath and physical signs, including pulmonary rales and peripheral edema, were noted. The heart rate, QRS duration, and QT interval corrected for heart rate were recorded from automated measurements of the wide QRS complex tachycardia ECG, with manual correction for obvious errors. If multiple wide QRS complex ECGs were available, data were recorded from the tracing taken closest to the amiodarone infusion. Measured patient weight was noted, as well as the serum potassium, magnesium, and calcium levels.

Response to treatment was evaluated as follows: termination was defined as conversion to the patient's known or presumed usual heart rhythm (eg, sinus, atrial fibrillation) within 20 minutes of initiation of amiodarone infusion. If the dysrhythmia reoccurred within 5 minutes of termination, then the treatment was considered unsuccessful. If a different antidysrhythmic medicine or direct current cardioversion was administered before dysrhythmia termination but within 20 minutes of initiation of amiodarone infusion, then the response to amiodarone was considered unknown. Magnesium infusion was recorded, but it was not considered an antidysrhythmic for this purpose because it has not been

shown to terminate sustained monomorphic ventricular tachycardia. If multiple boluses of amiodarone were administered, then these were summed so that the total dose during the total infusion period, up to 20 minutes, was considered.

A diagnosis of ventricular tachycardia as the underlying dysrhythmia was made based on 1 of 4 lines of evidence: all patients with ECG evidence of atrioventricular dissociation during the presenting tachycardia; all patients with ventricular tachycardia based on interrogation of an implanted automatic internal cardiac defibrillator; all patients with inducible ventricular tachycardia in the electrophysiology laboratory, with morphology similar to the presenting tachydysrhythmia; and all patients with a history and presenting ECG suggestive of ventricular tachycardia by established criteria,[23,24] as determined by an electrophysiologist blinded to treatment outcome. Episodes of wide QRS complex tachycardia that met none of these 4 criteria were considered supraventricular in origin and were excluded.

All apparent adverse effects of amiodarone treatment, including hypotension, bradycardia, new dysrhythmias including torsades de pointes, and other noncardiovascular effects, were tabulated. The hospital course after the dysrhythmia was reviewed. The criteria for diagnosis of a concurrent acute myocardial infarction included an abnormal elevation of the absolute creatine kinase, myocardial bound, fraction or troponin level above the normal limit for the respective laboratory tests in serum drawn within 24 hours of the dysrhythmia and a clinical diagnosis of acute myocardial infarction by the cardiology team consulted during the hospitalization after the event.

Outcomes Measures

The primary outcome was termination of ventricular tachycardia within 20 minutes of onset of amiodarone infusion. Secondary outcomes included termination of ventricular tachycardia at any time after amiodarone infusion but before infusion of another antidysrhythmic or direct current cardioversion and any adverse effects associated with amiodarone treatment.

Primary Data Analysis

The primary outcome was reported with 95% CI calculated using Stat Xact 3 software (Cytel Software, Cambridge, MA). Differences and associated 95% CIs of predictor variables between response groups were computed using Stat Xact 3 for dichotomous and SPSS 13 (SPSS, Inc., Chicago, IL) for interval data. No adjustments were made for multiple comparisons.

Based on our assumption of the ventricular tachycardia termination rate with amiodarone before initiation of the study, the approximate sample size needed to obtain a 95% CI whose upper bound would not cross 50% was estimated. Assuming that the ventricular tachycardia termination rate with a single 150-mg bolus of amiodarone was 30%, then if 50 patients with ventricular tachycardia were identified, the 95% CI would have a range of 18% to 45%.

Table 2. Methods for diagnosing ventricular tachycardia.

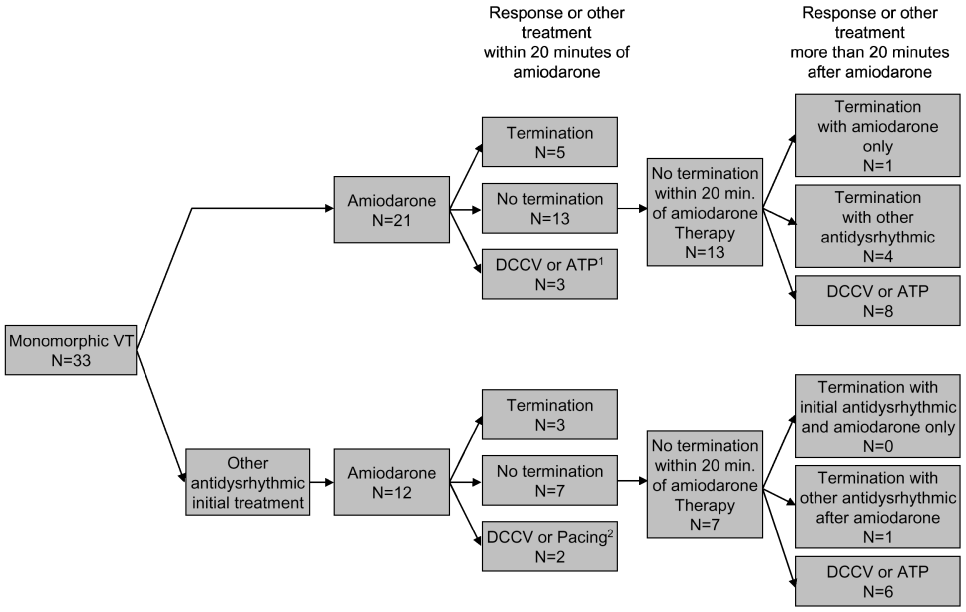
Diagnostic Method	Number of Patients (%)
Atrioventricular dissociation on ECG	4 (12)
Variable retrograde ventricular-atrial conduction on ECG	2 (6)
Fusion beats	1 (3)
Other ECG criteria	6 (18)
Implanted device interrogation	7 (21)
Tachydysrhythmia reproduced in the electrophysiology laboratory	13 (39)

RESULTS

Thirty-three patients who presented between September 1996 and April 2005 were enrolled. Details for each facility of the search dates and search criteria and the number of cases enrolled are presented in Table 1. Patients were diagnosed with ventricular tachycardia using the methods listed in Table 2. One patient was diagnosed with arrhythmogenic right ventricular dysplasia. Thirty-one patients received 150 mg and 2 patients received 300 mg of amiodarone. Of the 2 patients who received 300 mg, one did not experience ventricular tachycardia termination within 20 minutes, and the other had an unknown response because emergency direct current cardioversion was required within 10 minutes of initiation of amiodarone infusion.

The flow of patients through the study as a function of therapy and therapeutic response is depicted in the Figure. Five patients were treated with a second intervention within 20 minutes (average 11 minutes) of initiation of amiodarone infusion, so the response to amiodarone was unknown. Four of these 5 patients received direct current cardioversion, including 1 emergently for hypotension, and 1 patient received pacing for asystole. For the remaining 28 patients, the rate of successful ventricular tachycardia termination was 8 of 28, 29% (95% CI 13 to 49). Successful termination after amiodarone infusion was not associated with the presence, absence, or average value of any measured potential predictor variables, except that systolic and diastolic blood pressure were significantly lower in patients who experienced termination with amiodarone (Table 3).

There were 4 patients with adverse effects that occurred after amiodarone infusion. An 80-year-old woman received a total of 150 mg of lidocaine and then 150 mg of amiodarone infused during 15 minutes. The patient was noted to have "decreased blood pressure" after this, with no specific value documented, but the patient remained "alert and oriented." She received nonemergent successful direct current cardioversion 20 minutes after amiodarone infusion was initiated. An 88-year-old woman with an automatic internal cardiac defibrillator arrived in the ED with persistent recurrent



1) DCCV = Direct current cardioversion, ATP = antitachycardia pacing, VT = Ventricular tachycardia
2) One patient developed asystole and received pacing from an implanted automated internal cardiac defibrillator

Figure. Patient flow algorithm.

ventricular tachycardia. She received 2 100-mg lidocaine boluses; then 30 minutes later, she received 150 mg amiodarone, followed by an infusion at 1 mg per minute. Within 8 minutes of amiodarone infusion, she went into asystole, with an associated loss of blood pressure. The patient was emergently intubated, and her automatic internal cardiac defibrillator began pacing at 40 beats/min. The pacing rate was adjusted upward shortly thereafter, and ventricular tachycardia did not recur.

Two patients of 33, 6% (95% CI 1 to 20) required direct current cardioversion because of presyncope after amiodarone infusion. A 50-year-old man with initial blood pressure of 112/67 mm Hg and pulse rate of 234 beats/min developed diaphoresis and presyncope, with a blood pressure of 97/71 mm Hg and pulse rate of 193 beats/min 10 minutes after infusion of 150 mg of amiodarone. He was cardioverted 45 minutes after amiodarone infusion because of continued symptoms. A 65-year-old woman with blood pressure 120/85 mm Hg and pulse rate 176 beats/min received 2 boluses of 150 mg amiodarone slow IV push 5 minutes apart. Within 10 minutes of initiation of the first infusion, the patient was presyncopal, with a pulse rate of 170 beats/min on the monitor and a palpable pulse but no obtainable blood pressure. The patient received immediate successful direct current cardioversion.

Ultimately, 9 patients experienced ventricular tachycardia termination a median of 10 minutes (interquartile range 5.25 to 12.25) after initiation of amiodarone infusion without the initiation of another treatment (Figure). For the remaining 24 patients who received another treatment after amiodarone, the median time until the next treatment after initiation of amiodarone infusion was 25 minutes (interquartile range 20 to 42). Eighteen patients of 33 (55%), eventually required electrical therapy, including overdrive antitachycardia pacing, direct current cardioversion, or unsynchronized defibrillation for ventricular tachycardia termination.

LIMITATIONS

Because of the retrospective nature of this study, there were multiple potential limitations, including the risk of bias in enrollment and assessment of treatment by the primary physicians and the investigators, incomplete data collection, and nonstandardized treatment regimens. Data were collected from 4 hospitals, which may have led to an inhomogeneous patient population with a variety of ventricular tachycardia causes and presentations and could be interpreted as either a strength or weakness of the study.

Before any data collection, the primary outcome was chosen to be termination within 20 minutes of initiation of amiodarone infusion, which was an empiric choice allowing reasonable time for the agent to take effect but before other potentially confounding agents or modalities were used. It is possible that insufficient time was allowed for amiodarone to work. However, the median time to termination after amiodarone was 10 minutes, well within the 20-minute window, and the termination rate with medical treatment alone was less than 50%, regardless of any subsequent infusions the patients received. Therapeutic agents administered immediately before amiodarone may have also confounded the results, but

Table 3. Potential predictors of successful VT termination.*

Patient Characteristics	Total Patients, N=33	Terminated With Amiodarone, N=8	Not Terminated With Amiodarone, N=20	Response to Amiodarone Unknown, N=5	Mean Difference Between Terminated and Not Terminated (95% CI)
Historical					
Age (average)	67	69	66	70	3 (−12 to 18)
Sex (% male)	20 (61)	5 (63)	13 (65)	2 (40)	−3% (−45 to 39)
History of MI (% positive)	22 (66)	6 (75)	12 (60)	4 (80)	15% (−27 to 61)
Left ventricular ejection fraction (average %)	34	35	35	29 (N=4)	1 (−11 to 12)
History of sustained VT (% of patients)	13 (39)	4 (50)	6 (30)	3 (60)	20% (−23 to 67)
Chronic oral antidysrhythmic (% of patients)	10 (33)	1 (13)	7 (35)	2 (40)	−23% (−60 to 20)
AICD Implanted (% of patients)	13 (39)	3 (38)	7 (35)	3 (60)	3% (−39 to 52)
Event characteristics					
Heart rate (average beats/min)	162	160	160	179 (N=4)	1 (−29 to 30)
Systolic blood pressure (average mm Hg)	111	99	119	98 (N=4)	−21 (−40 to −1)
Diastolic blood pressure (average mm Hg)	70	62	75 (N=19)	63 (N=4)	−13 (−26 to −1)
QRS interval (average)	174	190	167 (N=18)	174 (N=4)	23 (−16 to 61)
Corrected QT interval (average)	546	540	556 (N=18)	520 (N=4)	−16 (−83 to 51)
Serum K+ (average mEq/L)	4.1	4.1 (N=7)	4.1	3.9	0.0 (−0.5 to 0.5)
Serum Mg^{2+} (average mEq/L)	1.7	1.5	1.7	1.7	−0.2 (–0.5 to 0.2)
Serum Ca^{2+} (average mg/dL)	8.6	8.4	8.4	9.6	0.0 (−2.4 to 2.3)
Recurrent VT despite AICD therapy (% of patients)	4 (12)	1 (13)	2 (10)	1 (20)	3% (−34 to 53)
Acute therapy					
Magnesium was infused before or within 20 minutes of amiodarone (% of patients)	3 (9)	1 (13)	2 (10)	0	3% (−34 to 53)
Amiodarone was initial antidysrhythmic treatment (% of patients; excluding adenosine or magnesium)	21 (64)	5 (63)	13 (65)	3 (60)	−3% (−45 to 39)
Amiodarone dose/weight (average mg/kg)	2.22	2.31	2.14	2.43	0.17 (−0.4 to 0.7)
Diagnosed postevent					
Acute MI associated with event (% of patients)	4 (12)	2 (25)	0	2 (40)	25% (−13 to 71)

MI, Myocardial infarction.
*Predictors not shown include physical examination findings, which were evenly distributed between groups, and duration of tachycardia prior to treatment, which was commonly unknown or uncertain.

whether or not amiodarone was administered first was not associated with termination rate in this sample (Table 3).

DISCUSSION

Amiodarone, administered intravenously with a median dose of 2.2 mg/kg, was poorly effective in terminating sustained monomorphic ventricular tachycardia within 20 minutes of infusion.

This result is not surprising based on the known acute electrophysiologic effects of amiodarone infusion on ventricular myocardium.[17,18] However, amiodarone could differentially alter conduction or repolarization in small subsets of normal, scarred, or ischemic myocardium and exert effects not evident on the surface ECG or during the electrophysiologic study of normal myocardium. For this reason, and because of the difference in mechanisms of various ventricular tachydysrhythmias, these results cannot be extrapolated to the treatment of ventricular fibrillation or nonsustained ventricular tachycardia, which may be associated with acute ischemia.

The dose of amiodarone most commonly used by treating physicians in this study was that recommended by the American Heart Association, 150 mg. However, this dose is less than the amiodarone dose most commonly studied, 5 mg/kg. It is possible that higher doses would be more effective. In this sample, patients who experienced termination with amiodarone received an 8% higher dose on average than patients who did not (Table 3). It is concerning, however, that 1 of the 2 patients who received 300 mg of amiodarone became hypotensive and required emergency direct current cardioversion.

IV amiodarone acutely depresses myocardial contractility,[19-22] and hypotension and cardiovascular collapse have been described in association with administration of amiodarone to terminate ventricular tachycardia.[12,13] Other less common adverse effects of IV amiodarone include bradycardias and prodysrhythmia, specifically torsades de pointes. The rate of bradycardia or heartblock was 1% to 5%, and torsades de pointes was about 1% in trials of IV infusions of amiodarone for hours to days for the treatment of refractory or recurrent ventricular tachycardia.[3-5] Hypotension and asystole were observed in this study. However, asystole occurred after infusion of lidocaine and amiodarone, and lidocaine alone has been reported to cause heartblock and asystole.[25,26] There was no torsades de pointes.

The relatively low doses of amiodarone administered by physicians in this study may have limited the efficacy and

adverse effects of the agent. Unfortunately, amiodarone dosing was beyond the authors' control because of the retrospective study design.

Lidocaine, procainamide, and sotalol have also been recommended by the American Heart Association for the termination of stable ventricular tachycardia. Lidocaine is generally safe but relatively ineffective for this purpose.[27,28] Based on limited data, agents that prolong the ventricular myocardial refractory period such as procainamide and sotalol are more effective for the termination of ventricular tachycardia.[29-31] However, procainamide, a sodium-channel blocker and negative inotrope, and racemic sotalol, which contains the β-blocking enantiomer L-sotalol, can cause hypotension, which is a particular concern in the setting of ventricular tachycardia in which the cardiovascular reserve is limited.[29-32] This study is part of a larger protocol designed to compare directly the efficacy and safety of amiodarone and procainamide for ventricular tachycardia termination.

Synchronized direct current cardioversion is the safest and most effective currently available treatment for the termination of sustained ventricular tachycardia.[1] It should be used early with appropriate sedation. The primary risks associated with direct current cardioversion are rare synchronization failure and subsequent ventricular fibrillation and the possibility of recurrent ventricular tachycardia. Antidysrhythmics such as amiodarone can be administered to prevent ventricular tachycardia recurrence.

Patients with recurrent persistent ventricular tachycardia, sometimes described as electrical storm, present the most difficult problem. As the population of automatic internal cardiac defibrillator patients expands, the incidence of this condition in emergency and critical care settings will increase. More study is required to understand why this condition occurs and its relationship to underlying myocardial scar and fibrosis, chamber dilatation, ischemia, or other abnormalities. Recurrent internal or external electrical therapy alone is insufficient treatment for this condition. Further study of current and future medical agents to determine optimal approaches is required.

In summary, a retrospective case series of patients with sustained monomorphic ventricular tachycardia treated with IV amiodarone was described. At the doses administered, amiodarone infrequently terminated ventricular tachycardia within 20 minutes of initiation of infusion. The primary adverse effect was occasional temporally associated hypotension. Direct current cardioversion with appropriate sedation remains the preferred modality for the rapid termination of sustained ventricular tachycardia in the emergency setting.

Supervising editor: Judd E. Hollander, MD

Author contributions: KAM conceived and designed the study and supervised its conduct and data collection. ISD and DKN collected and analyzed data. TOS and GSS assisted and facilitated data collection. JNR analyzed ECGs and assisted with analysis. KAM analyzed the data, performed the statistical analysis, and drafted the manuscript. All authors were involved in revision. KAM takes responsibility for the paper as a whole.

Funding and support: There was no external funding or support for this study. The authors have no financial interest in the sale of amiodarone or any competing products or in the companies that produce them.

Publication dates: Received for publication July 13, 2005. Revision received August 16, 2005. Accepted for publication August 23, 2005. Available online November 21, 2005.

Presented as an abstract at the Society for Academic Emergency Medicine annual meeting, May 2005, New York, NY.

Reprints not available from the authors.

Address for correspondence: Keith A. Marill, MD, Massachusetts General Hospital, 55 Fruit Street, Clinics 115, Boston, MA 02114; 617-726-6636, fax 617-724-0917; E-mail kmarill@partners.org.

REFERENCES

1. DeSanctis RW. Electrical conversion of ventricular tachycardia. *JAMA*. 1965;191:96-100.
2. American Heart Association, International Liaison Committee on Resuscitation. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care, part 6: advanced cardiovascular life support: 7d: the tachycardia algorithms. *Circulation*. 2000;102(8 suppl):I-158-I-165.
3. Levine JH, Massumi A, Scheinman MM, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. *J Am Coll Cardiol*. 1996;27:67-75.
4. Kowey PR, Levine JH, Herre JM, et al. Randomized, double-blind comparison of intravenous amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. *Circulation*. 1995;92: 3255-3263.
5. Scheinman MM, Levine JH, Cannom DS, et al. Dose-ranging study of intravenous amiodarone in patients with life-threatening ventricular tachyarrhythmias. *Circulation*. 1995;92: 3264-3272.
6. Mooss AN, Mohiuddin SM, Hee TT, et al. Efficacy and tolerance of high-dose intravenous amiodarone for recurrent, refractory ventricular tachycardia. *Am J Cardiol*. 1990;65:609-614.
7. Helmy I, Herre JM, Gee G, et al. Use of intravenous amiodarone for emergency treatment of life-threatening ventricular arrhythmias. *J Am Coll Cardiol*. 1988;12:1015-1022.
8. Klein RC, Machell C, Rushforth N, et al. Efficacy of intravenous amiodarone as short-term treatment for refractory ventricular tachycardia. *Am Heart J*. 1988;115:96-101.
9. Mostow ND, Rakita L, Vrobel TR, et al. Amiodarone: intravenous loading for rapid suppression of complex ventricular arrhythmias. *J Am Coll Cardiol*. 1984;4:97-104.
10. Saksena S, Rothbart ST, Shah Y, et al. Clinical efficacy and electropharmacology of continuous intravenous amiodarone infusion and chronic oral amiodarone in refractory ventricular tachycardia. *Am J Cardiol*. 1984;54:347-352.
11. Morady F, Scheinman MM, Shen E, et al. Intravenous amiodarone in the acute treatment of recurrent symptomatic ventricular tachycardia. *Am J Cardiol*. 1983;51:156-159.
12. Schutzenberger W, Leisch F, Kerschner K, et al. Clinical efficacy of intravenous amiodarone in the short term treatment of recurrent sustained ventricular tachycardia and ventricular fibrillation. *Br Heart J*. 1989;62:367-371.

13. Benaim R, Uzan C. Les effets antiarythmiques de l'amiodarone injectable (a propos de 153 cas). *Rev Med*. 1978;19:1959-1963.
14. Somberg JC, Bailin SJ, Haffajee CI, et al. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. *Am J Cardiol*. 2002;90: 853-859.
15. Wellens HJ, Brugada P, Abdollah H, et al. A comparison of the electrophysiologic effects of intravenous and oral amiodarone in the same patient. *Circulation*. 1984;69:120-124.
16. Singh BN. Antiarrhythmic actions of amiodarone: a profile of a paradoxical agent. *Am J Cardiol*. 1996;78(suppl 4A):41-53.
17. Morady F, Dicarlo LA, Krol RB, et al. Acute and chronic effects of amiodarone on ventricular refractoriness, intraventricular conduction and ventricular tachycardia induction. *J Am Coll Cardiol*. 1986;7:148-157.
18. Kulakowski P, Karczmarewicz S, Karpinski G, et al. Effects of intravenous amiodarone on ventricular refractoriness, intraventricular conduction, and ventricular tachycardia induction. *Europace*. 2000;2:207-215.
19. Kosinski EJ, Albin JB, Young E, et al. Hemodynamic effects of intravenous amiodarone. *J Am Coll Cardiol*. 1984;4:565-570.
20. Remme WJ, Kruyssen HA, Look MP, et al. Hemodynamic effects and tolerability of intravenous amiodarone in patients with impaired left ventricular function. *Am Heart J*. 1991;122:96-103.
21. Schwartz A, Shen E, Morady F, et al. Hemodynamic effects of intravenous amiodarone in patients with depressed left ventricular function and recurrent ventricular tachycardia. *Am Heart J*. 1983; 106:848-856.
22. Pfisterer M, Burkhart F, Muller-Brand J, et al. Important differences between short- and long-term hemodynamic effects of amiodarone in patients with chronic ischemic heart disease at rest and during ischemia-induced left ventricular dysfunction. *J Am Coll Cardiol*. 1985;5:1205-1211.
23. Wellens HJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *Am J Med*. 1978;64:27-33.
24. Brugada P, Brugada J, Mont L, et al. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*. 1991;83:1649-1659.
25. Gianelly R, Von der Groeben JO, Spivack AP, et al. Effect of lidocaine on ventricular arrhythmias in patients with coronary heart disease. *N Engl J Med*. 1967;277:1215-1219.
26. Pfeifer HJ, Greenblatt DJ, Koch-Weser J. Clinical use and toxicity of intravenous lidocaine. *Am Heart J*. 1976;92:168-173.
27. Armengol RE, Graff J, Baerman JM, et al. Lack of effectiveness of lidocaine for sustained, wide QRS complex tachycardia. *Ann Emerg Med*. 1989;18:254-257.
28. Marill KA, Greenberg GM, Kay D, et al. Analysis of the treatment of spontaneous sustained stable ventricular tachycardia. *Acad Emerg Med*. 1997;4:1122-1128.
29. Gorgels AP, van den Dool A, Hofs A, et al. Comparison of procainamide and lidocaine in terminating sustained monomorphic ventricular tachycardia. *Am J Cardiol*. 1996;78:43-46.
30. Sharma AD, Purves P, Yee R, et al. Hemodynamic effects of intravenous procainamide during ventricular tachycardia. *Am Heart J*. 1990;119:1034-1041.
31. Ho DSW, Zecchin RP, Richards DAB, et al. Double-blind trial of lignocaine versus sotalol for acute termination of spontaneous sustained ventricular tachycardia. *Lancet*. 1994;344:18-23.
32. Marill KA, Runge T. Meta-analysis of the risk of torsades de pointes in patients treated with intravenous racemic sotalol. *Acad Emerg Med*. 2001;8:117-124.