Electrophysiologic determinants and clinical experience in termination of sustained ventricular tachycardia[1]

Kenneth V. Adams, M.D.
Namburu V.R. Raju, M.D.
Richard Sterba, M.D.
Lon W. Castle, M.D.
Victor A. Morant, M.D.
James D. Maloney, M.D.

During a 19-month period, 60 patients underwent 86 procedures resulting in sustained ventricular tachycardia (VT). Pacing restored sinus rhythm in 45 procedures (52.3%). Cardioversion was required in 34 procedures (39.5%). Ventricular tachycardia was self-terminating in 7 procedures (8.1%). No procedural complication occurred. The relationship of cycle length to termination mode was assessed in 20 studies in which VT did not accelerate. The average cycle length of VT terminable by pacing was 369 msec; that of VT requiring cardioversion was 298 msec ($P < 0.05$). Seventy-one studies provided precise intervals of inducement and termination of stimuli. Cardioversion was needed in 0/6 studies when VT was induced with sensed single or double stimuli; in 2/9 studies (22%) in which VT was spontaneous, catheter-induced, or induced by atrial pacing; in 3/8 studies (38%) induced by paced single stimuli; in 16/39 studies (41%) induced by paced double stimuli; and in 6/9 studies (67%) induced by burst pacing ($P = 0.31$). Follow-up disclosed that 44/55 patients (80%) were alive at two days to 23 months after VT induction, with 7 deaths in the first six months. We conclude that: (1) VT induction is a safe procedure regardless of termination mode when meticulous technique is exercised; (2) termination by pacing may become less likely as the rate of VT or complexity of inducing stimuli increases; and (3) mortality is highest in the first four months after stimulation of sustained VT.

Index terms: Electric countershock • Heart function tests • Tachycardia, induced • Tachycardia, paroxysmal

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Programmed ventricular stimulation is a provocative electrophysiologic procedure employed for diagnosis and

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therapeutic guidance in the management of patients with known or suspected recurrent sustained ventricular tachycardia (VT).[1-3] Once sustained tachycardia has been induced, programmed electrical stimuli, along with drugs and vagal maneuvers, can terminate the tachycardia. If this fails to convert the arrhythmia or if the patient cannot tolerate the rhythm, direct current cardioversion (DCC) must be performed. In this study we (1) describe the electrophysiologic factors that influence the mode of termination of sustained VT; (2) document the clinical complications resulting from these procedures; (3) describe some practical aspects of management of sustained VT; and (4) relate our follow-up experience in this group of patients.

Materials and methods

Between 12 August 1982, and 29 March 1983, 60 patients underwent 86 stimulation procedures resulting in VT. The patients ranged in age from 3½ years to 75 years (mean 58.2 years). There were 52 males and 8 females. All had been referred to the Cleveland Clinic for evaluation of documented (or strongly suspected) recurrent VT or syncope. Most patients had atherosclerotic heart disease as an organic substrate for tachycardia. One patient had primary myocardial disease, one had right ventricular dysplasia, one had a structurally normal heart, 2 had mitral valve prolapse, and 3 had undergone previous open heart surgery (2 aortic valve replacements and one tetralogy repair). Informed consent was obtained orally from all patients before taking them to the Electrophysiology Laboratory. The patients were studied in the postabsorptive state with either intravenous diazepam (2.5 to 5 mg) or intramuscular pentobarbital (100 mg) given as sedation. As each patient reached the fluoroscopy table, two radiopaque conductive adhesive pads (Corotin Apex-Posterior, R2 Corporation, Skokie, IL) were attached to the skin, one at the apical region and the other between the scapular angles. These were attached by cables to a four-button control switch (R2 Corporation), which, in turn, was attached to a cardiac defibrillator (Physio Control Life Pack 6).

Diagnostic studies were done in the following manner. Three pacing and recording catheters were placed at the right ventricular apex, right atrial appendage, and near the bundle of His. Intracardiac electrograms were recorded on either a Mingograf or a Gould Electronics chart drive recorder at paper speeds of 50–250 mm/sec. Surface electrocardiogram leads I, aV_F, and V_1 were visualized at all times. Programmed extrastimuli were delivered by means of a Medtronic 5325 stimulator at voltages twice the existing diastolic threshold. Atrial pacing and premature atrial stimuli were delivered in the routine manner to determine sinus and atrioventricular node function. Single and double ventricular extrastimuli were first introduced into sinus rhythm, then into paced rhythms at one, two, or three drive cycle lengths. In every case, extrastimuli were made increasingly premature until ventricular refractoriness was reached or sustained ventricular tachycardia (defined as lasting at least one minute) was induced. If no VT had occurred at this point, burst pacing was done at cycle lengths of 333-240 msec for five to ten captures per burst. Burst pacing refers to a limited number of electrical stimuli delivered at a preset interval and energy level. If necessary, the same procedure was repeated in the right ventricular outflow tract. After induction of VT, oxygen was administered and close continuous clinical assessment begun, including level of consciousness (orientation, memory, speech pattern, responsiveness to questions and commands), cuff blood pressure, qualitative changes in pulse pressure as determined by finger plethysmography, and symptoms, particularly chest pain. Simultaneously with onset of VT, programmed electrical stimulation was begun with single and double extrastimuli. If needed, burst pacing was done at a cycle length 10–40 msec shorter than that of the VT, for five to 10 captures. If tachycardia persisted, or if the patient was thought to be unstable, cardioversion was accomplished immediately (occasionally preceded by a 40–60 mg intravenous injection of sodium methohexital). After restoration to sinus rhythm, drug testing was done. After the procedure, all catheters were usually removed and the patient sent to a monitored hospital bed. In some cases, a single catheter was left in the right ventricular apex for retesting.

Electrophysiologic data were collected retrospectively by reviewing procedure reports and/or the actual studies. All procedures were classed as either requiring or not requiring at least one cardioversion (or defibrillation), regardless of the number of times VT occurred within a single procedure. If no cardioversion was needed, the tachycardia was noted to have been ended either

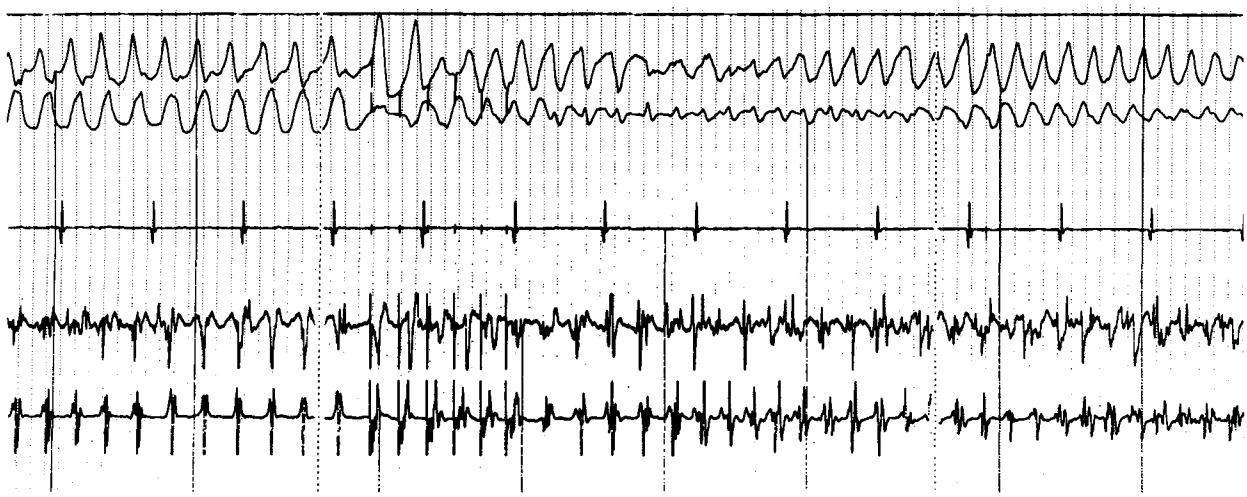

Fig. 1. Ventricular tachycardia (cycle length = 220 msec) of stable morphology is accelerated by burst pacing to torsade de pointes (cycle length = 180 msec). From top: ECG leads aV_F V_1; intracardiac electrograms from high right atrium, bundle of His, and right ventricular apex. Paper speed = 50 mm/sec.

by some form of pacing or spontaneously by termination. Follow-up was obtained by personal contact with patients, their families, or personal physicians. Statistical correlations were done by either two-sample t-test or chi-square analysis.

Results

Studies were evaluated in two groups (*Table 1*). Group 1 consisted of 86 studies performed in 60 patients. Group 2 consisted of 37 studies evaluable for presence or absence of acceleration of VT. Acceleration was present in 17 studies (*Fig. 1*). Correlation was made between tachycardia rate and mode of termination in 19 studies in which VT did not accelerate (*Fig. 2*). The mean cycle length of tachycardias that could be paced out of VT was 359 msec. The mean cycle length of patients requiring DCC was 278 msec ($P < 0.05$).

Seventy-one studies included precise intervals of extrastimuli used to induce and terminate VT (*Table 2*). No clinical complication (defined as death during the procedure, thromboembolic phenomenon, neurologic deficit, or chest discomfort persisting after termination of VT) was encountered.

One patient had refractory recurrent VT with cardiovascular collapse during the course of an acute myocardial infarction, and had been cardioverted at least 20 times during the 36 hours prior to electrophysiologic study. Tachycardia persisted despite drug administration combined with multiple attempts at pacing, and she died within 24 hours of the test.

Five patients underwent left ventricular aneurysmectomy with intraoperative electrophysiologic mapping after initial VT induction. Four are alive at nine to 14.5 months (mean 10.7 months) postoperatively, and none has had any further episodes of VT. One patient could not be weaned from cardiopulmonary bypass and died. Postmortem examination revealed a myocardial contusion with hemorrhage. He may have sustained a clinically inapparent infarction just prior to surgery.

Follow-up data were obtained in 55/58 pa-

Table 1. Relative frequencies of conversion modes among all studies (group I), and studies evaluated for acceleration (group II)

	Paced to NSR	Cardioverted	Spontaneous conversion	Total
I. All studies	45 (52.3%)	34 (39.5%)	7 (8.1%)	86
II. Studies evaluated for acceleration				37
A. Accelerators	3 (17.6%)	13 (76.5%)	1 (5.9%)	17
B. Nonaccelerators	9 (45.0%)	10 (50.0%)	1 (5.0%)	20

NSR = normal sinus rhythm $P < 0.10$

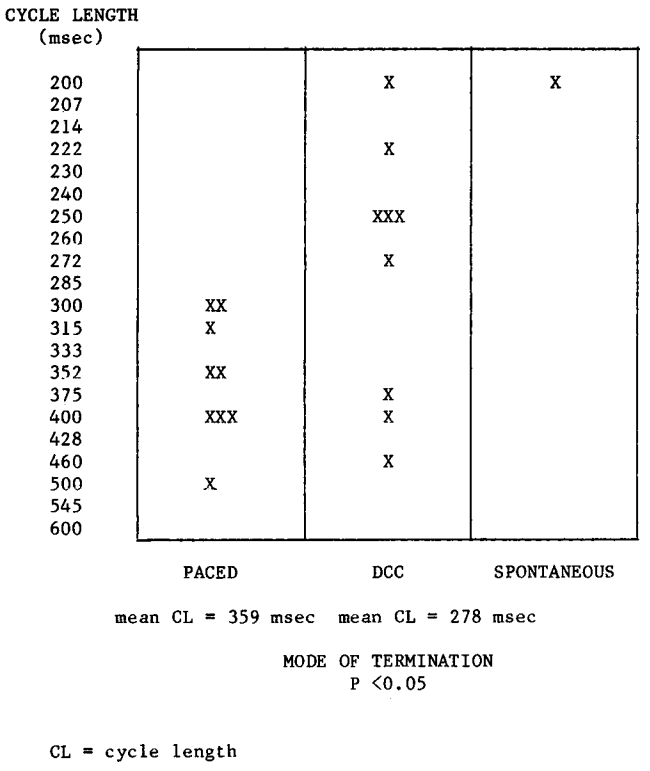

Fig. 2. Nonaccelerating tachycardias: rates vs. termination modes.

Table 3. Survival after induction of sustained VT: 55 patients: number of months followed

Months	Patients
1	00
1	0
2	
3	00 XXXX
4	0 XXX
5	XXX
6	0 XX
7	XX
8	X
9	XXXXXXX
10	XX
11	0 XXXX
12	00 XX
13	0 XX
14	XXX
15	X
16	XX
17	X
18	
19	X
20	
21	X
22	XX
23	X

0 = died
X = alive

tients (95%) who were followed from two days to 23 months (mean 9.6 months) from the date of their initial study. Eleven deaths (20%), all sudden, occurred in this group. All cases followed were arranged according to length of follow-up in months from the date of first VT induction (*Table 3*). Most deaths (6/11) occurred within the first four months after electrophysiologic study. In 16 studies, no significant association was found between subsequent death and induction mode, termination mode, rate, or presence of acceleration during VT. Acceleration data were present in only 5/16 studies, but other data were present in nearly all studies.

Table 2. Mode of induction of VT and probability of requirement for DCC

Mode of Induction	# Studies	# Cardioverted	%
Sensed singles	2	0	0
Sensed doubles	4	0	0
Spontaneous, atrially paced, or catheter-induced	9	2	22%
Paced singles	8	3	38%
Paced doubles	39	16	41%
Burst pacing	9	6	67%
TOTALS	71	27	38%

P = 0.31

Discussion

Induction of ventricular tachycardia is a provocative technique that offers electrophysiologists a scientific approach to the treatment of patients afflicted with a grimly inexorable disease process.[4–6] One apprehension commonly voiced by those unfamiliar with the technique centers around the risks and consequences of provoking unstable rhythms which may require DCC countershock.[7–9] Lepeschkin has analyzed repolarization changes in electrocardiograms of patients after cardioversion, and has described gross and microscopic damage inflicted by multiple cardioversions performed on open human hearts (Lepeschkin E, Jones JL, Rush S, and Jones RE. Analysis of cardiac damage following elective cardiac defibrillation. Unpublished manuscript). Ehsani et al[10] provoked modest elevations in creatine phosphokinase isoenzyme (CPK-MB) in 2 of 30 patients who underwent shock as treatment for atrial arrhythmias. However, these 2 patients received more than 400 joules apiece, which is much more than others were given in the study.

Marchlinski et al[11] reported serial myocardial enzyme studies after cardioversion done during the course of electrophysiologic studies in 9 patients, 7 of whom had coronary artery disease. All 9 patients' CPK-MB fractions were negative. Ditchey and Karliner[12] reported the delivery of countershock to 21 patients for atrial arrhythmias. All were on nontoxic doses of digoxin (serum levels <3 ng/ml); 17 were on other antiarrhythmic agents. No patient had ventricular tachycardia induced, and the incidence of other arrhythmias did not increase after electroversion. Thus, our low complication rate confirms the consensus that VT induction is a safe practice provided the arrhythmia is promptly terminated when necessary.

Several factors have aided in this last respect. First, placement on the patient of adhesive pads connected to the defibrillator at the start of every electrophysiologic procedure eliminates suddenly having to move a heavy piece of equipment in an emergency, thus narrowing the time lag between hypoperfusion and its reversal. Second, our practice of constant clinical support and assessment during sustained tachycardias, including oxygen delivery, verbal interaction with the patients, and cuff blood pressures every 20 seconds, is an effective means of judging the need for electroversion. Our approach avoids arterial line placement and contrasts with the opinion of Mason and Winkle[13] that arterial cannulation is necessary. In addition, what Fisher et al[14] describe as the physical and psychologic discomfort of cardioversion has been obviated in many of our cases by quickly administered intravenous doses of methohexital just before the shock, causing unconsciousness within five seconds. This drug has been used prior to electroversion of atrial arrhythmias and is equally well suited for our use. Further, it assures that patients have amnesia for the event, which is seldom, if ever, obtained by minor tranquilizers and analgesics.

We report a 42.5% incidence of cardioversions for the series, an 8.1% rate of spontaneous conversion, and a 52.3% rate of conversion by pacing. These figures are fairly close to those of Mason and Winkle[13] (*Table 4*) who used cardioversion in 29% of 51 studies in 33 patients. Seventeen patients (52%) were shocked in that series. Naccarelli et al[15] performed 89 studies in 57 patients and was able to terminate VT in 67% of procedures by pacing. This figure is similar to the percentage of paced cardioversions added to the spontaneous conversions in our series. Naccarelli et al[15] also noted that 84% of patients could be paced out of VT if the cycle length was greater than 350 msec, and only 51% if the cycle length was less than 349 msec. We have obtained similar results in this series using only studies proved not to have been affected by tachycardia acceleration, and this result is statistically significant. A similar relationship of reported cycle length to ease of termination is present in our entire series. Roy et al[16] used DCC in only 29 of 139 patients (21%). They noted that half of the patients whose tachycardia cycle length was ≤300 msec were cardioverted. They speculated upon the possible relation between modes of induction and ease of termination of VT. To our knowledge, our data are the first to document this association, although this correlation did not achieve statistical significance, probably because of the fairly small patient sample. Moreover, we believe that a statistical trend is present, and additional data will further establish the association. The concept that ease of induction facilitates termination appears justified in light of the reentrant model, which has been proposed as the mechanism of VT.[1] If the circuit of reentry is anatomically small or distant from the stimulating site, or if the rate of reentry is fast, it is possible that the difficulty of penetrating and interrupting the circuit will be proportional to that encountered in activating it.

We believe that acceleration of VT is a frequent result of pacing during tachycardia. However, these rhythms might have become faster whether or not extrastimuli were being produced. Certainly the future application of antitachycardia pacing must allay concern that the treatment may be worse than the disease.[17] An implantable, low-energy, transvenous cardioversion unit may provide a safeguard if rates become too fast.[18–20] More must be learned about tachy-

Table 4. Relative frequency of cardioversion during VT
(Various authors)

Author	Number of patients	Number of studies	Number of patients cardioverted (%)	Number of studies cardioverted (%)
Mason[24]	33	51	17 (52%)	15 (29%)
Naccarelli[15]	57	89	23 (40%)	29 (33%)
Roy[16]	139	139	29 (21%)	29 (21%)
Authors	60	80	28 (46.6%)	34 (39.5%)

cardia acceleration and its relation to therapeutic termination.

Actuarial statistics in our series suggest that our patients' lives have probably been prolonged by our therapy, which uses VT induction studies to guide a comprehensive program of drug selection and follow-up in the hospital, clinic, and at home by telemetry. A randomized, prospective, controlled protocol is difficult to envision in practical terms. Historical control studies show mortality for untreated inducible patients to be 50% for the first year after induction.[21-23] Mason and Winkle[24] and Ruskin et al[25] describe much higher death rates for patients who could not be protected from VT induction by drugs than for patients in whom electrophysiologic testing indicates that drug therapy will be protective. Similar survival data have been obtained by noninvasive methods by Graboys et al[26] who undertook sequential drug tests using serial Holter monitoring and stress tests. The high incidence of death in the first four months after electrophysiologic study can be interpreted in two ways: perhaps these patients were so sick that our intervention failed to affect their course. In Swerdlow et al,[27] the description of class IV congestive failure as an independent predictor of early mortality supports this view. Second, more assiduous post-procedure assessment might reveal high-risk patients. The 80% survival of our small group of guided aneurysmectomy patients suggests that surgery is feasible in refractory cases, and indicates that these patients may enjoy long periods of freedom from VT if they can survive surgery.

We conclude that: (1) VT induction is a safe procedure regardless of termination mode; (2) termination by pacing may become less likely as the rate of VT or complexity of inducing stimuli increases; (3) mortality is highest in the first four months after stimulation of sustained VT.

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