**Efficacy of Direct Injection of Ethanol into the Myocardium
to Control Aconitine-induced Ventricular
Tachycardia in Anesthetized Dogs**

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SUMMARY

The effect of injecting ethanol directly into the myocardium to control aconitine-induced ventricular tachycardia (VT) was evaluated in anesthetized dogs. In 17 dogs, VT was induced by injecting aconitine (1.0 μ g, median dose) directly into the epicardium. After inducing persistent VT for up to 3 min, 0.6 ml (median volume) of 96% ethanol was injected into the same epicardial region. Regular sinus rhythm reappeared in 15 dogs with no change in systolic blood pressure; the other 2 dogs died of ventricular fibrillation (VF). In another 13 dogs, VT was induced by injecting aconitine directly into the endocardium using a Variocath needle catheter. After persistent VT for up to 3 min, a regular sinus rhythm was restored in 7 dogs by injecting 2.0 ml (median volume) of 96% ethanol; the remaining 6 dogs died of VF. Histology showed no transmural necrosis and the subendocardial necrotic areas were essentially the same in the dogs that recovered from VT as in those that died. There was no statistically significant relationship between doses of ethanol and VT duration. These preliminary results suggest that the injection of ethanol into the myocardium may efficiently terminate VT when other techniques fail. (Jpn Heart J 1996; 37: 611-625)

Key words: Chemical ablation Ventricular tachycardia Ethyl alcohol

PATIENTS with sustained ventricular tachycardia (VT) are at high risk of sudden cardiac death when the arrhythmia cannot be controlled by antiarrhythmic drug therapy and/or devices.^{1,2)} Treatment of VT by radio-frequency catheter ablation is less promising than the ablation of accessory and/or dual atrioventricular (AV) pathways.³⁻⁶⁾ Further, surgical intervention to inflict a lesion by direct myocardial resection, or to stimulate the myocardium by the

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implantation of an autonomic defibrillator has disadvantages.⁷⁻¹¹⁾ Inoue et al¹²⁾ reported the usefulness of ethyl alcohol or phenol directly injected into coronary artery in ablating VT. Although several investigators^{13,14)} modifying their method using ethyl alcohol reported that it terminates VT, clinical application of this procedure is difficult because the technique of infusing ethanol into a coronary artery can lead to necrosis in normal and unrelated regions of the endocardium.¹⁵⁾

Direct subendocardial injection of ethyl alcohol via a catheter was first reported by Weismuller et al,¹⁶⁾ who found that their procedure led to a controlled local lesion. However, they did not examine the procedure with respect to its possible usefulness in controlling VT. Therefore, the present study was designed to determine whether or not the direct injection of ethyl alcohol into the myocardium, wherein a lesion controlled for location and size would be inflicted, could be used to terminate VT without causing unnecessary damage in other areas of the myocardium. To accomplish this purpose, we used an endoscopic needle catheter to inject ethanol into the myocardium of a VT model following a technique that is used successfully in sclerotherapy for esophageal varices.^{17,18)}

MATERIALS AND METHODS

Epicardial experiment: Seventeen mongrel adult dogs of either sex, weighing 13–28 kg, were anesthetized with ketamine hydrochloride, 2 mg/kg IM followed by pentobarbital (30 mg/kg, IV); additional doses were administered as needed to maintain deep anesthesia. The dogs were intubated with a cuffed endotracheal tube and ventilated with room air using a Harvard respirator. Under positive pressure ventilation, the chest was opened through a left thoracotomy at the fourth or fifth intercostal space and the heart was suspended in a pericardial cradle. A fluid-filled cannula was inserted into the left cervical artery and connected to a transducer (Statham p-23 dB) to monitor arterial pressure, and a left-cervical venous cannula was used to infuse normal saline at 30 ml/h to replace spontaneous fluid loss. A 1 ml syringe with a 26-gauge needle was carefully placed in the epicardium of the area between the first and second diagonal branch of the left anterior descending (LAD) coronary artery (Figure 1) in preparation for aconitine administration. Surface electrocardiographic (ECG) lead II and arterial pressure were recorded throughout the experiment. A left ventricular electrogram (LVE) was also recorded using a bipolar electrogram catheter inserted from the right femoral artery. Recordings were made on a Nihonkoden recorder (RMC 1100) at paper speeds of 5, 10, 25, 50 and 100 mm/sec, as needed. Recordings from Marquette Holter ambulatory monitoring leads, aVf and CM5, were maintained throughout the experiment. Once the animals had

Figure 1. Site of aconitine administration used to induce VT during open heart surgery in the dog, and of ethanol administration using direct injection into the epicardium to suppress the induced VT.

been fully connected to the instruments, and after a control recording time period of at least 10 min, 1.0 μg (median dose; range 0.25 to 3.75 μg) of aconitine was carefully injected into the epicardium until VT was provoked. After the VT had persisted for at least 3 minutes, 0.6 ml (median dose; range 0.2–2.0 ml) of 96% ethanol was carefully injected into the same epicardial region through a separate 26-gauge needle until the normal sinus rhythm reappeared. Recovery from VT to normal sinus rhythm was determined by the appearance of a normal rhythm that lasted for at least 3 min while the animal was hemodynamically stable, as defined previously.¹⁹⁾ The return to normal sinus rhythm and hemodynamic stability was confirmed by the failure of normal saline injected through the needle to alter these physiological events. Recording from the Holter ambulatory monitoring leads also confirmed the recovery from VT. If the systolic blood pressure fell below 90 mmHg, 2 to 3 ml of methoxamine (0.1 to 0.3 mg/kg) were administered IV. The alpha adrenergic receptor agonist effects of methoxamine were expected to increase blood pressure and maintain the coronary perfusion pressure by prolonging the diastolic phase and slowing the heart rate. The dogs were sacrificed with a lethal IV dose of pentobarbital at the end of the experiment and while still under deep anesthesia. The hearts were examined for acute morphologic damage and samples of left ventricular tissue were obtained from the morphologically damaged areas and from the visibly normal surrounding

tissue. Gross measurements were made (in mm) of the greatest length, width, and depth of the acute lesions; the necrotic areas produced by ethanol were easily recognized macroscopically. Assuming that the area of the lesion is that of an ellipse and the volume of the lesion is that of a flattened rotated ellipsoid, the size of the lesion was calculated as follows: $\text{area} = \text{length} \times \text{width} \times \pi/4$; $\text{volume} = \text{area} \times \text{depth}/1.5$. Thereafter, tissues were removed and fixed in formalin for at least two weeks before being examined histologically. Lesioned areas were sectioned and stained with phosphotungstic acid hematoxylin-eosin and Masson's trichrome stain to assess myocardial injury.

Endocardial experiment: Thirteen mongrel dogs were anesthetized as described above. After each dog was intubated and ventilated, a fluid-filled cannula was inserted into the femoral artery to monitor arterial pressure. A bipolar catheter to record the left ventricular electrogram was placed in the left ventricle from the right femoral artery, as described above. A 23-gauge needle catheter (Variocath type C) that is used to cauterize esophageal varices was then carefully introduced into the left ventricle using a 9F guiding catheter. As shown in Figure 2, the tip length of the needle could be controlled (from 1 to 5 mm) using a nut adjustment. Aconitine mixed with 0.2 ml contrast medium, in an amount equivalent to that used in the epicardial experiment, was carefully injected into the endocardium through the needle catheter. This was carried out under fluoroscopic control after pushing the needle 1–2 mm out of the lumen of the guiding catheter (Figure 3). The criteria for VT were the same as in the epicardial injection experiment. After VT was induced, the needle catheter was quickly withdrawn and changed to another 23-gauge needle catheter, to be used for the ethanol injection. The needle catheter was carefully introduced into the left ventricle using the same 9F guiding catheter and the needle tip was extended out to the same length as before. The tip could not penetrate the myocardium since the length of the needle tip was controlled by a nut adjustment. The positions of the

Figure 2. Variocath type C needle catheter. Tube length was 1000 mm and lumen diameter was 1.80 mm (left); tip of needle showing 2 mm out of guiding catheter (right).

Figure 3. The position of tip of needle from the Variocath and the position of the bipolar electrogram catheter in the left ventricle.

needle tips were the same during all procedures. After the duration of VT was confirmed, the same amount of 96% ethanol was injected into the endocardium until the VT was replaced by a regular sinus rhythm. Both ECG and systemic hemodynamics were monitored during these manipulations. Procedures for euthanization, inspection of morphologic damage, and preparation of tissue for histology necrosis were the same as those described in the epicardial experiments

Analysis of data: Data are expressed as mean $\pm$ SD or median and range. Differences between means were analyzed by paired and unpaired t-tests and a level of $p < 0.05$ was considered statistically significant. The doses of ethanol and duration of VT were analyzed with linear regression analysis.

RESULTS

Epicardial study; Induction and termination of VT: Administration of 1.0 μ g (median dose) of aconitine induced VT in the 17 dogs (Table I). After VT had persisted for at least 3 min, 0.6 ml (median dose) of 96% ethanol at 21°C administered to the epicardial region of each dog terminated VT in 15 of the dogs (group A); the remaining 2 (group B) died of ventricular fibrillation (VF), showing a consequent fall in both systolic and diastolic blood pressure (Table I). In the 15 dogs in Group A, a regular sinus rhythm was restored after the ethanol injection and stable hemodynamics persisted for at least 30 min thereafter. There were no differences between groups A and B with respect to cycle length (pre- and during VT) or blood pressure during VT. Data on each of the dogs used in the study are presented in Table I. The sequence of the induction and suppression of VT in a representative dog (#5) is shown in the Holter monitoring record (Figure 4). The

first three lines show the normal heart rate of the dog at the outset of the procedure, starting at 15:35. VT was induced shortly thereafter 15:38 (arrow) and was fully apparent and steady by 15:40. The administration of ethanol at 15:46 (point 1) was followed by an immediate cessation of VT for the remainder of that minute. However, VT resumed during the next minute (15:47) and continued until a further dose of ethanol was administered at time 15:50 (point 2). VT reappeared shortly thereafter and ethanol was again administered at 15:56 (point 3). Following a brief respite, VT resumed and a fourth dose of ethanol was administered at 16:02 (point 4). Thereafter, VT gradually diminished and a regular sinus rhythm was established by 16:07; systemic hemodynamics were stable thereafter. In another animal (#8), the aconitine induced VT was converted into a regular sinus rhythm immediately after ethanol administration into the epicardium. At the same time, systolic blood pressure rose dramatically from 74 to 100 mmHg (Figure 5). Gross and histologic examinations revealed myocar-

Table I. Data for the 17 Dogs Used in the Epicardial Study

(A) Group A (converted to regular sinus rhythm)						
Dog number	Aconitine (μ g)	Ethanol (ml)	Cycle length (msec)			BP (VT)
			Pre VT	During VT	Post VT	
(1)	1.0	0.6	460	224	510	149/98
(2)	1.75	1.6	450	240	410	118/78
(3)	0.75	2.0	380	208	400	142/112
(4)	0.25	1.0	352	108	304	159/101
(5)	1.75	0.8	330	184	350	107/87
(6)	1.0	0.6	450	262	460	105/64
(7)	2.75	0.2	400	272	328	126/85
(8)	1.0	0.6	450	320	492	74/59
(9)	0.75	0.4	500	175	507	126/100
(10)	2.0	0.4	490	260	480	150/70
(11)	0.75	0.4	520	260	320	119/103
(12)	1.0	0.6	380	210	400	105/82
(13)	1.0	0.6	460	260	500	107/60
(14)	1.25	0.2	360	210	340	93/62
(15)	3.75	0.4	520	170	480	140/100
1.51 $\pm$ 1.02			433 $\pm$ 62	224 $\pm$ 52	419 $\pm$ 77	121 $\pm$ 23/84 $\pm$ 18*
(B) Group B (death by VF)						
(1)	1.25	0.4	304	216	VF	95/72
(2)	0.75	0.2	390	200	VF	51/28
1.0 $\pm$ 0.36			347 $\pm$ 61	208 $\pm$ 11		73 $\pm$ 31/50 $\pm$ 31

Data include amounts of aconitine needed to induce VT, doses of 96% ethyl alcohol administered to the epicardium to suppress the induced VT, cycle lengths and blood pressures. Suppression of VT was successful in 15 dogs (A); the other 2 (B) died following ventricular fibrillation.

VT = ventricular tachycardia; VF = ventricular fibrillation; BP = blood pressure (mmHg) during VT.

* $p < 0.05$ (group A vs. group B). Values are mean $\pm$ SD.

Figure 4. The Holter monitoring record of a representative case (dog #5) from time 15:35 through 16:08. VT was induced by aconitine injection during 15:53 (arrow) and disappeared gradually after administration of 96% ethanol into the epicardium (15:46, point 1). After several injections of 96% ethanol into the epicardium (points 2, 3 and 4), the VT was completely gone by 16:07.

dial necrosis following the administration of ethanol in all dogs studied. The necrosis was observed in all animals, i.e., those that reverted to regular sinus rhythms (group A, $n = 15$) and those that died of VF (group B, $n = 2$) following the epicardial administration of ethanol. Histologic examination showed no transmural necrosis and no luminal fibrin or thromboses in either group. The volume of necrosis in the dogs that reverted to regular sinus rhythm appeared to be larger than in those that died of VF (Table II). Further, neither the depth nor the maximum diameters of the regions were different in the two groups (Table II). A typical example of the macroscopic extent of the lesion is shown in Figure 6, and a typical example of the microscopic examination by HE staining is shown in Figure 7.

Figure 5. The record of a case (dog #4) wherein the induced VT was converted to a normal sinus rhythm immediately after an ethyl alcohol injection into the epicardium. Systolic blood pressure immediately increased from around 50 mmHg to 100 mmHg. ECG = surface electrocardiography; HBE = His bundle ECG; LVECG = left ventricular ECG; LVMP = LV monophasic potential ECG; AP = aortic pressure.

Table II. Characteristics of the Lesions in the Epicardial Study

	Group A dogs (<i>n</i> = 8)	Group B dogs (<i>n</i> = 2)
Volume (mm ³)	61 ± 47	124 ± 92.0
Depth (mm)	1.5 ± 0.5	1.3 ± 1.0
Maximum diameter (mm)	15.6 ± 8.5	14.5 ± 10.8

Group A (converted to regular sinus rhythm), Group B (death by VF), Values are mean ± SD.

Endocardial study; Induction and termination of VT: The administration of aconitine, 1.75 μ g (median dose; range 1.0–23.75 μ g) induced VT in all 13 dogs (Table III); this dosage of aconitine was not significantly different from that used in the epicardial experiments. VT in 7 of the 13 dogs was converted to a regular sinus rhythm after administration of 2.0 ml of ethanol (median; range, 0.6–3.5 ml), a dose that was not significantly different from that used for the epicardial studies. The remaining 6 dogs failed to respond to ethanol administration and died of VF. Cycle lengths in the two groups before and during VT were not significantly different, but systolic and diastolic blood pressures in group C were significantly higher than in group D during VT (Table III). Gross and histologic examinations revealed a necrosis similar to that created by epicardial injection of ethanol. Neither the depth nor the maximum diameters of the lesions were different in the two groups. The size of necrotic areas of those that died of VF were not significantly different from those that recovered to a regular sinus rhythm (Table IV). In both the epicardium and endocardium studies, doses of ethanol in dogs

Figure 6. Transverse ventricular slices showing the typical endocardial necrosis (whitish yellow) induced by the administration of 96% ethanol; dog #5 (group C) of endocardial study.

Figure 7. Histologic examination of the left ventricle of dog #5 showing necrosis of myocardial cells with disarray and dislodgment. (Phosphotungstic Acid Hematoxylin Eosin staining)

that recovered to normal sinus rhythm were compared to the ethanol doses of the dogs that died of VF; there was no significant difference between the groups (Table V). Further, there was no significant relationship between the dose of ethanol and VT duration (Figure 8).

Table III. Data for the 13 Dogs Used in the Endocardial Study

(C) Group C (converted to regular sinus rhythm)						
Dog number	Aconitine (μ g)	Ethanol (ml)	Cycle length (msec)			BP (VT)
			Pre VT	During VT	Post VT	
(1)	1.0	0.6	430	260	420	149/98
(2)	23.75	0.6	460	290	390	118/78
(3)	15	3.0	530	230	610	125/80
(4)	15	1.6	418	270	440	69/52
(5)	0.5	2.0	404	141	500	76/69
(6)	1.0	2.0	468	185	544	137/57
(7)	2.0	3.5	383	157	410	144/99
	8.3 $\pm$ 9.5	1.90 $\pm$ 1.10	442 $\pm$ 49	219 $\pm$ 59	473 $\pm$ 81	116 $\pm$ 32*/76 $\pm$ 10*
(D) Group D (death by VF)						
(1)	1.75	0.5	520	230	VF	68/50
(2)	12.5	2.1	360	150	VF	56/40
(3)	1.25	5.0	380	260	VF	84/60
(4)	0.5	1.0	413	180	VF	109/82
(5)	1.25	5.0	430	83	VF	31/6
(6)	8.75	3.0	390	76	VF	46/9
	4.33 $\pm$ 5	2.77 $\pm$ 1.94	416 $\pm$ 57	163 $\pm$ 75		66 $\pm$ 30 / 42 $\pm$ 30

Data include amounts of aconitine needed to induce VT, doses of 96% ethyl alcohol administered to the endocardium to suppress the induced VT, cycle lengths and blood pressures. Suppression of VT was successful in 7 dogs (C); the other 6 (D) died following ventricular fibrillation.

VT = ventricular tachycardia; VF = ventricular fibrillation; BP = blood pressure (mmHg) during VT. * $p < 0.05$ (group C vs. group D), Values are mean $\pm$ SD.

Table IV. Characteristics of the Lesions in the Endocardial Study.

	Group C ($n = 4$)	Group D ($n = 4$)	p values
Volume (mm ³)	69 $\pm$ 40	126 $\pm$ 92	ns
Depth (mm)	2.5 $\pm$ 0.5	1.3 $\pm$ 0.5	ns
Maximum diameter (mm)	7.6 $\pm$ 3	8 $\pm$ 5	ns

Group C (converted to regular sinus rhythm), Group D (death by VF), Values are mean $\pm$ SD

Table V. Epicardial and Endocardial Studies: Ethanol Doses and VT Duration in Dogs that Survived (wherein VT was Converted to Regular Sinus Rhythm) and Dogs that Died of VF

	Converted to regular sinus rhythm ($n = 22$)	Death by VF ($n = 8$)	p values
Ethanol (ml)	1.76 $\pm$ 2.3	2.2 $\pm$ 1.9	ns
VT duration (seconds)	154 $\pm$ 156	79 $\pm$ 95	ns

Values are mean $\pm$ SD.

Figure 8. Correlation of ethanol doses (ml) versus VT duration (seconds).

DISCUSSION

In this study, 15 dogs recovered from aconitine-induced VT after ethanol was injected directly into the epicardium, while 2 others died of VF following aconitine administration. In addition, 7 of the 13 dogs recovered from aconitine-induced VT after ethanol was injected into the endocardium with the Variocath type-C needle catheter, while 6 others died of VF. Pathologic examination suggested that injection of 96% ethanol directly into the epi- and endocardium created a necrotic myocardial region that could terminate the aconitine-induced VT. By keeping the size of the lesion small, effective VT suppression could be accomplished without inducing severe hypotension. In the surviving dogs, the blood pressures in both epi- and endocardial experiments were significantly higher than those pressures recorded in dogs that died of VF during the experiment. Therefore, the VF that led to the deaths probably resulted from a secondary subendocardial ischemia caused by hypotension. This seems likely because the blood pressure in these dogs could not be maintained despite the infusion of methoxamine.

Recent basic and clinical studies indicate that lesions induced by intracoronary injection of 50–100% ethanol can terminate VT. Inoue et al¹²⁾ first reported that intra-coronary injection of 25% phenol or 50–100% ethanol could suppress aconitine-induced VT in dogs. Gursay et al²⁰⁾ described the clinical use of ethanol injections into coronary arteries in treating recurrent sustained VT, and reported that ethanol injected into a tachycardia-related artery successfully terminated VT. However, a transient elevation of creatine phosphokinase (CPK) and chest pains followed shortly thereafter. De Paola et al²¹⁾ also reported on the

use of 96% ethanol to generate transcoronary chemical ablations to control VT in chronic Chagas myocarditis; they claimed to successfully induce an ablation in the tachycardia-related coronary artery without a consequent acute clinical or laboratory deterioration of left ventricular function. However, because the proximal coronary artery supplies a large area, its use for ethanol infusion risks damage to an excessive amount of myocardium. Instead, intracoronary infusion of ethyl alcohol should be used in only selected instances, such as those in which there is an anatomically well-defined area of arrhythmogenic VT and where the infused coronary artery supplies the peripheral artery. In such cases there would be no chance of massive myocardial necrosis that could elevate the ST segment or the CPK. Surgical interventions for the treatment of VT have involved cryoablation techniques and direct transmural ventriculostomy. Cryosurgery for ablation of sustained monomorphic VT due to coronary artery disease has proven to be a useful procedure. However, since the depth of a cryolesion depends upon the duration and degree of freezing, the procedure can increase the duration of an operation. In any event, the depth of the lesion is difficult to evaluate with current techniques.²²⁾ Direct transmural ventriculostomy is a useful procedure for recurrent VT, but it poses a higher mortality risk than does ICD implantation²³⁾ and it requires an open heart endocardial resection approach that involves peeling off a layer of endocardium and/or often the resection of the rim of an aneurysm. ICD devices are now markedly improved over what they were in the past. Kron et al²⁴⁾ and others⁸⁻¹¹⁾ reported that ICD can be used for treating VT, and a randomized study of the use of an ICD for controlling VT in patients 2-4 weeks after MI has shown its effectiveness.²⁵⁾ However, such equipment is in the trial stage, and it is still to be determined whether or not an ICD is better than antiarrhythmic agents.²⁶⁾ One disadvantage is that the generator must be changed every few years, depending on its frequency of usage. Furthermore, an ICD is costly and somewhat risky. Nunain et al²⁷⁾ reported that hardware-related complications and/or inappropriate shocks have occurred in patients after ICD devices were implanted. Therefore, patients implanted with ICD devices may be subjected to additional and unnecessary physical stress. Weismuller et al¹⁵⁾ reported on the feasibility of chemical ablation by a subendocardial injection of ethanol via a catheter. They recognized the possibility of generating a local necrotic area of limited and controlled size after 25 days of initial chemical ablation, and they further suggested the possibility of chemical ablation by direct injection into the endocardium. They also reported that the necrotic region created by an ethanol injection into the endocardium does not impair left ventricular function after 3 weeks because the initial zone of necrosis is nearly completely subsumed by retracting connective tissue. The present data suggest that both direct epi- and endocardial injection of ethanol can terminate VT, possibly by

inducing ablation in the VT related epi- and endocardial region. However the doses of ethanol in those cases where VT was replaced by regular sinus rhythms were not significantly greater than in those cases where dogs died of VF. Further, there was no significant relationship between the dose of ethanol and VT duration. Therefore, optimal ethanol doses could not be determined in this study. The technique of ethanol administration used in the present study may be a more suitable procedure than radiofrequency ablation for a VT that originates at a deep region of the endocardium, since radiofrequency ablation cannot reach intermediate or epicardial regions. The present method may be superior for use in polymorphic VT because the necrotic region is rather large, although after several weeks necrotic regions might be replaced by retracting connective tissue.

There are several limitations in the present study. First, aconitine-induced VT differs from VT observed in patients. Aconitine-induced VT is believed to stem from enhancement after depolarization due to cathodal currents or in response to low external Ca^{++} concentrations, which in turn cause altered Na^{+} and Ca^{++} exchange.²⁸⁾ Therefore, the model used in the present study does not necessarily apply to other cases of VT that stem from depolarization induced automatically in the Purkinje fibers or ventricular muscle. Further, studies of chronic infarction with inducible VT in an animal model are also required to demonstrate the applicability and effectiveness of transluminal chemical catheter ablation in this situation. This study was limited in scope to a model in which recovery from VT was not followed beyond several hours.

Second, the injection of ethanol directly into the ventricular epi- and endocardium does not always result in ethanol entering the myocardium exclusively; some of the fluid may leak into the surrounding areas. This is substantiated by our finding that the amounts of ethanol injected via the endocardial approach may have been slightly greater than the amounts injected via the epicardial approach. However, there were no serious hemodynamic effects. Nevertheless, this point should be examined in detail in subsequent studies. Third, the possibility of perforation should be considered. In the Weismuller et al¹⁵⁾ study, perforation occurred twice but there were no further complications. We did not encounter any perforations in the present study, possibly because we employed a fine needle catheter that is used clinically in our hospital for the treatment of esophageal varix and gastric ulcer. Nevertheless, in a clinical setting, application of this technique in cases having a thin ventricle (e.g., dilated cardiomyopathy) should be avoided because of the increased risk of perforation; further adjustment to make an appropriately sized lesion might be difficult.

Despite these limitations, the present pilot study suggests that such a technique might be feasible in a clinical setting. This would be true if a catheter is specially designed so that the balloon in the catheter tip is kept in the same

position inside the ventricle to minimize leakage of ethanol. In addition, this technique could be useful if conventional methods, such as antiarrhythmic agents and/or radio-frequency techniques, are unsuccessful.

In summary, VT induced by direct myocardial injection of aconitine into anesthetized dogs can be converted to regular sinus rhythm by direct epicardial or endocardial injection of ethanol into the same myocardial region. These preliminary data suggest the utility of this chemical ablation technique for terminating VT.

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