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**Effects of Digoxin on Ventricular Vulnerability During Atrial Defibrillation via Implanted Transvenous Catheter Electrodes in the Sterile Pericarditis Model**

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**Objectives** Because digoxin is often used in patients with atrial fibrillation, we evaluated its effects on the relative risk of inducing ventricular tachyarrhythmia (VT/VF) during synchronized atrial defibrillation shocks delivered through implanted transvenous catheter electrodes.

**Methods.** Sterile pericarditis was created in 10 mongrel dogs. Two catheter electrodes with a 6 cm coil tip were inserted through the right external jugular vein and placed in the distal coronary sinus and in the right atrial appendage. To systematically achieve variable preceding ventricular coupling intervals (VCIs), the ventricles were paced during sinus rhythm before delivery of the atrial shock in two protocols: 1) rapid pacing (8 stimuli, S<sub>1</sub>S<sub>1</sub> protocol); and 2) double extra stimuli (long-short intervals) following 8 basic stimuli (S<sub>1</sub>S<sub>2</sub>S<sub>3</sub> protocol). The VCI (S<sub>1</sub>S<sub>1</sub> or S<sub>2</sub>S<sub>3</sub>) was shortened by 10 ms until the ventricular effective refractory period was achieved. After sensing the last paced ventricular beat, an atrial shock was delivered with a 2 ms delay at a shock intensity of 300 V (3.2-3.6 J), i.e., maximum shock intensity of the device. Atrial shocks were delivered 4 times at each VCI in each protocol. VCI was normalized by subtracting the QT interval in the surface ECG from the actual VCI. The relationship between the VCI and induction of VT/VF was evaluated in the drug free state and after administration of a therapeutic (50 µg/kg, 5 dogs) or toxic (to obtain toxic arrhythmia, 5 dogs) dose of digoxin.

**Results.** Data are shown in mean ± SD, for each protocol (S<sub>1</sub>S<sub>1</sub> or S<sub>1</sub>S<sub>2</sub>S<sub>3</sub>). To calculate a 99% probability of sinus rhythm (SR) after an atrial shock, logistic regression was used. No significant difference was observed before and after digoxin administration. The longest VCI that preceded VT/VF induction was QT + 50 ms (290 ms) before digoxin and QT + 50 ms (310 ms) after digoxin.

	digoxin	QT (ms)	VT/VF induction	99% probability of SR (ms)
control	226±22	6/377 (S <sub>1</sub> S <sub>1</sub> )	27/303 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )	QT - 4 (S <sub>1</sub> S <sub>1</sub> ) QT + 71 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )
therap	237±44	8/178 (S <sub>1</sub> S <sub>1</sub> )	16/151 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )	QT + 1 (S <sub>1</sub> S <sub>1</sub> ) QT + 73 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )
toxic	220±8	8/177 (S <sub>1</sub> S <sub>1</sub> )	15/142 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )	QT + 1 (S <sub>1</sub> S <sub>1</sub> ) QT + 63 (S <sub>1</sub> S <sub>2</sub> S <sub>3</sub> )

**Conclusions.** Digoxin did not significantly change the risk of VT/VF induction during atrial defibrillation shocks delivered through transvenous catheter electrodes at maximum shock intensity. Shocks delivered at VCIs longer than 310 ms resulted in no VT/VF induction.

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**Internal Atrial Defibrillation: Need for Concomitant Pacemaker Therapy and Its Effect on Pacemaker Function**

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Implantable defibrillators are being considered as a therapeutic option for the chronic management of atrial fibrillation (AF). The need for concomitant pacemaker therapy and the effect on existing pacemaker function of internal atrial defibrillation (IAD) shocks is unknown. 21 pts, mean age 67 ± 11 yrs, mean LA diameter 43 ± 7 mm, mean LV ejection fraction 36.5 ± 15%, underwent IAD for drug refractory AF. 7 pts had existing VVIR pacemakers. Lead configurations tested for IAD using a step up protocol were RV-RA, RV-SVC, RV-axillary patch, RA-left pulmonary artery (LPA) or RA-coronary sinus (CS). RA-LPA or CS were preferred for pts with pacemakers. **Results;** Of 178 biphasic shocks delivered with energies of 1 to 20J, 36 were successful in cardioverting AF and these were analyzed. R-R interval preceding the successful shock ranged from 200 to 1000 ms. Mean time to the first post shock QRS was 1076 ± 368 ms (vs mean proshock R-R interval of 780 ± 18 ms, p = 0.005) and the mean first sinus cycle length of 168 ± 490 ms (vs 780 ± 18 ms proshock R-R interval, p < 0.005). Significant post shock bradycardias occurred in 6 pts (28%). 1 pt had sinus arrest with third degree AV block lasting for 7.5s. 2 pts had third degree AV block requiring ventricular pacing support. 3 pts had post shock pauses >2s with 1 pt having a persistent sinus cycle of 2.5s for 6s. There was no correlation between energy used, lead configurations and the incidence of bradycardias. The 7 pts with existing VVIR pacemakers were successfully cardioverted using the RA-LPA or CS configuration at a mean energy of 9.81 ± 8.6J. There was no effect of IAD using the RA-LPA or CS configuration on pacemaker function. **Conclusions;** 1. Pts undergoing IAD may have a transient bradycardia following successful cardioversion which may require backup concomitant ventricular pacing. 2. IAD can be performed safely in pts with existing pacemakers using RA-LPA or CS lead configurations without affecting pacemaker function at the energy levels tested. 3. An implantable atrial defibrillator should incorporate concomitant ventricular pacing support.

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**Atrial Defibrillation Using a Unipolar, Single Lead Right Ventricular to Pectoral Can System**

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**Background;** The active can, unipolar right ventricular (RV) single lead system has been shown to be very effective for ventricular defibrillation using an RV<sup>+</sup> → CAN<sup>-</sup> biphasic pulsing configuration. If this same RV<sup>+</sup> → CAN<sup>-</sup> unipolar system could perform double-duty, providing atrial as well as ventricular defibrillation, it would broaden the role of current implantable cardioverter-defibrillators (ICDs). It would provide an atrial ICD without multiple shock electrodes, simultaneously providing backup protection for inadvertent induction of ventricular fibrillation (VF) following atrial defibrillation.

**Methods and Results;** The purpose of this study was to determine the RV<sup>+</sup> → CAN<sup>-</sup> atrial defibrillation threshold at the time of ICD surgery for 10 VT/VF patients using the Medtronic 7219C ICD shell with a biphasic 65% tilt, 60 µF capacitance pulse delivered one minute following induction of AF. Initial pulse strength was 100 V and was incremented in 100 V steps every minute if atrial fibrillation persisted. The atrial defibrillation threshold data with the RV<sup>+</sup> → CAN<sup>-</sup> system were 8.3 ± 4.1 joules, 511 ± 128 volts, and 58 ± 8 ohms.

**Conclusions;** Although the RV<sup>+</sup> → CAN<sup>-</sup> lead system does not provide atrial defibrillation at energies likely to be painless (e.g., <0.2J), the safety and simplicity of this system has advantages that must be considered when compared to more complicated two and three lead electrode systems. If detection algorithms for AF, using near and far field electrogram analysis prove accurate, such a simple lead system may prove clinically viable.

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**Is Coronary Revascularization Complete Therapy for Secondary Prevention of Ischemic Cardiac Arrest?**

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Coronary revascularization has been suggested as sole therapy for secondary prevention of sudden cardiac arrest associated with ischemia. Among 412 consecutive patients receiving an implantable defibrillator (ICD), 23 (6%) were identified as: sudden cardiac arrest survivors, noninducible with programmed stimulation, unstable angina or ischemia on a functional study, and underwent successful coronary revascularization. In follow-up, 10 (43%) of the 23 patients received ICD shocks (8 ± 8 per patient, range: 1-22) shocks and 9/10 had syncope/presyncope associated with at least one ICD discharge.

Clinical Characteristics:	ICD firings (n = 10)*	No ICD firings (n = 13)*
Follow-up (months)	39 ± 13	31 ± 21
Age (years)	63 ± 7	63 ± 12
Male gender	8	9
Mean left ventricular ejection fraction (%)	36 ± 10	40 ± 14
Previous history of a myocardial infarction	10	10
Presence of a left ventricular aneurysm	4	1
Q-wave infarction pattern on electrocardiogram	7	5
Sudden cardiac arrest presenting with exertion, angina, or CPK elevation	8	8
Mean number of vessels with coronary disease	2.2 ± 0.8	2.3 ± 0.9
Mean severity of coronary stenosis (%)	87 ± 18	88 ± 16
Coronary revascularization considered complete	7	10
β-blocker therapy	5	5
Antiarrhythmic therapy	8	12

\*p value > 0.05

No clinical characteristic was statistically different between patients with and without ICD shocks. In conclusion, coronary revascularization alone may be inadequate therapy for survivors of sudden cardiac arrest associated with ischemia who are noninducible with programmed stimulation, and clinical variables cannot predict which patients are likely to experience recurrent malignant ventricular arrhythmias. Therefore, ICD therapy should be considered in these patients.

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**Noninvasive Predictors of Successful Implantation of Transvenous Defibrillator Lead Systems**

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Successful implantation of transvenous defibrillator lead systems depends on achieving an intraoperative defibrillation threshold (DFT) of ≤25 J. Since defibrillation is related to a critical mass of myocardium, we sought to determine whether left ventricular (LV) mass or LV volume could predict successful

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