patients following myocardial infarction. However, little is known about the prognostic value of these tests in patients with malignant ventricular tachyarrhythmias.

Method: The deep breath test (DB) is a widely used cardiovascular reflex test. It measures the difference (Diff) between maximum heart rate during deep inspiration and minimum heart rate during deep expiration. The test is noninvasive, straight forward and easy to perform. 36 pts (34 male, 2 female, mean age: 52 ± 12 yrs), who had been resuscitated from ventricular fibrillation underwent DB before implantation of a cardioverter/defibrillator (ICD).

Results: The mean heart rate during the pretest period was 75 ± 13 beats/min.. During deep inspiration mean heart rate increased to 84 ± 13 beats/min and decreased after expiration to 67 ± 12 beats/min (p = 0.001). 18/36 (50%) pts had an abnormal DB (Diff ≥ 15 beats/min) (group A), the remaining 18 demonstrated a normal test (group B). Group A and B were comparable with respect to age, gender, underlying heart disease (CHD: 68 vs 68%), ejection fraction (42 vs 43%), NYHA-class (class III 22 vs 27%), inducibility during programmed stimulation (48 vs. 54%).

During a mean follow up of 14 ± 4 months 20/36 (56%) pts developed appropriate shocks, 16 (89%) pts in group A, but only 4 (22%) pts in group B (p = 0.001).

Conclusion: The DB is appropriate for assessing cardiac autonomic nervous dysfunction in ICD-patients. An abnormal DB predicts an unfavorable clinical outcome and indicates specific trigger mechanisms for subsequent arrhythmic events.

983-32 Long-Term Stability of the Defibrillation Threshold With A Pectoral Unipolar Defibrillation System

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The chronic stability of the defibrillation threshold (DFT) in patients (pts) with an active pectoral defibrillation system remains still to be defined. We report on our experience with a single lead unipolar pectoral defibrillation system in 52 consecutive pts during a follow-up period of 12 months. The defibrillation lead system consisted of a single transvenous right ventricular electrode (cathode) and the shell (anode) of an active, left pectorally implanted defibrillator device (Medtronic, model 7219 C). DFT was determined in all pts using a binary search protocol at the time of implant, at discharge, after 3, 6, and 12 months.

	Stored energy [J]	Resistance [Ohm]
At implant:	9.8 ± 6.3	53 ± 5
At predischarge:	7.4 ± 4.9	47 ± 6
At 3 month:	8.3 ± 5.9	56 ± 6
At 6 month:	7.9 ± 6.1	59 ± 7
At 12 month:	8.8 ± 6.8	60 ± 6

Intraoperatively, the defibrillation success rate at 9 J, 12 J, 18 J, and 24 J was 58%, 83%, 90%, and 98%, respectively. There was no significant change in stored energy at the DFT for the total patient population during a 12 month follow-up period. Furthermore, a significant rise in defibrillation energy requirements did not occur in a single patient throughout the observation period.

Conclusions: Defibrillation thresholds remain stable over a 12 month follow-up period using the unipolar pectoral defibrillation lead system. This observation has important clinical implications for future implantable cardioverter-defibrillators with reduced energy outputs to ensure an adequate energy safety margin for continued effectiveness.

983-33 Reduced Defibrillation Thresholds With Endocardial Administration of Artilide

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A defibrillation energy requirement (DER) with adequate safety margins is required for implantation of a defibrillator. Many patients require antiarrhythmic (AA) drugs to decrease the frequency of ICD therapies, and these drugs may elevate the DER. Therefore, the purpose of this study was to determine the effect of local endocardial delivery of artilide, a type III AA, on the DER of dogs. In 11 dogs, the chest was opened and two epicardial defibrillation patches (CPI model L67) were placed. DER determinations used an up-down protocol. The likelihood of 50% (ED 50) and 100% (ED 100) successful defibrillation were calculated. Five minutes were allowed to elapse between each induction of ventricular fibrillation. A transvenous lead (TVL; CPI model 0060) with a controlled release drug delivery system attached to the tip was then placed transvenously into the apex of the right ventricle. In 6 dogs the delivery system was impregnated with 20% artilide, and in 5 dogs no drug (control) was put into the system. Thirty minutes later, the DER was determined again. Before the TVL was positioned, the ED 50 and ED 100 were 8.6 ± 2.7 J and 14.0 ± 2.2 J in the control animals, and 8.0 ± 2.0 J (p = NS vs control) and 13.3 ± 4.1 J (p = NS vs control) in the dogs treated with the artilide, respectively. After positioning the TVL, the artilide ED 50 and ED 100 decreased to 4.8 ± 0.9 J (p = 0.0005) and 8.5 ± 3.7 J (p = 0.02), respectively. For the control dogs, the ED 50 and the ED 100 did not change significantly (6.2 ± 2.4J, 13.0 ± 2.7J, respectively). In conclusion, artilide appears to decrease the acute ED 50 and ED 100 in dogs. This effect can be achieved with the endocardial administration of this agent via a controlled release system that results in low systemic concentrations.

983-34 Timing of the Upper Limit of Vulnerability Is Different for Monophasic and Biphasic Shocks: Implications for the Relation to the Defibrillation Threshold

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Background and Methods. The upper limit of vulnerability (ULV) has been used in clinical studies to predict the defibrillation threshold (DFT) in patients with implantable defibrillators. Despite the ULV-DFT correlation, uncertainties about the optimal timing of the ULV determination remain. To compare the precise timing of the ULV for monophasic (Mo) and biphasic (Bi) shocks, 10 isolated rabbit hearts were immersed in a tissue bath flanked by shock electrodes. Mo and Bi T wave shocks (overall duration 5 ms) were randomly delivered during the vulnerable period at shock strengths above and below the ULV with a 10 ms resolution for shock coupling intervals. The ULV was defined as the highest VF inducing shock strength.

Results. The ULV showed a substantial intra-individual variation with respect to shock timing and voltage. However, in all hearts the ULV for Bi shocks occurred at longer coupling intervals as compared to Mo shocks (186 ± 9 vs 173 ± 5 ms, p < 0.001). This resulted in a more complete repolarization level (RL) at the Bi ULV (81.1 ± 7.5 vs 66.9 ± 9.0% RL, p = 0.002) measured by simultaneously recorded monophasic action potentials from different endocardial and epicardial sites. As expected, the ULV voltage was lower for Bi than for Mo shocks (difference 44 ± 42V, p = 0.012) reflecting the higher defibrillation efficacy of the Bi waveform.

Conclusions. The ULV for Bi shocks occurs at a more complete repolarization state as compared to Mo shocks, thereby resulting in a rightward shift of the ULV towards longer coupling intervals. This is important for the determination of the ULV-DFT correlation. Our findings also explain why different ULV coupling intervals with respect to the T wave have been proposed in previous studies.

983-35 Relationship Between Amiodarone and Desmethylamiodarone Levels and Ventricular Defibrillation Energy Requirements

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Prior studies have suggested that amiodarone raises defibrillation energy requirements (DER), however no studies have assessed the effect of plasma concentrations of amiodarone or desmethyl-amiodarone (DEA) on the DER. We prospectively assessed the effect of amiodarone, amiodarone levels, and DEA levels on the DER. The DER was determined with a monophasic waveform with a single transvenous lead system. Intraoperative DER was determined using a step-down protocol: 25, 20, 15, 10, 5, 3 and 1J. The DER was defined as the minimum energy that converted ventricular fibrillation to sinus rhythm. If the DER was > 25J, the DER was considered elevated and was estimated to be 30J. Prospective data were collected in 102 pts (76 M; 24 F). The mean age was 62 ± 13, the mean ejection fraction was 0.31 ± 0.14. There were no differences between patients receiving amiodarone (n = 39) vs those not receiving amiodarone (n = 62) in age, gender, ejection fraction, type of heart disease, prior coronary artery bypass surgery, body surface area and left ventricular internal diastolic dimension and mass. The mean duration of amiodarone use was 24 ± 33 days (range: 7–181 days). The mean DER was 18 ± 9J. At implantation, the mean amiodarone level was 0.3 ± 0.6 mg/L, and the mean DEA level was 0.25 ± 0.4 mg/L. Pts receiving amiodarone had a significantly higher DER (22 ± 10J vs 17 ± 9J, p = 0.01). There was no significant correlation between amiodarone level (p = 0.4) or DEA level (p = 0.1) and the DER. In conclusion, the use of amiodarone is associated with elevated acute DER's, regardless of plasma amiodarone or DEA concentrations, and even when these concentrations are subtherapeutic.