ology. During the last years the devices became much smaller and hence the surgical approach became easier. Today the implantation of ICDs in the catheterization laboratory under local anesthesia is state-of-the-art. According to this progress in the intra- and postoperative management of ICD-patients it seems justified to raise the question if pre-discharge testing is necessary any more?

Methods: Since September 97 we implanted 72 ICDs in the catheterization laboratory using local anesthesia. A short acting narcotic was used for intraoperative evaluation of the defibrillation threshold (DFT). The DFT was tested according to a step-down protocol until failure. There were 11 female and 61 male patients (pts) with a mean age of 62 ± 10 years. The mean ejection fraction was 42 ± 18%. 41 pts suffered from primary ventricular fibrillation and 31 pts from ventricular tachycardias.

Results: The mean intraoperative DFT was 11.2 ± 5.4 J with a high-voltage impedance of 55.6 ± 10.5 Ohms. The mean DFT prior to discharge was 8.7 ± 4.9 Joule. In 9/59 pts the DFT rose above the intraop level (group 1), in 32/59 pts the DFT declined (group 2) and in the rest it was unchanged (group 3).

	Group 1 (Joule)	Group 2 (Joule)	Group 3 (Joule)
Intraoperative	9.1 ± 4.3	12.8 ± 5.9	8.6 ± 3.7
Pre-discharge	14.1 ± 6.1	7.2 ± 4.2	8.6 ± 3.8

In only one patient in group 1 an increase of more than 10 Joule was observed. Three months later the DFT had declined by 10 Joule to 4 Joule above the intraoperative level. All other pts had an increase of less than 7 Joule. The device was programmed according to the pre-discharge evaluation with a safety margin of 10 Joule above the DFT. There were no perioperative complications and no early mortality. During a mean follow-up of 6 ± 4 months only one patient died 4 months after implantation due to intractable cardiac failure. There was a total of 84 arrythmia episodes that were terminated successfully.

Conclusion: According to our data a pre-discharge test is not necessary anymore. In 58 out of 59 pts there was no relevant increase in DFT and in one patient the DFT declined to a significant amount within a short time. This concept reduces the hospital stay and thereby costs.

1150-178 When do T Wave Shock Induce VF? Observations From Monophasic Action Potential Recordings

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Introduction: Shocks given during the vulnerable period of cardiac repolarization may induce ventricular fibrillation (VF). This vulnerable period has been measured in relationship to the peak of the T wave. How the vulnerable period relates to cellular repolarization as measured by the monophasic action potential (MAP) has not yet been reported in humans, however.

Methods: Eleven patients undergoing implantable cardioverter defibrillator (ICD) implant had a MAP catheter positioned in the right ventricle (RV). The local MAP90 duration was measured during pacing at 400 ms as the time to 90% depolarization. VF inductions were attempted by pacing at 400 ms for 10 cycles and then giving a 1.0 joule monophasic T wave shock at varying coupling intervals (CI). The maximum and minimum CI that induced VF were determined and mapped in relation to the MAP90 recording.

Results: The average paced MAP duration was 275 ± 20 ms. The minimum and maximum CI to induce VF were 255 ± 24 ms and 325 ± 36 ms respectively. This ranged from 93% to 118% of the MAP90 duration.

Conclusion: VF is inducible with 1.0 joule T wave shocks ranging from 93% to 118% of the local MAP90 duration. The shorter CI that induce VF may initiate it near the site of pacing when myocardium is in late repolarization whereas the longer CI intervals may initiate VF at sites distant from the site of pacing where both depolarization and repolarization occur later and when sites near pacing are already refractory as evidenced by the CI being longer than 100% of the MAP90.

1151 Doppler Echocardiography

Tuesday, March 31, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1151-113 Etiology of the Pulmonary Venous Systolic Flow Wave: An Answer From Wave Intensity Analysis

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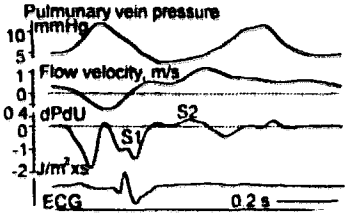
The increase in pulmonary vein (PV) flow during ventricular systole (S wave) seen in PV Doppler flow traces has been attributed to both atrial relaxation and descent of the mitral annulus. We tested the hypothesis that the S wave is caused by forward propagation of the pulmonary artery pressure pulse.

Methods: In 8 pts during CABG we recorded pulmonary vein pressure (P) and velocity (U) by microsensors and flow by ultrasound transit time. Wave intensity (dPdU) was the product of change in pulmonary vein pressure (dP) and velocity (dU) at 5 ms intervals. dP >0 is a *compression wave* and dP <0 *expansion wave*. dPdU >0 indicates net forward going wave and dPdU <0 *backward going* wave.

Results: Fig. shows a representative patient. Note that during early systole P falls and U rises, hence dPdU is negative, -0.62 ± 0.17 J/m² × s (N ± SE). This represents a backward going expansion wave (S1 in fig.), and is compatible with suction of blood into the left atrium. Later in systole both P and U rise and dPdU becomes positive, 0.30 ± 0.08 J/m² × s. This indicates a net forward going compression wave (S2) which "pushes" blood towards the atrium.



In conclusion, wave intensity analysis indicates that the late D. systolic increase in PV flow reflects forward propagation of the pulmonary artery pressure pulse.

1151-114 Relative Duration of Pulmonary Venous and Mitral A Waves: A Measure of LV Compliance or LVEDP? Mathematical Analysis

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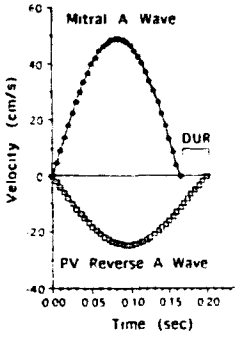
The difference in pulmonary venous (PV) and mitral A wave duration (ΔDur) is used to assess LV end diastolic pressure (LVEDP), with increasing ΔDur reflecting higher LVEDP. But there are interindividual differences in LVEDP for the same ΔDur. The theoretical underpinnings of these observations have not been described. We therefore explored the basic determinants of ΔDur in a mathematical model of LV filling. In principle, ΔDur is determined by LV stiffness relative to those of the atrium and PVs, which influences the time course of the pressure gradients that drive flow. In contrast, LVEDP depends not only on LV stiffness but also on chamber volume relative to a zero-pressure volume.

Methods: Using the unsteady Bernoulli equation, a system of differential equations for atrial contraction and diastolic filling were solved for the forward and reverse A waves. LV stiffness was varied from 0.05 to 0.2 mmHg/cc and LVEDP before atrial contraction was held constant at 10 mmHg by varying the zero-volume intercept; atrial and PV tree stiffness were held constant.

Results: At the same LVEDP, as ventricular stiffness increased from 0.05 to 0.2 mmHg/cc, ΔDur increased from 0 to 100 ms. In contrast, at the same ventricular stiffness, if LVEDP was varied by changing LV volume, ΔDur did not change.



Conclusions: In many patients, increased LVEDP is due to increased LV stiffness, hence the correlation between ΔDur and LVEDP is reasonable but less than perfect because LVEDP can be elevated for reasons other than increased LV stiffness, such as increases in LV volume. Changes in ΔDur are fundamentally determined by changes in LV stiffness, not pressure *per se*.

TUESDAY POSTER