2:45

850-4 Radiofrequency Catheter Ablation Using High Power

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Radiofrequency (RF) catheter ablation requires a tissue temperature (T°) ≥52°C. Some substrates require either higher T° or more energy to achieve a successful ablation and most generators are limited to a maximum of 50 W. We studied 904 consecutive ablations performed in our laboratory. 544 ablations were done using a standard RF generator (50 W max., non thermistor). 360 ablations were performed with a high power generator (EPT1000XP, 0–150 W, thermistor) using T° monitoring (set at 60–70 °C). Of these 360 ablations (all successful), 135 required >50 W to achieve the targeted T°. The substrates were AVNRT: 75, WPW: 35 (posteroseptal (PS): 12, right sided: 5, left sided: 18), AVN: 18 and atrial flutter in 8 pts. Five PS pathways were recurrences and 2 right anterior and 1 PS pathways were prior unsuccessful ablations. The mean fluoro. time was 19 ± 14 min with a median number of RF applications of 9. The effective lesion required a mean of 67 ± 11 W (mean power set at 84 ± 12 W) for 50 ± 17 sec to achieve a mean T° of 51 ± 4 °C (mean max. T° achieved 62 ± 7 °C, median 60 °C) with a mean impedance of 126 ± 14 Ω. When the mean effective lesion T° was <60 °C, a "bonus" lesion was applied increasing the power until the targeted T° was obtained during all the RF application (bonus: mean: 62.5 ± 16.4 W with median power set at 85 W). All procedures were successful and no coagulum formation was noted. In comparison, a 14% failure rate was observed in the 544 ablations done with the standard RF generator. There was no arrhythmia recurrence at a mean follow-up of 9 months.

In Conclusions: High power was required to obtain the desired T° in 38% of cases. Since T° monitoring is required to use more than 50 W (otherwise the output is locked at 50 W), it should be beneficial to use T° monitoring in all cases with this generator. The initial success rate is higher and prior ablation failures can be done successfully. The influence of this technology on the recurrence rate will need further studies.

3:00

850-5 Electroanatomic Mapping of Atrial and Junctional Tachycardia

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Catheter ablation of atrial and junctional tachycardia (AT/JT) can be complex and time consuming. The aim of this study was to determine the feasibility and safety of nonfluoroscopic electroanatomic mapping and ablation in 21 consecutive patients (pts) with AT or JT (5 men, 16 women, mean age 47 ± 15 years).

Results: electrophysiologic study and CARTO mapping of the right atrium was performed in 24 tachycardias and the mechanism determined as: junctional in 3 pts, incisional in 3 pts, reentrant in 4 pts, focal in 14 pts (4 left atrial). We created 24 maps with a mean of 79 ± 48 different catheter positions within the right atrium. The mapping procedure took 47 ± 16 min. CARTO mapping criteria for focal tachycardia could be defined as radial impulse propagation away from the site of earliest activation, clearly distant earliest and latest activation and a different tachycardia cycle length (CL) and activation time (366 ± 115 ms vs 94 ± 30 ms). Reentry tachycardias were characterized as close proximity of earliest and latest activity and a comparable tachycardia CL and activation time (236 ± 44 ms vs 240 ± 56 ms). The ablation of the 4 left AT was not attempted in the first ablation session. In 15 of 17 (88%) right AT and 2 of 3 JT, ablation was performed successfully. No complications occurred.

Conclusions: the visualization and 3D presentation of the atrial activation sequence with the CARTO system allows the differentiation of tachycardia mechanisms and the determination of the successful site of ablation in right atrial focal and junctional tachycardias.

3:15

850-6 True Anatomic Ablation of AV Nodal Reentry Using Radiofrequency Current Guided by Intravascular Ultrasound

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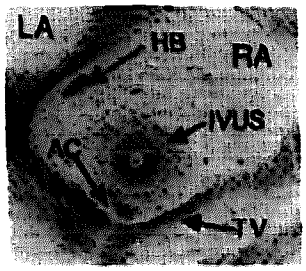
Radiofrequency (RF) ablation of the slow pathway of AV Nodal Reentry (AVNRT) is usually guided by fluoroscopic anatomy which shows the relative location of a mapping catheter to reference catheters. Intravascular ultrasound (IVUS) provides precise anatomic detail of the tricuspid annulus and coronary sinus and may be useful for mapping and ablation procedures requiring anatomic lesions. Accordingly, in sixteen consecutive patients with typical AVNRT, an IVUS probe (6.2F, 12 MHz) was placed along the tricuspid annulus. RF current was applied through an ablation catheter (AC) placed using IVUS, in front of the os of the coronary sinus on the tricuspid annulus (see Figure). In all 16 pts, slow pathway conduction was eliminated. Successful ablation was achieved with 1–7 RF pulses (median, 2). Fluoroscopic location of RF pulses was mid-septal in 4 pts and postero-septal in 6 pts despite IVUS location of the AC in the posteroseptal region in 16 pts.



Conclusions: Selective slow pathway ablation of AVNRT can be achieved using IVUS to place anatomic RF lesions. Fluoroscopic location of the AC is less accurate than IVUS. Once validated, this technique could reduce radiation exposure and the number of RF lesions.

851 Molecular Mechanism of Heart Failure

Tuesday, March 31, 1998, 2:00 p.m.–3:30 p.m.
Georgia World Congress Center, Room 255W

2:00

851-1 Adenosine Inhibits Cardiac Expression of Tumor Necrosis Factor-alpha in the Failing Human Heart

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Background: Tumor necrosis factor-alpha (TNF) has been implicated in the pathogenesis of CHF. We have previously shown that adenosine inhibits the lipopolysaccharide (LPS)-induced expression of TNF in rat cardiomyocytes and rat papillary muscle. The aim of this study was to determine whether adenosine has the same effect in the failing human heart muscle.

Methods: Trabecular muscles were isolated from the hearts of cardiac transplant recipients and stimulated with LPS (10 μg/ml). TNF release was measured with enzyme linked immunosorbent assay. Muscle sections were analyzed immunchistochemically for the presence of TNF.

Results: In contrast to healthy rat papillary muscles, trabecular muscles from failing human hearts released TNF in the absence of LPS (287 ± 91 pg/ml/g wet weight). However, addition of LPS induced a further 10-fold increase in TNF. The adenosine A2 receptor agonist DPMA (10 μM) inhibited the ability of LPS to activate myocardial TNF by 94% (n = 7, p < 0.05). Iodotubercidin (10 μM), which increases endogenous adenosine concentration, also inhibited TNF expression in trabecular muscle by 99% (n = 7, p < 0.05). Immunohistochemistry identified the myocyte as a primary source of TNF in the failing human heart.

Conclusion: Adenosine can significantly diminish TNF levels in the failing human heart and may provide a new pharmacologic approach in CHF.

2:15

851-2 Increased Protein Kinase C Expression in Failing Human Heart

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Background: The aim of the study was to determine protein kinase C (PKC)-β1 and β2 expression in failing and nonfailing myocardium to ascertain if re-expression of PKC-β is a marker of heart failure.

Methods: Explanted hearts of patients with idiopathic dilated cardiomyopathy (DCM) or coronary artery disease (CAD) were examined for PKC-β content by Western blot, *in situ* hybridization, immunostaining, and enzymatic activity, and compared with nonfailed (NF) left ventricle (LV) from hearts rejected for transplant.

Results: Western blots showed that PKC-β was significantly increased in membrane fractions of failed hearts (n = 12) compared to NF (n = 11) (β1: 76 ± 7 vs. 49 ± 9 units, P < 0.04; β2: 78 ± 9 vs. 52 ± 4 units, P < 0.02); there were no differences between DCM and CAD failed hearts. Immunostaining