endocardium covered by APA alternans increased significantly from 14.15±6.5% at 1Hz to 32.97±7.3% at 5Hz (Fig.1E).

**Conclusion:** TWA can serve as a biomarker of dynamic changes in ischemic substrates and localized APA and APD alternans underlie arrhythmogenesis in chronic MI ovine hearts.

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**PO-645-06**

**EPICARDIAL ELECTRICAL HETEROGENEITY AFTER AMIODARONE TREATMENT INCREASES VULNERABILITY TO VENTRICULAR ARRHYTHMIAS IN A SWINE MODEL UNDER THERAPEUTIC HYPOTHERMIA**

Chin-Yu Lin MD; Ting-Yung Chang MD; Yu-Feng Hu PhD; Fa-Po Chung MD, PhD; Shih-Lin Chang MD, PhD; Li-Wei Lo MD, PhD; Yenn-Jiang Lin MD, PhD; Hung-I Yeh MD, PhD; Yi-Jen Chen MD, PhD; Yu-Cheng Hsieh PhD; Yu-Xuan Wu and Shih-Ann Chen MD

**Background:** Amiodarone is commonly used during therapeutic hypothermia (TH) following cardiac arrest due to ventricular arrhythmias.

**Objective:** Electrophysiological changes and proarrhythmic risk after amiodarone treatment have not yet been explored in TH.

**Methods:** Epicardial high-density bi-ventricular mapping was performed in pigs under baseline temperature (BT), TH (32-34°C), and amiodarone treatment during TH. The total activation time (TAT), conduction velocity (CV), local electrogram (LE) duration, and wavefront propagation from pre-specified segments were analyzed during sinus rhythm (SR) or right ventricular (RV) pacing (RVP), along with tissue expression of connexin 43. The vulnerability to ventricular arrhythmias was assessed.

**Results:** Compared to BT, TH increased the global TAT, decreased the CV, and generated heterogeneous electrical substrate during SR and RVP. During TH, the CV reduction and LE duration prolongation were greater in the anterior mid RV than in the other areas, which changed the wavefront propagation in all animals. Compared to TH alone, amiodarone treatment during TH further increased the QTc/TPeak-Tend, TAT and LE duration and decreased the CV in both the global heart and regional areas during SR and RVP. Heterogeneous conduction was partially attenuated after amiodarone treatment. After TH and amiodarone treatment, the connexin 43 expression in the anterior mid RV was lower than that in the other areas, compatible with the heterogeneous CV reduction. The animals under TH and amiodarone treatment had a higher incidence of inducible ventricular arrhythmias than those under BT or TH.

**Conclusion:** Electrical heterogeneity during amiodarone treatment and TH was associated with vulnerability to ventricular arrhythmias.

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**PO-645-07**

**SLEEP-RELATED CHANGES IN CARDIOVASCULAR AUTONOMIC REGULATION IN APOE KNOCKOUT RATS: NEURAL MECHANISM FACILITATING ARRHYTHMIA IN HYPERCHOLESTEREMIA**

Wei-Lun Lin PhD; Li-Wei Lo MD, PhD; Chun-Ting Lai; Yu-Hui Chou MS; Shin-Huei Liu MD; Wen-Han Cheng MD; Tsung Ying Tsai MD; Cheryl C.H. Yang; Terry B.J. Kuo and Shih-Ann Chen MD

**Background:** The plasma levels of total cholesterol and low-density lipoprotein (LDL) cholesterol are important risk factors for coronary heart disease. Several reports suggest that elevated plasma cholesterol also leads to fetal arrhythmias.

**Objective:** We aimed to evaluate the LDL cholesterol and its relationship to cardiac autonomic activity, especially its relationship to sleep cycle.

**Methods:** Wireless transmission of polysomnographic recording was performed in wild type (n=5) and ApoE knockout (hypercholesteremia with elevated LDL-C, n=4) rats during 24 hours. Spectral analyses of the electroencephalogram (EEG) and electromyogram (EMG) were evaluated to define active waking (AW), quiet and paradoxical sleeps (QS, PS). Cardiac autonomic activities were measured by analyzing the power spectrum of heart rate variability (HRV).

**Results:** In the HRV analysis, we found that the LF/HF ratio and LF significantly increased in ApoE knockout rats during QS stage compared to that in normal rats (Fig A and C). The decreased QS times were noted in ApoE knockout rats compared to the normal rats (Fig E). Cardiac autonomic activities were measured by analyzing the power spectrum of heart rate variability (HRV).

**Conclusion:** We found that sympathetic hyperactivity developed in ApoE knockout rats, especially at QS stages. Those findings suggest that autonomic dysfunction is a possible mechanism for impairment of sleep quality and increased sudden cardiac death risk during QS stage in hypercholesteremia.