greater involvement of outflow and inflow tracts compared with RV apex and evidenced a lesser involvement of septal wall compared with free wall. In all patients concordance was noted between EA mapping results and non-invasive morphological-functional RV evaluation.

Conclusions: voltage mapping appears to be a promising method to study ARVC.

14. VENTRICULAR FIBRILLATION & OUT-OF-HOSPITAL CARDIAC ARREST

14.1 THE CHAOS THEORY AND ELECTROPHYSIOLOGY IN VENTRICULAR FIBRILLATION

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Introduction: Chaos theory is particularly attractive to those, who are studying ventricular fibrillation, because of the way it describes complex phenomena.

Methods: The study group consisted of 15 rabbits, which were anesthetized (midazolam dose 0.75 mg/kg/h) and then ventricular fibrillation (VF) was induced: in 5 cases by pacing transesophageal electrode (12 V, 50 Hz); in 5 by epinephrine (1mg/kg) and in 5 by calcium intravenously (0.001/kg). After 5 minutes of cardiac arrest defibrillation shock was delivered. If defibrillation was unsuccessful the cardiopulmonary resusculation was started. ECG data were analyzed using a numerical technique, based on discrete signal wavelet packet analysis of determining chaotic states of a signal.

Results: Transition from a non-chaotic state to a chaotic state manifested itself in the magnitude and distribution of system response wavelet expansion coefficients. No features connected with chaotic processes were observed during sinus rhythm and ventricular fibrillation. Features connected with chaotic processes were associated only with ventricular tachycardia.

Conclusions: 1. The standard EKG registration does not allow the analysis of developed chaotic process in myocardium during ventricular fibrillation. 2. Chaotic characteristic could be found in ventricular tachycardia.

14.2 NEW MODEL OF VENTRICULAR FIBRILLATION

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We developed a new model of ventricular fibrillation (VF) in the isolated rabbit heart, to produce a more efficient and stable arrhythmia. We also observed the effects of increased extracellular calcium in the stability and reversibility of VF. A classical Langendorff preparation was used. VF was induced by electrical stimulation (2.0 mA; pulse = 3 ms; 50 Hz; 10 V; duration of stimulation = 5 minutes). The hearts were divided into two groups, with different concentrations of calcium in the perfusates. All hearts in both groups fibrillated with this method. In the group with higher extracellular calcium there was no reversion to sinus rhythm after addition of antiarrhythmic drugs. VF was induced to the other group were allowed to fibrillate for 5, 20 or 30 minutes before addition of antiarrhythmic drugs, which induced reversion of VF in all hearts.

Our high rate of success and the exceptionally stable and long-lasting VF (longer then all studies ever published in rabbit hearts) turn our model very effective for studies of antiarrhythmic interventions in the isolated rabbit heart.

14.3 DOES ATRIAL FIBRILLATION BEGET VENTRICULAR FIBRILLATION?

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AF during MI is associated with increased mortality, the cause remains uncertain. We postulated that AF might trigger ventricular arrhythmias by bombarding the vulnerable post infarct ventricle with rapid extra-stimuli. We studied 500 consecutive MI patients. 124 patients with AF [67 onset prior to MI (chronic), 57 onset post MI (new onset)], 376 without AF. AF patients demonstrated higher mortality (53% versus 23%, p = 0.001) and an increased incidence of VF (13% versus 6.4%, p = 0.013). Co-morbidities could cause the increased risk and univariate analysis identified significant correlations with VF for age (p < 0.001), diabetes (p = 0.003), infarct size (p =0.043) and AF (p = 0.001). However multivariate analysis showed that only AF was significantly and independently correlated with VF. The correlation was stronger with chronic AF (p < 0.001) than new AF (p = 0.034).

Conclusions: This is the first evidence that AF may precipitate VF in post infarct patients, which may cause some of the increased mortality. Chronic AF is more significant than new AF, which is an important observation.

14.4 THE CHANGING LANDSCAPE OF VENTRICULAR FIBRILLATION IN CARDIAC ARREST

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Objective: To define the apparent changes in ventricular fibrillation (VF) as cause of out-of-hospital cardiac arrest. (OHCA)


Results: In the study period there were 338 all-cause arrests, with 203 (57%) in homes, 85 (24%) in public locations, and 69 (19%) in other locations (hotels, nursing homes). VF incidence during 1991-1997 was 24/100 000/person-year and during 1998-2004 it was 11/100 000/person-year (p<0.001). During 1991-1997, 61/110 (55%) of arrests in homes were in VF and from 1998-2004, 32/93 (34%) were in VF (p=0.003). During 1991-1997 48/51 (94%) of arrests in public places were in VF and from 1998-2004 22/34 (64%) were in VF (p=0.001).

Conclusion: VF as the cause of OHCA declined dramatically in both home and public settings, both in absolute numbers and in percentage of initial rhythm. This decreased incidence has obvious implications for potential cost-effective and therapeutic benefit from placement of automated external defibrillators.

14.5 TWO YEARS OF EXPERIENCE IN AN EARLY DEFIBRILLATION PROJECT

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Early defibrillation projects have shown their efficacy increasing survival in cases of sudden death. In Fano in 2002 we established “Un Cuore che Rinascce – Città di Fano” (UCR) an early defibrillation project based on first-responder volunteers using semiautomatic external defibrillator (AED). 14 mobile positions were located in police, city police, carabinieri, Italian Red Cross volunteers’ vehicles. 5 AED were placed in bathing establishments during summer and schools during winter.

310 lay volunteers were trained in AED use. Territorial network of AED is activated by local Emergency Medical System (EMS). During the first 24 months AED was applied in 31 cases. The first cardiac rhythm analysis shows: 9 asystoles, 7 pulsless electrical activity (PEA), 11 ventricular fibrillation, 1 ventricular tachycardia, 3 others. 12 “shockable” rhythms were treated with AED. 6 successfully, 6 unsuccessfully. 6 patients were resuscitated using AED, 5 of them were admitted in hospital, 1 died in the ambulance. Of the 5 patients admitted: 3 were discharged from the hospital, 1 of them with severe neurological damage; 2 patients died in hospital.

14.6 THE RESUSCITATION OUTCOMES CONSORTIUM (ROC): PURPOSE, PROGRESS & POTENTIAL

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The ROC consists of 10 regional centers in the USA and Canada, funded primarily by NIH for the purpose of conducting definitive trials of resuscitation strategies/therapies for out-of-hospital cardiac arrest and life-threatening trauma. During the 1st year of funding the organizational infrastructure has been maturing (including processes for measuring CPR components) and several protocols developed, including a combined study of hypertonic saline for trauma populations (hypovolemic shock and traumatic brain injury), a study of the Impedence Threshold Device for cardiac arrest patients, and a registry of all serious emergent medical out-of-hospital events. Over the initial 5 years of funding, we estimate the ROC has the potential to improve survival following out-of-hospital cardiac arrest from 5% to 10% and to reduce morbidity and mortality following trauma by 10 to 25%.