ΙΔΡΥΜΑ ΤΕΧΝΟΛΟΓΙΑΣ ΚΑΙ ΕΡΕΥΝΑΣ

ΕΡΕΥΝΗΤΙΚΟ ΙΝΣΤΙΤΟΥΤΟ ΧΗΜΙΚΗΣ ΜΗΧΑΝΙΚΗΣ
ΚΑΙ ΧΗΜΙΚΩΝ ΔΙΕΡΓΑΣΙΩΝ ΥΨΗΛΗΣ ΘΕΡΜΟΚΡΑΣΙΑΣ
Οδός Σταδίου, Ρίο, Τ.Θ. 1414, 265 04 Πάτρα
Τηλ.: 2610 965 300 & 3, Fax: 2610 990 987
www.iceht.forth.gr

ΣΕΜΙΝΑΡΙΟ

ΟΜΙΛΗΤΗΣ: Dr. Antonis A Armoundas
Biomedical Engineering
Harvard Medical School

ΘΕΜΑ: Translation, Translation, Translation: Impact of Basic Science in Clinical Cardiology

ΤΟΠΟΣ: Αίθουσα Σεμιναρίων ΙΤΕ/ΕΙΧΗΜΥΘ

ΗΜΕΡΟΜΗΝΙΑ: Τρίτη, 20 Δεκεμβρίου 2005

ΩΡΑ: 12:00

ΠΕΡΙΛΗΨΗ

Cardiovascular disease accounted for 40.6% (~950,000 lives) of all deaths in the US in 1998, and that figure accounted for more than the next three causes of death (cancer, accidents, HIV) combined. These numbers underlie the significance of: (i) identifying populations at risk, (ii) developing effective preventive strategies and (iii) introducing novel treatment modalities.

Despite the absence of any visible beat-to-beat changes of the T-wave, microvolt level T-wave alternans, an alternating pattern in the repolarization phase of the electrocardiogram, has been associated increased susceptibility to life threatening cardiac arrhythmias in patients with very diverse patho-physiological conditions.

While intracellular Ca^{2+} alternans is likely to give rise to action potential alternans and T-wave alternans, intracellular Ca^{2+} alternans may result from stress-induced deficiencies in any number of Ca^{2+} transport processes including Ca2+ entry into the cytoplasm, recovery of ryanodine receptors from inactivation and trigger of sarcoplasmic reticulum (SR) Ca^{2+} release, SR Ca^{2+} uptake and intra-SR Ca^{2+}
redistribution, and linking of intracellular Ca^{2+} handling to surface membrane voltage. Consequently, the mechanisms of cellular alternans may reside anywhere in this multi-step process. However, it is not possible to open the closed-loop system of the sub-cellular Ca2+ homeostatic mechanisms in the cardiac myocyte and study their individual effect on the action potential, without inadvertently perturbing the system under study.

In this seminar we will discuss a novel approach in dissecting the mechanisms underlying intracellular Ca^{2+} alternans-type oscillations and also provide a direct link between intracellular Ca^{2+} and cytosolic-membrane potential oscillations.

It is anticipated that pharmacologic and/or electrical therapies aimed to suppress alternans-type oscillations in the cardiac myocyte, are likely to eliminate conditions predisposing patients to increased risk of ventricular arrhythmias and sudden cardiac death.