In the last decade a series of large randomized trials have provided strong evidence that in the last decade a series of large randomized trials have provided strong evidence that in appropriately selected patients with left ventricular dysfunction, the use of ICDs improves overall survival at 2-5 years. Demonstrated efficacy of ICDs in primary prevention was initially established in patients with prvious myocardial infarction and left ventricular dysfunction (MADIT, I, MUSTT, MADIT II trials) and was then extended to patients with left ventricular dysfunction and heart failure (NYHA class II and III) of either ischemic or non-ischemic etiology (SCD-HeFT trial). However, although randomized clinical trials on the role of ICD in patient with left ventricular disfunction/heart failure included a variable proportion of patients without prior myocardial infarction, affected by so called non ischemic cardiomyopathy, no single prospective randomized controlled trial on ICD therapy in non ischemic cardiomyopathy provided conclusive evidence of mortality reduction. A meta-analysis of randomized controlled trials has analysed among the 1854 patients with non ischemic Cardiomyopathy enrolled in five primary prevention trials. Pooled analysis suggests a significant reduction in total mortality among patient randomized to ICD (or CRT-D in COMPANION) vs medical therapy (RR = 0.69, 95% CI 0.55-0.87 , p = 0.002). Assuming a mortality of approximately 7% for year (averaged control group mortality of the 5 trials) and a 33% relative risk reduction, the absolute risk reduction for all-cause mortality is approximately 2% for year, with on NNT (number needed to be treated) = 25 at 2 years (to be compared with a NNT around 18 for ischemic Cardiomyopathy).

Arrhythmogenic right ventricular cardiomyopathy
Although ICDs are currently used for prevention of sudden death in high-risk patients with various types of underlying heart disease, most prospective studies have focused either on ischemic heart disease or non-ischemic dilated cardiomyopathy. However, ICDs are also currently used for other arrhythmogenic right ventricular Cardiomyopathy, which is characterized by a younger age on onset and a longer expected survival (once sudden death has been prevented). The efficacy and safety of implantable cardioverter/defibrillator (ICD) in the context of ppatients affected by rrhythmogenic right ventricular Cardiomyopathy have been reported in small, single-center studies and recently in larger observational registries. No prospective randomized trial has compared ICD therapy with dtruc therapy or catheter ablation in arrhythmogenic right ventricular cardiomyopathy.

In a large multicenter registry (DARVIN 1) which enrolled 132 patients with arrhythmogenic right ventricular cardiomyopathy patients who received an ICD because of either cardiac arrest or ventricular tachycardia with hemodynamic compromise experienced a high incidence of ventricular fibrillation/flutter (10% per year of follow-up) despite antiarrhythmic drug therapy, thus confirming that they were ideal candidates for ICD therapy. Patients presenting with unexplained syncope derived much benefit from ICDs because of the similar annual rate of ICD interventions (8T per year of follow-up). On the other hand, patients implanted because of ventricular tachycardia without hemodynamic compromise had a statistically significant better outcome. The low predictive value of programmed ventricular stimulation makes it application in risk stratification questionable.

NOTES BY SHARON BATES Email: sharon@anthonybates.org

G. Bonani - Non ischemic CM had originally studied ICD implants and found over half received appropriate shock. Could be due to sustained arrhythmias.
Disease with normal cardiac function have a higher risk of sudden arrhythmia death. ICD implant is more appropriate. ARVD 1 : 5000 in general population. ICD therapy for prevention (Corrado paper) 2003. Sycope was a good stratisfication to implantable defibrillater – single most important more strides required for ARVD.

9:00 In Athletes & the young
D. Corrado (Padua, I)

LECTURE ABSTRACTS

IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR THERAPY FOR PREVENTION OF SUDDEN DEATH IN PATIENTS WITH ARRHYTHMOGENIC RIGHT VENTRICULAR CARDOMYOPATHY /DYSPLASIA

DOMENICO CORRADO Email: domenico.corrado@unipd.it
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Division of Cardiology, Department of Cardiac, University of Padua, Padua, Italy

Background: Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) is a condition

associated with the risk of sudden death (SD).
Methods and results: We conducted a multicenter study of the impact of the implantable cardioverter-defibrillator (ICD) for prevention of SD in 132 patients (93 males and 39 females, age 40 ± 15 years) with ARVC/D. Implant indications were a history of cardiac arrest in 13 patients (10%), sustained ventricular tachycardia in 82 (62%), syncope in 21 (18%), and other in 16 (12%). During a mean follow-up of 39 ± 25 months, 64 patients (48%) had appropriate ICD interventions, 21 (16%) had inappropriate interventions, and 19 (14%) had ICD-related complications. Fifty-three (83%) of the 64 patients with appropriate interventions received antiarrhythmic drug therapy at the time of first ICD discharge. Programmed ventricular stimulation was of limited value in identifying patients at risk of tachyarrhythmias during the follow-up (positive predictive value 49%, negative predictive value 54%). Four patients (3%) died, and 32 (24%) experienced ventricular fibrillation/flutter that in all likelihood would have been fatal in the absence of the device. At 36 months, the actual patient survival rate was 96% compared with the ventricular fibrillation/flutter-free survival rate of 72% ($p < 0.001$). Patients who received implants because of ventricular tachycardia without hemodynamic compromise had a significantly lower incidence of ventricular fibrillation/flutter (log rank = 0.01). History of cardiac arrest or ventricular tachycardia with hemodynamic compromise, younger age, and left ventricular involvement were independent predictors of ventricular fibrillation/flutter.
Conclusions: In patients with ARVC/D, ICD therapy provided life-saving protection by effectively terminating life-threatening ventricular arrhythmias. Patients who were prone to ventricular fibrillation/flutter could be identified on the basis of clinical presentation, irrespective of programmed ventricular stimulation outcome.

Circulation 2003 Dec 23; 108(25): 3084-91, Epub 2003 Nov 24.

NOTES BY SHARON BATES Email: sharon@anthonybates.org

D. Corrado – Advances in the recognition of genetic disorder and associated to SCD.
> 50% of causes from HCM
32% of primary electrical disease
VF in context of structural CMPs
- ARVD, HCM, etc.

Channelopathies are common denominator
ICD is the only therapy for prevention of SCD
ICD therapy in HCM
Primary implantation 20% appropriate shocks in 5 years
Secondary[+] implantation 80% appropriate shocks in 5 years ([+]already had a SCD event)

Device related & procedural complications
20% inappropriate shocks
9% pocket infection
5% device malfunction
3.8% lead fracture
3.8% dislodgement
38% device related anxiety

Depression in common (due to restrictions in life)
ICD confers optimal protection
What about young competitive athletes
Risk in sports
- damage to device or lead
- Innumerable risk of Arrhythmias

Rate of death on athletics is 3 times higher than regular people. Only 11% of survival after ICD implant (Drezner's study). Sport induced disease progression despite ICD. HCM – exersice induced ischemia causes cell death heat. Molecular decomposition occurs with training.

9:15 Prevention and management of ICD-related complications
G. Ricciardi (Florence, I)

LECTURE ABSTRACTS

PREVENTION AND MANAGEMENT OF ICD-RELATED COMPLICATIONS
GIUSEPPE RICCIARDI Email: vpric73@hotmail.com

Pacemaker and defibrillator therapy is on the rise as a result of expanding indications. Unfortunately, this trend is associated with an increased number of cardiac device-related complications. Inappropriate ICD shocks, lead failure, device infection and vascular complications are not uncommon and may cause significant patient morbidity and mortality. Furthermore, the considerable variability in the approach to deal with device-related complications not infrequently leads to additional problems and complications. Proper implantation techniques, with primary consideration for prevention of complications and planning for future procedures, such as the upgrades, generator changes, or extractions, are essential. Proper use of sterile techniques, implementation of peri-operative antibiotics, minimizing the amount of implanted hardware and the length of the procedures, and careful consideration in high-risk patients will decrease the likelihood of infectious complications.
Coagulase-negative styphylococci and Stephylococcus aureus, which have been reported in 42% and 29% of case, respectively, are the leading pathogens of CDI. Most patients (98%) need to undergo complete device removal.
In the Madit II trial 83 (11.5%) of the 719 ICD patients had an inappropriate shock and this episodes constituted 184 of the 590 total shock episodes (31.2%). Frequent ICD shocks are detrimental for patient's well-being and represent a significant problem. Patients implanted with dual-chamber (DC) have less inappropriate shocks if compared to single-chamber (SC) implantable cardioverter defibrillator (ICD).
In patients with Hypertrophic Cardiomyopathy (HCM) ICD implantation is well established for sudden death prevention. In a multicenter registry after a FU of 3.7 years reported an incidence of appropriate discharges in patients with previous cardiac arrest of 10.6% per year, and 3.6% per year in primary prevention patients. However inappropriate discharges occurred in 27% of patients, mostly related to atrial fibrillation. In our Florence experience as of October 2007 out of total 59/772 patients (7.6%) ICD implanted (44 patients for primary and 15 for secondary prevention), ICD interventions appropriately terminated ventricular tachycardia/fibrillation in 14 patients (24%) (8 ICD for primary prevention and 6 for secondary prevention, with an incidence respectively of 6 and 8% per year). ICD complications (inappropriate discharge, infections or lead dislodgement) occurred in 11 patients (19%) and 4 additional young patients experienced moderate to severe depression. The problem of appropriate selection of patients in incompletely resolved, above all in young people.

NOTES BY SHARON BATES Email: sharon@anthonybates.org

G. Ricciardi – normal battery depletion is 3 years after implantation
ICD Failure causes
73% normal battery depletion
10% recall

Results are life threatening or can lead to death

Inappropriate shock seems larger problem to patients
19% experience inappropriate shock
27% inappropriate shock - Maron's study shows
19% inappropriate shock – Cecchi groups

Lead failure common mis-behaviors of ICD
Lead perforation another problem (developes 1 – 10 days after implant)

These save lives but doctors pay price with the problems caused.

9:30 Discussion
9:50 **P. Spirito** (Genoa, I), **B.J. Maron** (Minneapolis, USA), **M. Ackerman** (Rochester, USA), **D. Corrado** (Padua, I), **F. Cecchi** (Florence, I)

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NOTES BY SHARON BATES Email: sharon@anthonybates.org

P. Spirito – "Once you die suddenly – you are dead!"
So the weight of the problems must be considered with patients and families.
Appropriate setting of ICD and preliminary preparations prior to implants.
Not as much data in young patients and their complications.

B. Maron & M. Ackerman – Bethesda conference disqualify athletes with ICDs but it is not illegal to participate in sports with an ICD. Moving target with new ICD sports registry.

M. Ackerman – Include surgery as another option to reduce Sudden Death & its prevention options.

D. Corrado – open to this option in selected patients. Open to ablation for additional option.

P. Spirito – Genetic screening not done to stratify risks. Not available or cost effective at this time.

D. Corrado – How much exercise? Fibrosis is kind of damaging. Risk is proportionate to LV hypertrophy. Quality of life should always be taken into account, not a depressed person.

F. Cecchi – Had implanted ICD and genotyped patients (outside of decisions to implant) appropriate shocks occur more often with saccomere genotype HCM. Make up of race is changing the face of heart disease, too.

D. Corrado – Adjusting the need for continued research.

9:50 Young Investigator Awards. Selected poster presentation
10:10 **F. Cecchi** (Florence, I)

First place award went to Poster # 35 – Francesca Stillitano

POSTER ABSTRACTS

MOLECULAR AND CELLULAR REMODELING IN FAMILIAR HYPERTROPHIC CARDIOMYOPATHY: A STUDY IN HUMAN BIOPSIES

Francesca Stillitano
F. Stillitano[1], S. Ssuffredini[1], R. Coppini[1], L. Sartinani[1], F. Cecchi[2, 3], I. Olivotto[3], A. Mugelli[1], E. Cerbai[1]
[1]Center of Molecular Medicine C.I.M.M.B.A., [2]Department of Medical Surgical Critical Area, University of Florence, Italy; [3]Referral Center for Myocardial Diseases, Azienda Ospedaliera Universitaria Careggi, Florence, Italy

Purpose: Familial Hypertrophic Cardiomypathy (fHCM) is the most common of the genetic cardiovascular diseases, characterized by high rate mortality, especially in young people. Molecular mechanisms of underlying ventricular remodeling in fHCM, likely predisposing to fatal arrhythmias, remains largely unknown. The aims of this study in fHCM were twofold: (i) to investigate the functional and molecular abnormalities of f-channel (HCN), since previous studies demonstrated its overexpression in ventricular hypertrophy, and (ii) to assess the role of serotonin 5-HT_{2B} receptors, whose overexpression causes compensated hypertrophic cardiomyopathy in animal models. Methods: biopsies were obtained from fHCM patients with severe obstructive disease undergoing septal myectomy; undiseased hearts were not used for transplantation served as controls. The experiments were carried out by using patch-clamp technique for electrophysiological recordings, and TagMan Real Time PCR for relative quantification of HCN2/4 isoforms and 5-HT_{2B} receptor genes.
Results: Gene expression results demonstrated a significant increase of HCN2/HCN4 ratio in hypertrophied samples relative to the controls. Electrophysiological data demonstrated that f-current activities at more negative potential (Vh = -94 mV) compared to control ventricular myocytes (-84 mV). This shift is consistent with a relative over-expression of HCN2 isoform, since heterologous re-experssion of human HCN2 gives a current activating at more negative voltages (Vh = -92 mV) than HCN4 (-81 mV), 5-HT_{2B} mRNA was significant increased in fHCM with respect to the controls; functional coupling of 5-HT_2 was assessed by measuring the effect of the 5-HT_2 selective agonist alpha-metil-serotonin (alpha-Me5HT, 1 um, a) on action potential in single fHCM myocytes. Alpha-Me5HT increased the action potential duration, an effect which was reverted by wash-out.
Conclusions: Our results demonstrate that in fHCM, specific molecular mechanisms can be responsible

for the induction of an altered functional phenotype in the human heart. Understanding the molecular mechanisms underlying ventricular and septal remodeling could identify functional arrhythmogenic mechanisms and, importantly, entirely novel targets for effective pharmacological interventions aimed to prevent and treat fHCM.

Second place award went to Poster # 58 – H. Lie-Venema

POSTER ABSTRACTS

THE ROLE OF EPICARDIUM-DERIVED CELLS IN THE DEVELOPMENT OF NON-COMPACTION CARDIOMYOPATHY

H. Lie-Venema

H. Lie-Venema, M.M. Bartelings, A.C. Gittengerger-de Groot
Department of anatomy and Embryology, Leiden University medical Center, The Netherlands

Third place award went to Poster # 39 – Luca Pacini

POSTER ABSTRACTS

THE ROLE OF EPICARDIUM-DERIVED CELLS IN THE DEVELOPMENT OF NON-COMPACTION CARDIOMYOPATHY

Luca Pacini

L. Pacini[1], S. Sufffredini[4], R. Riaccavento[5], G. D;Amati[2], G. Lembo[3], E. Cerbai[4], P. Di Nardo[5], G. Frati[1], G. Ragona[1], A. Calogero[1]
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10:10 Coffee Break
10:30

NOTES BY SHARON BATES Email: sharon@anthonybates.org

During the break I discussed a meeting in Padua with Dr. Corrado. Took pictures with Dr. Maron and Dr. Olivetto. I was given great compliments by Dr. Olivetto for the work we are doing with screening work in the US.

Dr. Barry Maron & Sharon Bates – a rare moment together.

Dr. Barry Maron and Dr. Iacopo Olivotto – Mentor & Student

10:30 **Session 9**
12:06 **New Advances in CRT, LVAD, Heart Transplant and Regenerative medicine**
Chairmen: **F. Cecchi** (Florence, I), **M. Marzilli** (Pisa, I)

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Referral Center for Cardiomyopathies, Cardiology 1, Heart and Vessels Department, Careggi University Hospital, Florence, Italy
MARIO MARZILLI Email: marzilli@med.unipi.it
Cardio Thoracic Department, Division of Cardiology, University of Pisa, Pisa, Italy

10:30 New directions for cardiomyopathies: What's next
L. Tavazzi (Padua, I)

LECTURE ABSTRACTS

NEW DIRECTIONS FOR CARDIOMYOPATHIES: WHAT'S NEXT

LUIGI TAVAZZI Email: l.tavazzi@smatteo.pv.it
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The estimates of the prevalence of the different forms of cardiomyopathies (CMP) are sparse, dated, defined in non representative settings, largely approximate and probably unreliable. These estimates are in striking contrast with the data reported in large HF Surveys in which the patients diagnosed as "idiopathic CMP" accounts for about 1/3. This is probably an overestimation due to a lack of accurate diagnostic investigation, but it may be an epidemiological flag of a sizable population of undiagnosed patients affected by some forms of myocardial diseases.
Nowadays there are several areas of interest to be explored in the CMPs domain
They include:

- Emerging burden and increasing clinical needs: thereby need of observational research and dedicated Registries.
- Need for continuous updating due to the rapidly expanding knowledge on CMPs
- Advent of molecular genetic diagnostics, that implies the need for clinical attention for healthy carriers and the need for time-consuming family screening and counseling.
- The implementation of clinical Units aimed at managing the diagnostic-therapeutic processes of patients with CMP, operating in the context of departments of general Cardiology.
- The inherited diseases prompt the unique opportunity to test treatments of properly designed randomized controlled trials in patients affected by a precisely defined disease both symptomatic or asymptomatic.

NOTES BY SHARON BATES Email: sharon@anthonybates.org
L. Tavazzi – The old position was that > 50% of children in a family affected by cardiomyopathies would be affected. Now the gross CMP phenotypes is > 70% of children in the family would be affected by HCM. Position Statement: Patient workup, genetic workup, Lab to bedside implementing knowledge aware of genotype limitations and complications.

10:54 Patient selection and expected benefits of cardiac resynchronization therapy
L. Padeletti (Florence, I)

LECTURE ABSTRACTS

PATIENT SELECTION AND EXPECTED BENEFITS OF CARDIAC RESYNCHRONIZATION THERAPY

LUIGI PADELETTI Email: elettrofisiologia@dfc.unifi.it
Institute of Internal Medicine and Cardiology, University of Florence, Florence, Italy

Rysynchronization therapy (RCT) with biventrical-pacemaker is a widely used tool for treatment of advanced heart failure (HF). As confirmed by many different studies and randomized clinical trials. RCT gives an additional benefit compared with drug therapy in terms of survival, reduction of hospitalizations for HF, quality of life and functional capacity, alone or associated with implantable cardioverter defibrillator (ICD). Latest European and American guidelines consider RCT as a level IA indication for treatment of NYHA class III and IV patients and optimal medical therapy (ACE-I and B-blockers), LV systolic dysfunction (FE $\leq$ 35%), sinus rhythm and evidence of ventricular dyssynchrony, identified by QRS complex duration $\geq$ 120 ms. It has been estimated that up to 3 % of dismissed patients after a hospitalization for HF, satisfy such criteria. At least 50% of them have a compelling indication for ICD. As indicated in a recent metanalysis (McAlister et al, Jama 2007), the benefit in terms of survivial for patients treated with RCT compared with controls, is already present at 6 months (NNT 29, to avoid 1 death for HF), and increases with time (NNT 13, at 2 years; NNT 9, at 3 years in CARE-HR extended trial). RCT is a technically easy and safe procedures; in 6123 patients, evaluated in 54 different studies, implantation was completed in 93%, the incidences of implantation related deaths was 0.3% and the incidence of pacemaker dysfunction was 5% after six months. This therapy has been proved economically sustainable: combined data from COMPANION and CARE-HF extended trials show an average cost of 7500 EURO per quality adjusted life-year gained. This economical advantage is reduced but still present with RCT is associated with ICD. A peculiar characteristic of this therapy is pharmacological therapy optimization, in particular with B-blockers, with synergistic benefit. However some problems still remain unsolved. In clinical practice the average patient who undergoes implantation is 10 years older than the average patient in the above mentioned clinical trials, and is also affected by comorbidity. Moreover, most of the implanting centers are not as experienced, with higher numbers of implanation-related complications. Last but not least is the absence of any clinical improvement from RCT in about 30$, of correctly implanted patients, although they had proved ventricular dyssnchrony, as requested by international guidelines. Finally the most controversial topic is the definition of ventricular dyssynchrony (now QRS duration $\geq$ 120 ms). This parameter has proved effective for the identification of possible responders, but there are evidence that RCT could be beneficial also for patients with narrow QRS and ventricular dyssinchrony identified by different means. At present the accurate definition of ventricular dyssynchrony and the best methods for its accurate identification, in order to reduce the number of non-responders, is still a challenge for all investigators. Many different parameters, mostly echographic, have been proposed as markers of dyssynchrony, but none of them has been validated by any reandomized multicentric clinical trial. In NYHA Class II and ventricular dyssyncchrony. Clinical trials are ongoing. In conclusion, RCT is a effective and efficient therapy for advanced HF patients (NYHA Class III and IV) with moderate-severe systolic dysfunction and proved ventricular dyssynchrony, notwithstanding optimal pharmacological therapy. However criteria for identification of optimal targets of this therapeutic approach are still investigated.

NOTES BY SHARON BATES Email: sharon@anthonybates.org

L. Padeletti – Double blind study done on a CRT on CRT off to determine in the reverse study the improvement with pacing. LVH remodeling was done but 1st Heart Failure was not improved on with variables.

Echo parameters are not being used for disease ?/? thorasic impetence is measure by the tip of the lead.

11:42 Gene therapy and stem cells: dream or reality?
R.J. Hajjar (New York, USA)

LECTURE ABSTRACTS

GENE THERAPY AND STEM CELLS: DREAM OR REALITY

ROGER J. HAJJAR Email: roger.hajjar@mssm.edu
Mount Sinai School of Medicine, New York, NY, USA

While progress in conventional treatment modalities has improved survival in patients with congestive

heart failure, the disease progresses relentlessly in these patients. Recent advances in understanding of the modecular basis of myocardioal dysfunction, together with the evolution of increasingly efficient gene transfer technology, has placed heart failure within reach of gene-based therapy. One of the key abnormalities in both human and experimental heart failure is a defect in sarcoplamic reticulum (SR) function, which is responsible for abnormal intracellular calcium handing. Deficient SR Ca2+ uptake during relaxation has been identified in failing hearts from both human and animal models and has been associated with a decrease in the expression and activity of SR Ca2+-ATPase (SERCA2a). Our work has led to the characterization of recombinant adeno-associated vectors (AAV) types 1, 6, and 9 as ideal for cardiac muscle gene transfer. In addition, we have developed new techniques for gene therapy trials including 1) a Phase ½ clinical trial of dose escalation of AAV1. SERCA2a administered percutaneously that will determine the optimal safe dose of this vector and will be powered to detect biological activity, and 2) a Phase ½ clinical trial dose of AAV6. SERCA2a administered surgically either at the time of or > 30 days following left ventricular assist device placement in patients with advanced heart failure. These human studies will be the first trials of gene therapy in heart failure using adeno-associated vectors and will test the hypotheses that restoring SERCA2 levels by gene therapy will improve ventricular function in patients with advanced heart failure.
The notion of replacing lost cardiomyocytes through cell implantation to attenuate or slow the deleterious structural and functional and remodeling is appealing and holds enormous therapeutic promise. Many different cell types have been implanted into injured myocardium including embryonic, fetal and neonatal cardiomyocytes, skeletal muscle sstem cells, fetal smooth muscle cells, and dermal fibroblasts. Recently bone marrow derived stem cells were shown to exhibit remarkable functional plasticity with the capacity to differentiate into cardiac myocytes. A number of recent clinical trials have shown that intracoronary infusion of adult progenitor cells in patients with AMI beneficially affects post-infarction remodeling processes. These trials will be reviewed in the setting of a new strategy for the treatment of congestive heart failure.

NOTES BY SHARON BATES Email: sharon@anthonybates.org

R.J. Hajjar – Cell therapy & gene therapy – treatments of heart failure. Permanent loss of myocyts need cell therapy. Calcium handling in a human heart. Dilated Cardiomyopathy – elong calcium handling lead to gene clinical / therapy work. Viral vectors for gene therapy. Partial size matters – small gets better profusion. Long term expression of genes is important in heart failure. Injection into heart through catheter lab on bi-pass. Spill over of virus was same in all trials. Improvement in MRG after study with injection. Improvements in LV size and BPN. Trials underway with HCM patients (only 4 now). New vectors to specifically target heart. Stem Cell: LV remodeling after infarction. Several variations of type and source of stem cells. 3 – 4% increase in Ejection Fraction after stem cell therapy. Induction of Pherapotent stem cells and cardiac progenitor cells – creates a personalized cell at time of transplant; induce cells within 17 – 30 days and for regenerative options.

12:15 Lecture – New advances in reverse remodeling and regenerative medicine
M. Yacoub (London, UK)

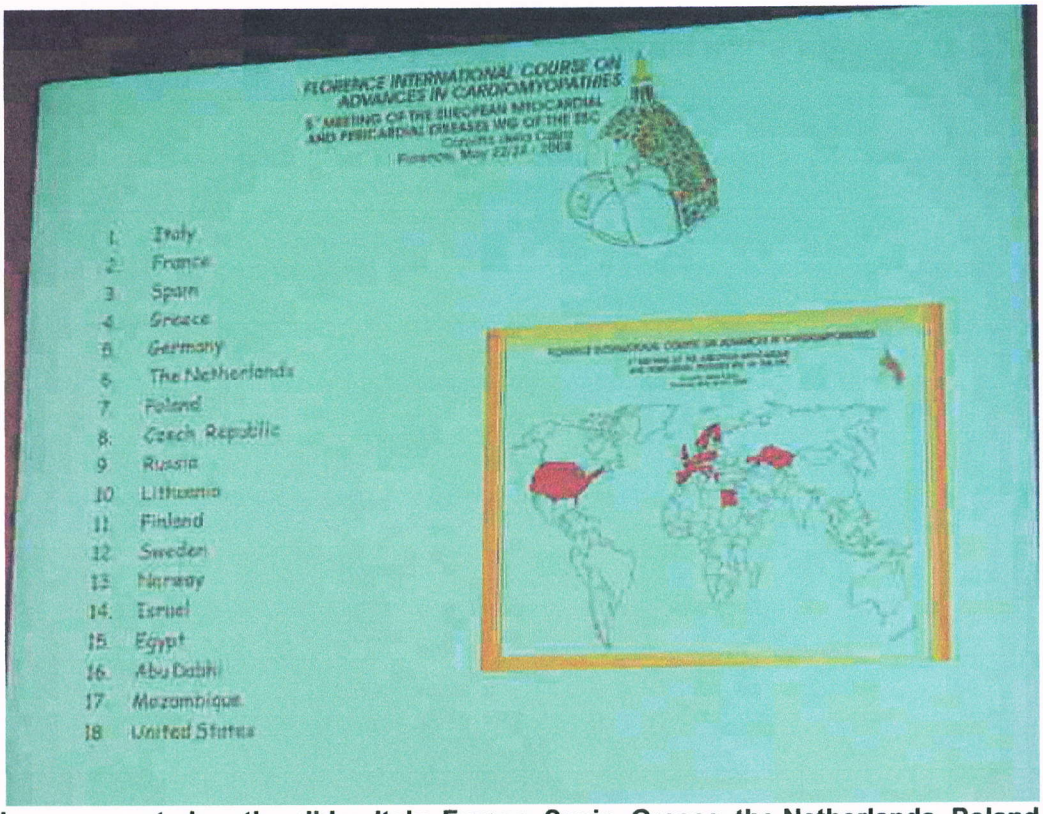
LECTURE ABSTRACTS

NEW ADVANCES IN REVERSE REMODELING AND REGENERATIVE MEDICINE

MAGDI YACOUB Email: m.yacoub@imperial.ac.uk
Imperial College, London, UK

NOTES BY SHARON BATES Email: sharon@anthonybates.org

M. Yacoub – From transplantation to regeneration and back. There is a shortage of donor organs and donor dysfunction / available. Profusion donor hearts with main blood help and new studies. Peter Hadaware (transplantation) grand father. 14,000 LV assisted devices have been implanted (5,000 last year) technologies have improved but some of materials are the same that were created in the 1960's. Challenges and opportunity of surgery. Stem cell therapy is new and further research in lab is needed. We must be cautious before we move into clinics. We must go back to the lab – any clinical application we should be careful. We must have discipline / freeze what we are doing, to do the work with dedication to the patients. He did the presentations for the awards to the esteemed colleagues. 23 countries were present at this conference.



Countries represented on the slide: Italy, France, Spain, Greece, the Netherlands, Poland, Czech Republic, Russia, Lithuania, Finland, Sweden, Norway, Israel, Egypt, Abu Dabhil, Mozambique, and the United States.

1:00 Closing remarks
F. Cecchi (Florence, Italy)

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NOTES BY SHARON BATES Email: sharon@anthonybates.org

F. Cecchi – Efforts for clinical trials must continue. Spread the culture!

Poster Sessions

P1 Unique epidermolytic bullous dermatosis with associated lethal cardiomypathy related to novel dismoplakin mutations.
A. Asimaki, P. Syruss, D. Ward, L.G. Gurereta, J.E. Saffitz, W.J. McKenna *(Boston, USA)*

P2 Endomyocardial Biopsy guided by electroanatomic voltage mapping in arrhythmogenic right ventricular cardiomyopathy.
A. Avelia, G. D'Amati, A. Pappalardo, F. Re, F. Laurenzi, P.F. Silenzi, P. De Girolamo, P. Baratta, E. Zachara, C. Tondo *(Rome, I)*

P3 African American RACE is associated with a higher prevalence of abnormal ECG partially related to higher incidence of early repolarization and sinus bradycardia.
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P4 Markedly increased prevalence of suspected hypertrophic cardiomyopathy in African American healthy teenagers undergoing screening echocardiography
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P5 Prevalence of suspected hypertrophic cardiomyopathy in healthy teenagers undergoing screening echocardiography
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P6 Obesity is associated with left ventricular hypertrophy and hypertension in healthy teenagers undergoing screening echocardiography
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P7 The majority of participants with suspected hypertrophic cardiomyopathy documented during screening echocardiography have normal ECG findings
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P8 Reported physical symptoms on a questionnaire during screening echocardiography is no associated with the presence of suspected hypertrophic cardiomyopathy
M.R. Movahed, S. Bates, A. Martinez, S. Sattur *(Phoenix, USA)*

P9 Prevalence of mitral valve prolapse and assocated valvular abnormalities in healthy teenagers undergoing screening echocardiography
S. Sattur, S. Bates, A. Martinez, M.R. Movahed *(Phoenix, USA)*

P10 Kinetics of force generation and relaxation in human cardiac myofibrils from FHC patient carrying the R403Q mutation in myosin heavy chain
A. Bellus, N. Piroddi, B. Scellini, C. Tesi, F. Girolami, I. Olivotto, M. Yacoub, F. Cecchi, C. Poggesi *(Florence, I)*

P11 Initial experience of contrast enhanced cardiac magnetic resonance imaging in patients with hypertrophic cardiomyopathy
S. Bongloanni, A. Chiribiri, A. Sibona Masi, I. Salvetto, M. Casetta, B. Gerhard, P. Angelino, R. Bonamini, M.R. Conte *(Turin, I)*

P12 Surgical treatment of midventricular obstruction associated with the subaortic obstruction in HOCM patients
K.V. Borisov, A.G. Starovoitenko, A.A. Diuzhokov *(Rostov, RUS)*

P13 Unbalanced GLA mRNAs ratio results in Fabry disease: a real time PCR study in fibroblasts
A. Caciotti, C. Filoni, L. Carraresi, R. Mignani, R. Parini, M. Filocamo, F. Saliani, L. Simi, R. Guierrini, E. Zammarchi, M.A. Donati, A. Morrone *(Florence, I; Rimini, I; Monza, I; Genoa, I; Reggio Emilia, I; Florence, I)*

P14 High correlation of LV ejection fraction with the extent of noncompaction in noncompaction cardiomyopathy; a new tool prognostic tool?