

**INTERNATIONAL SYMPOSIUM ON:
ADVANCES
IN
HEART FAILURE, CARDIOMYOPATHIES
AND PERICARDIAL DISEASES**
Trieste (Italy), May 25-27, 2017
Highlights

Introduction



Prof. Sinagra, chairman of the symposium, opened the congress, by highlighting the high scientific level of this meeting, dedicated to Heart Failure, Cardiomyopathies and Pericardial diseases. This congress was a very unique occasion for a full update on these topics and a moment of deep sharing among clinicians, translational and basic scientists coming from all the world together with the top Italian researchers of these fields and

many young physicians attending the University of Trieste and other Italian Universities regarding the most updated novelties on clinical aspects, genetics, devices, interventional cardiology, cardiovascular biology, regenerative medicine and bioengineering.

To follow the presentations of this congress, click on the link below:

<http://www.fondazione-menarini.it/Home/Eventi/Advances-in-Heart-Failure-Cardiomyopathies-and-Pericardial-Diseases/Video-Slide> ... and, after having logged in, enter in the multimedia area.

Pericardial Disease: from guidelines to clinical decision

Diagnostic Criteria for Pericarditis


Pericarditis	Definition and diagnostic criteria
Acute	Inflammatory pericardial syndrome to be diagnosed with at least 2 of the 4 following criteria:
Classical clinical criteria	1. Pericardial chest pain
	2. Pericardial rubs
	3. New widespread ST elevation or PR depression on ECG
	4. Pericardial effusion (new or worsening)
Biomarkers	Additional supporting findings:
Imaging to evaluate pericardial inflammation	Elevation of markers of inflammation (i.e. C reactive protein, erythrocyte sedimentation rate and white blood cell count) Evidence of pericardial inflammation by an imaging technique (computed tomography, cardiac magnetic resonance)
RECURRENT PERICARDITIS IF A SYMPTOM FREE INTERVAL > 4-6 weeks Or INCESSANT PERICARDITIS IF SYMPTOM FREE TIME < 4-6 weeks	Higher risk of constriction?

2015 ESC guidelines criteria; Table from Imazio M. Myopericardial Diseases Springer 2016

Pericardial Disease: from guidelines to clinical decision, was the topic discussed by Prof. Imazio in his lecture. The speaker, coming from Turin (IT), went deeper in his talk and presented very interesting data on diagnosis, etiology, triage and admission, anti-inflammatory therapy and finally on complications and prognosis. Speaking about diagnosis, Prof. Imazio presented very interesting data on the clinical manifestations of

pericarditis, by highlighting that the chest pain is present in more than 85% of patients. The main diagnostic criteria for pericarditis are chest pain, rubs, new widespread ST elevation or PR depression on ECG and effusion, the speaker pointed out. In the main part of his lecture, Prof. Imazio spoke about biomarkers and imaging and highlighted that in more than the 75% of patients affected by pericarditis, Troponin and EF/WMSI are normal. Talking about the pericarditis most common causes, the speaker highlighted that in the 55% of cases,

What are the most common causes?



	Pennacchi Mondino (Spain)	Zeytin (Spain)	Imazio (Italy)	Rostoff/Smith (Mexico)	Garcia (France)
Patients (n)	231	100	453	233	933
Years	1977-1981	1991-1993	1996-2004	1995-2006	2007-2012
Geographic area	Western Europe	Western Europe	Western Europe	Mexico	Western Europe
Idiopathic	109 (46.0%)	76 (76.0%)	377 (83.2%)	152 (65.2%)	516 (55.0%)
Specific etiology	32 (14.0%)	22 (22.0%)	76 (16.8%)	201 (86.3%)	417 (44.6%)
Neoplastic	3 (1.3%)	7 (7.0%)	21 (4.6%)	22 (9.4%)	85 (9.0%)
Tuberculosis	9 (3.9%)	4 (4.0%)	17 (3.8%)	16 (6.9%)	61 (6.5%)
Autoimmune	4 (1.7%)	3 (3.0%)	33 (7.3%)	12 (5.2%)	197 (21.1%)
Parasitic	2 (0.9%)	1 (1.0%)	3 (0.7%)	5 (2.1%)	29 (3.0%)

Lymphoma, Leukemia, SLE, Rheumatoid arthritis, Scleroderma, Sjogren syndrome + Post-cardiac injury syndromes

Reprinted from: Imazio M. Myopericardial Diseases Springer 2016

Complications and prognosis

- Recurrences in 20 to 30% of cases (pre-colchicine time) but halved by colchicine
- Risk of cardiac tamponade very low during follow-up if specific causes excluded (e.g. systemic inflammatory diseases, bacterial and neoplastic etiologies)
- Risk of constriction related to the etiology and not the number of recurrences (never reported in idiopathic recurrent pericarditis)



- 20-30% bacterial etiologies (TB, purulent)
- 2-5% neoplastic etiology, systemic inflammatory diseases, post-cardiac injury syndromes
- <1% viral or "idiopathic" pericarditis

Circulation. 2011;124(11):1270-5

pericarditis is an idiopathic form, neoplastic in the 5-10% of cases and less than 5% secondary to tuberculosis, but this form will increase in the future due to the immigration of people coming from region where tuberculosis is an endemic disease. Finally, Prof. Imazio spoke about therapy, by highlighting that aspirin and NSAID plus colchicine are the first line anti-inflammatory treatments.

- What are the main diagnostic criteria for pericarditis from the speaker point of view?
- What are the main differences between pericardial effusion and pericarditis based on the data presented by the speaker?
- What's about the triage of pericarditis?
- What's about the first line anti-inflammatory therapy, based on the data presented by the speaker?

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Acute Pericarditis: who is the patient at risk of recurrences?

PERICARDIAL DISEASES

- Acute pericarditis: intensely inflammatory disease (ESR and CRP), recurring in 20-40%, idiopathic (IL1) (85%), benign
- Symptomatic Pericardial effusions with normal PCR.
- Asymptomatic pericardial effusions

Prof. Brucato from Bergamo (IT), spoke about Acute Pericarditis: who is the patient at risk of recurrences? The speaker talked about diagnosis, risk factors in acute pericarditis, trigger factors and finally about treatment and tapering. Going deeper in his lecture, Prof. Brucato presented very interesting imaging data on diagnosis starting

PRECIPITATING FACTORS

- Upper respiratory viral infections
- Sore throat (Still Disease)
- Cold temperature (cryopirin)
- Physical activity (MINORITY of pts)
- > PRE-EMPTIVE TREATMENT? Eg. Ibuprofen 600 mg or indomethacin 25 mg x 3 for 1 week

from the acute pericarditis and the symptomatic and asymptomatic pericardial effusions. More in particular the speaker talked about the high-risk patients characterized by high fever, large effusion and lack of response to aspirin or NSAID and highlighted that the presence of elevated CRP after 1 week is an independent risk factor for recurrences and in these cases, it is necessary to go on with therapy till CRP are negative. Finally, the speaker

talked about therapy and more in particular about colchicine, NSAIDs and corticosteroids and highlighted that it is necessary to go on with steroids also at higher levels than the ones usually administered to patients in a cardiologic setting. In conclusion, Prof. Brucato pointed out that in case of incomplete response to aspirin/NSAIDs and colchicine, corticosteroids may be used, but they should be added at low to moderate doses to aspirin/NSAIDs and colchicine as triple therapy.

TRIPLE THERAPY: «the magic bullet»

In cases of incomplete response to aspirin/NSAIDs and colchicine, corticosteroids may be used, but they should be added at low to moderate doses to aspirin/NSAIDs and colchicine as triple therapy, not replace these drugs, in order to achieve better control of symptoms. Corticosteroids at low to moderate doses (i.e. prednisone 0.2–0.5 mg/kg/day) should be avoided if infections, particularly bacterial and TB, cannot be excluded and should be

1. NSAIDs at the RECOMMENDED HIGH dosages (indomethacin, ibuprofen, naproxen, ASA), INTRAVENOUSLY
2. colchicine: 0.5-1.0 mg, if tolerated (7% diarrhea)
3. steroids LOW DOSE (5-10mg) and then very slow tapering (months) never increasing again the steroid dosage

– REASSURANCE: don't panic; good prognosis, normal life

- What's about the major pericarditis precipitating factors based on the data presented by the speaker?
- What's about the high risk of recurrence, based on the data presented by the speaker?
- Who is the patient at risk of recurrences, based on the data presented by the speaker?
- What's about corticosteroids from the speaker point of view?
- What are the key points of the use of colchicine from the speaker point of view?
- What's about the triple therapy based on the data presented by the speaker?

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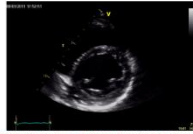
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Echocardiographic assessment of pericardial disease

ECHO ASSESSMENT OF PERICARDIAL DISEASES

Echocardiography in acute pericarditis

- Absence of pericardial effusion by echo **does not rule out pericarditis**
- A **very small amount** of pericardial fluid is normal (up to 30 mL)
- Trivial pericardial effusions may be visible only in systole



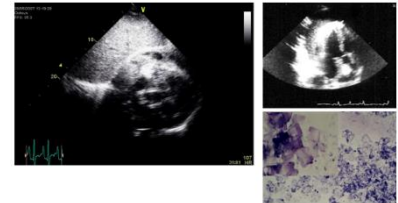
Department of Cardiac, Thoracic and Vascular Sciences, School of Medicine, University of Padua, Italy

Echocardiographic assessment of pericardial disease, was the topic Prof. Badano spoke about in his lecture. The speaker coming from Padua (IT), started his talk, by presenting very interesting data on echocardiography in acute pericarditis and highlighted that the absence of pericardial effusion does not rule out pericarditis. Talking about the semiquantitative assessment of the pericardial effusion, Prof. Badano pointed out that this symptom can be absent or in trace, small, moderate and finally severe

with a diastolic separation more than 20 mm. Going deeper in his lecture, Prof. Badano spoke about the differential diagnosis of the pericardial free space and presented very interesting imaging data on pericardial fat, hemopericardium, pneumopericardium and finally on a cholesterol pericarditis in a rheumatoid arthritis patient. In the main part of his presentation, the speaker talked about cardiac tamponade, by highlighting that this picture can occur in pericardial effusion of any cause and discussed the main echo and clinical signs of this picture. More in particular the speaker highlighted that the diagnosis of the cardiac tamponade is mainly based on the patient's

ECHO ASSESSMENT OF PERICARDIAL DISEASES

Differential diagnosis of pericardial free space



Cholesterol pericarditis in rheumatoid arthritis

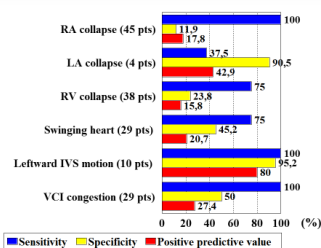


Department of Cardiac, Thoracic and Vascular Sciences, School of Medicine, University of Padua, Italy

clinical setting. Finally, Prof. Badano presented very interesting imaging data on the most important echo signs of the cardiac tamponade. Prof. Badano spoke also about pericardiocentesis, constrictive pericarditis and its differential diagnosis. In conclusion, Prof. Badano pointed out that echocardiography is the most important tool for assessing the spatial distribution and the functional significance of the pericardial effusion, but these data can be matched with the clinical signs for the best possible pericarditis diagnosis.

ECHO ASSESSMENT OF PERICARDIAL DISEASES

Cardiac tamponade. Echo signs vs. invasive hemodynamics



Department of Cardiac, Thoracic and Vascular Sciences, School of Medicine, University of Padua, Italy. Ristic AD et al. ACC 2001

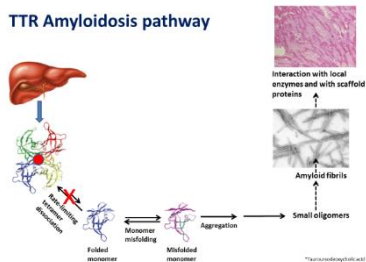
- Is the fluid quantity relevant for the diagnosis of cardiac tamponade from the speaker point of view?
- What are the main cardiac tamponade echo signs presented by the speaker?
- What's about pericardiocentesis, based on the data presented by the speaker?
- What are the most important differential diagnosis between constrictive and reactive pericarditis?

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Restrictive Cardiomyopathy and Amyloidosis

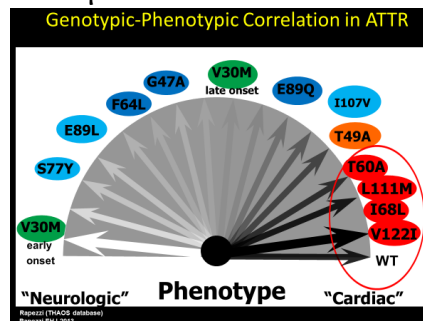
TTR Amyloidosis pathway



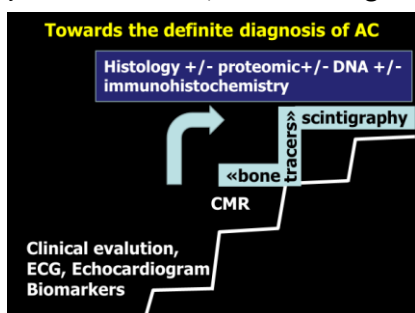
Restrictive Cardiomyopathy and Amyloidosis, was the topic at the core of the lecture discussed by Prof. Rapezzi. The speaker, coming from Bologna (IT), spoke about the TTR amyloidosis pathway leading to the onset of this disease and presented very impressive data on survival, by highlighting that yesterday the only possibility for treating these patients was the liver transplantation, but today we have other therapeutic

opportunities and the first one is to make visible amyloid to our immune system, the speaker pointed out. Going deeper in his lecture, Prof. Rapezzi spoke about cardiac amyloidosis as a restrictive cardiomyopathy and presented very interesting data on prevalence and the hemodynamic profile and highlighted that the prognosis depends only by the level of the heart failure without any involvement of the EF level.

In the main part of his lecture, the speaker talked about the amyloidotic cardiomyopathy and presented very interesting data on the chronic infiltrative CMP and on the acute toxic



myocardial disease due to circulating precursor protein in AL amyloidosis. Prof. Rapezzi presented also data on the TTR related amyloidosis, by highlighting that its diagnosis is based on the combination of Clinical evaluation, ECG, echocardiography, CMR, bone scintigraphy and histology, proteomic, DNA and immunochemistry examinations. In conclusion, the speaker pointed out that amyloidosis cardiomyopathy is no longer a rare disease.



- What's about histology as a diagnostic standard for CA, based on the data presented by the speaker?
- What are the main characteristics of the amyloidotic cardiomyopathy from the speaker point of view?
- What are the main therapeutical opportunities of the TTR amyloidosis, based on the data presented by the speaker?
- What are the key points of the TTR amyloidosis pathway presented by the speaker?

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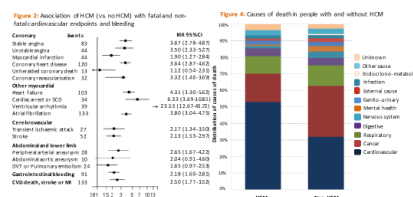
Multiparametric approach to sudden cardiac death risk stratification. The hope and the hurdles



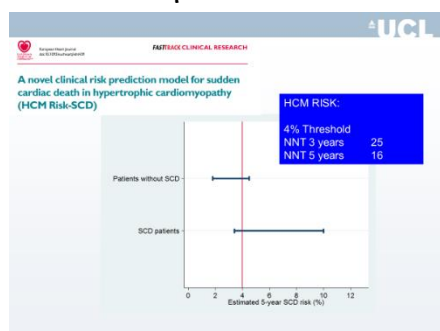
The Multiparametric approach to sudden cardiac death risk stratification. The hope and the hurdles was the topic at the core of the lecture discussed by Prof. Elliott. The speaker, coming from London (UK), talked about sudden death, its most common risk factors, the principle of risk stratification and the related limitations. Going deeper in his lecture, Prof. Elliott presented very interesting data on the first discovery of HCM and its relation to sudden death. More in

particular speaking about secondary prevention, Prof. Elliott pointed out that 1% of HCM patients die for sudden death. In the main part of his talk, the speaker presented very interesting data given by clinical studies on the identification and stratification of patients at risk for SCD, pointing to some limitations, like the binary nature of some risk factors and the influence of age. More in particular Prof. Elliott talked about the comparison between the data derived from well managed patients in clinical trials and those ones managed in the real clinical setting, by highlighting the profound differences between them. There is no evidence that the most important risk factors for sudden death in HCM patients differ in prognosis from a statistical point of view, the speaker pointed out. Speaking about the development of new biomarkers, Prof. Elliott pointed out that their performance basically depends on the technique used. Finally,

Prognosis of patients with hypertrophic cardiomyopathy: a contemporary population record linkage cohort in England
M. Pujades-Rodriguez^{1,2}, O. Guttmann¹, A. Gonzalez-Izquierdo¹, B. Duyx¹, C. O'Mahony¹, P. Elliott¹, H. Hemingway¹



Prognosis in a national sample of 1,160 people with hypertrophic cardiomyopathy vs. 11,204 age, sex, general practice matched people without hypertrophic cardiomyopathy. Cumulative incidence and 95% confidence intervals. Submitted for publication May 2017

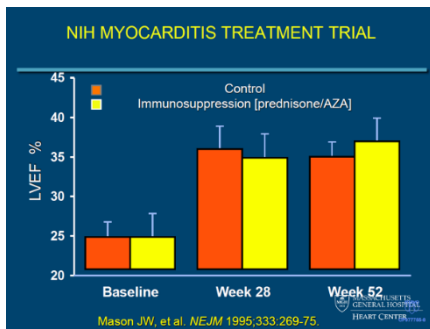


the speaker presented a new risk model, based on more than one risk factors like demographic, historical, ECG/Holter, Echo and talked about its validation. Prof. Elliott talked also about the limitations in the current prognostic factor research and about the phases of an affordable prognostic model development. In conclusion, the speaker pointed out that HCM is not a single disease and dividing this disease in more subtypes should be a valid method for a better prediction of the risk stratification.

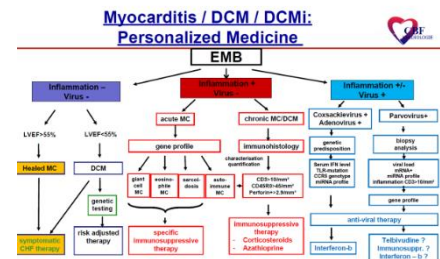
- How do we improve in the interpretation of the risks for Sudden death, based on the data presented by the speaker?
- What's about the prognostic factor research in hypertrophic cardiomyopathy?
- What's about the prognostic model presented by the speaker?
- What's about the novel risk prediction model for sudden death in HCM patients presented by the speaker?
- What are the phases of the prognostic model development presented by the speaker?
- Is HCM a single disease?

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Advances on antiinflammatory and immunosuppressive treatments

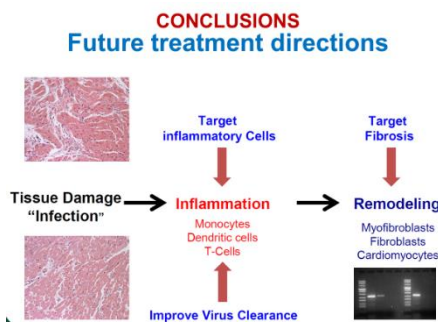


The Advances on antiinflammatory and immunosuppressive treatments was the topic of Prof. Ristić presentation. The speaker, coming from Belgrade (RS), talked about the ESC consensus 2013 treatment recommendations, by highlighting that immunosuppression should be started only after ruling out active infection on EMB by PCR and that steroid therapy is indicated in cardiac sarcoidosis.



Going deeper in his lecture, the speaker presented very interesting data on the conventional immunosuppressant like corticosteroids, antimetabolites, calcineurin inhibitors and mTor inhibitors. In the main part of his lecture, Prof. Ristić talked about immunosuppression for the biopsy-proven autoimmune myocarditis and presented very interesting data demonstrating that immunosuppression is able to dismantle the immunological machinery that fosters the myocardial inflammation and prevents the relapses and the evolution to DCM.

In the last part of his presentation, Prof Ristić talked about myocarditis and personalized medicine, by highlighting that immunosuppression may be considered in experienced centers for virus negative myocarditis with persistent heart failure or arrhythmia and ventricular dysfunction and presented also very interesting data on the future treatment directions.



- What's about immunosuppression from the speaker point of view?
- What are the main indications of the steroid therapy based on the data presented by the speaker?
- What's about azathioprine based on the data presented by the speaker?
- In case of immunosuppression for biopsy-proven autoimmune myocarditis, which patients should be treated from the speaker point of view?

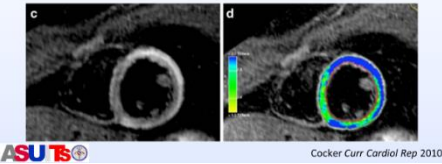
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The advancements of Cardiac Magnetic Resonance imaging in myocarditis

T2-weighted Imaging

- Inflammatory processes increase the amount of intra and extra-cellular free water.
- Protons in free water have increased T2 compared to bound water.
- T2-weighted sequences are able to detect localized edema.

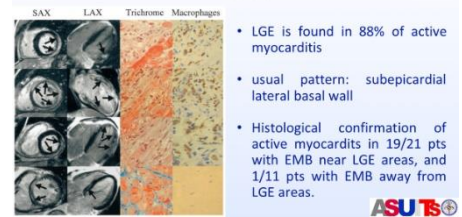


the speaker presented very interesting data about the diagnosis of myocarditis through the late gadolinium enhancement technique. Speaking about chronic myocarditis, Prof. Vitrella pointed out that the CMR varies according to the clinical presentations and the extent of the cell necrosis. In the second part of his lecture, the speaker presented very

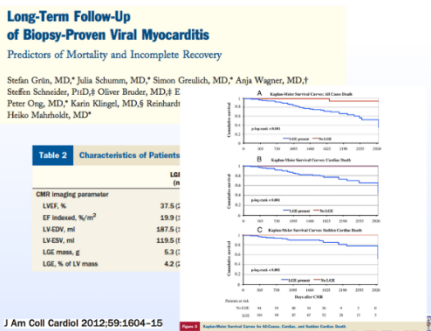
Prof. Vitrella coming from Trieste (IT) spoke about the advancements of Cardiac Magnetic Resonance imaging in myocarditis and presented very interesting data on the inflammatory phases of myocarditis and its main clinical presentations. Going deeper in his lecture, Prof. Vitrella spoke about diagnosis and presented very interesting imaging data on the T2 and T1 weighed sequences. In the main part of his talk, the

Diagnosis of Myocarditis

Cardiovascular Magnetic Resonance Assessment of Human Myocarditis: A Comparison to Histology and Molecular Pathology
Heiko Mahrholdt, Christine Goedecke, Anja Wagner, Gabriel Meinhardt, Anastasios Athanasiadis, Holger Voelgelberg, Peter Fittz, Karin Klingel, Reinhard Kandolf and Udo Sechtem
Circulation 2004;109:1250-1258; originally published online Mar 1, 2004;



interesting imaging data on the T1, T2 mapping and on the strain analysis, by highlighting that with T2 mapping there is a better diagnostic accuracy. Finally, the speaker talked about prognosis, by highlighting that the LEG technique is very effective in predicting the clinical outcomes. In conclusion, Prof. Vitrella, pointed out that cardiac MR is a useful imaging tool for a non-invasive diagnosis in myocarditis and that the LEG burden and pattern aids in prognostic stratification.

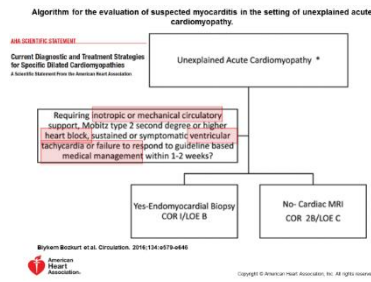


- What's about the diagnosis of myocarditis through CMR based on the data presented by the speaker?
- What's about the prognostic stratification?
- What are the main characteristics of the T2-weighted Imaging, based on the data presented by the speaker?
- What's about the application of the Global Relative Enhancement from the speaker point of view?

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The role of Endomyocardial Biopsy: a critical approach

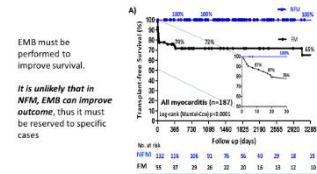


The role of Endomyocardial Biopsy: a critical approach, was the topic at the core of Prof. Ammirati presentation. The speaker, coming from Milan (IT), presented very interesting data starting from personal considerations on a diagnostic approach to myocarditis that does not necessarily reflects the expert' opinion. Going deeper in his lecture, Prof. Ammirati pointed out that

endomyocardial biopsy is a reference standard more than a gold standard for the diagnosis of myocarditis and presented very interesting data on the correct strategies to be implemented before asking for a EMB. In the main part of his lecture, the speaker talked about clinical cases and presented very interesting imaging data on patients affected by acute myocarditis and fulminant myocarditis and talked about the need for performing EMB only in the fulminant one. Finally, the speaker

NIGUARDA – San Matteo Experience 2001-2016

All types of ACUTE myocarditis:
Fulminant vs. non-fulminant presentation



HF at presentation

Direct comparison of the diagnostic capability of cardiac magnetic resonance and endomyocardial biopsy in patients with heart failure

Study	Sensitivity		Specificity		PPV	NPV	LR+	LR-
	CMR	EMB	CMR	EMB				
COU-16	81%	89%	100%	100%	100%	80%	4.3	1.75-16.7
ICM-16	87%	100%	100%	100%	100%	100%	9	0.40-100
CS-17	96%	100%	100%	100%	100%	100%	9	-
HEP-18	88%	100%	100%	100%	100%	100%	3	0.31-4.07

Table 1 Agreement of endomyocardial biopsy, cardiac magnetic resonance, or combined diagnosis with clinical data, echocardiogram, plus cardiac magnetic resonance with final diagnosis in 138 patients based on endomyocardial biopsy indication

Final diagnosis, n	Number	EMB diagnosis, n (%)	CMR diagnosis, n (%)	Combined diagnosis, n (%)
EMB indication (class)	1	1 (100)	1 (100)	1 (100)
Myocarditis, n	4	3 (75)	1 (25)	4 (100)

talked about other indications for EMB in different clinical scenarios, like HF with hemodynamic impairment or with new ventricular arrhythmias or with DCM of any duration and presented a very interesting clinical case of a young patient with a suspected diagnosis for fulminant myocarditis. In conclusion Prof. Ammirati pointed out that CMR should be preferred as first line exam in clinically suspected uncomplicated myocarditis and that EMB should be performed in the setting of clinically suspected myocarditis complicated by fulminant presentation-

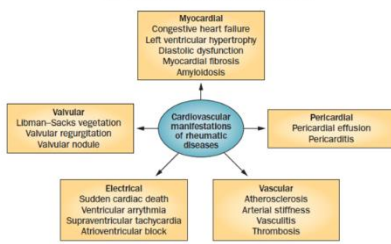
- What's about the algorithm for the evaluation of suspected myocarditis in the setting of unexplained acute cardiomyopathy presented by the speaker?
- What's about the indications to EMB in different clinical scenarios presented by the speaker?
- What's about EBM in fulminant myocarditis from the speaker point of view?

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Self-Host immune reaction in inflammatory heart disease: the heart of a matter

CV involvement in RDs

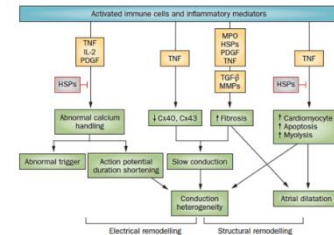


Nat. Rev. Cardiol. 12, 168–176 (2015)

Prof. Ferraccioli from Rome (IT), spoke about the self-Host immune reaction in inflammatory heart disease: the heart of a matter, by presenting very interesting data on three models: rheumatoid arthritis, systemic lupus erythematosus and systemic sclerosis. Going deeper in his lecture, Prof. Ammirati talked about the cardiovascular involvement in the rheumatologic diseases, by highlighting that the CV manifestations are at the core of the Rheumatologic diseases involving the CV system

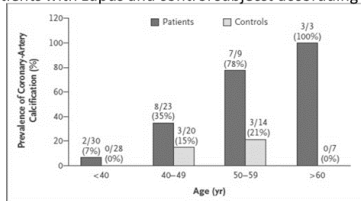
and presented very interesting data demonstrating that the processes affecting the vessels leading to the atherosclerosis lesions are the same affecting the joints in the rheumatoid arthritis setting. In the main part of his lecture, the speaker presented very interesting data demonstrating this tight correlation between CVD and RD and discussed a very interesting clinical case. Prof. Ferraccioli, presented also other data on the epidemiology of AF and stroke in RA by highlighting that AF is an inflammatory disease. Finally, the speaker talked about Lupus and the cardiac involvement, the myocardial insults and the role played by atherosclerosis in these patients. Finally, Prof. Ferraccioli talked about systemic sclerosis, by highlighting the presence of the endothelial damage in this disease, the heart involvement and the early myocardial

AF as an inflammatory disease



Nat. Rev. Cardiol. 12,230–243(2015)

Prevalence of coronary artery calcification among patients with Lupus and control subject according to age



Asanuma; NEJM; Dec 18, 200:

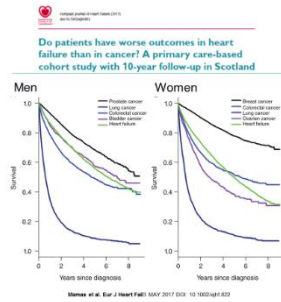
remodelling and presented very interesting data on the pathogenesis of the primary heart involvement. In conclusion, Prof. Ferraccioli pointed out that the primary cardiac involvement in progressive autoimmune chronic inflammatory disease usually remains silent for a long-time period, but is associated with a very poor diagnosis when symptoms become apparent and in all ACIDs the early control of active disease remains the key stone for controlling late cardiac damages.

- What's about the pathogenesis of the primary heart involvement in SS patients, based on the data presented by the speaker?
- What are the main therapeutic strategies in SS patients for preventing the onset of heart diseases from the speaker point of view?
- What's about the early myocardial remodelling in SS based on the data presented by the speaker?
- What are the main myocardial insults in patients affected by rheumatoid arthritis?

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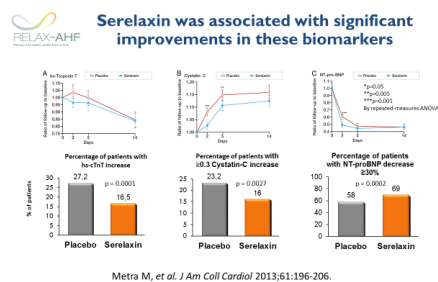
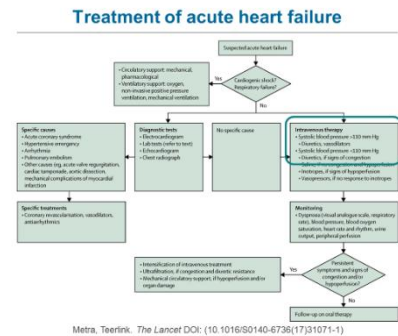
<http://www.fondazione-menarini.it/Home/Eventi/Advances-in-Heart-Failure-Cardiomyopathies-and-Pericardial-Diseases/Video-Slide...> and, after having logged in, enter in the multimedia area.

Acute Heart Failure: beyond clinical trial failures



Acute Heart Failure: beyond clinical trial failures, was the topic discussed by Prof. Metra from Brescia (IT), more in particular the speaker presented very interesting data on the treatment of acute heart failure. Going deeper in his lecture, the speaker talked about this topic starting from the specific causes and the related therapies mainly characterized by diuretics. More in particular, Prof. Metra pointed out that this disease is yet characterized by a very high rate of mortality. In

the main part of his talk, the speaker presented very interesting data on the diuretic response in AHF patients, on the effects of oral tolvaptan in hospitalized patients and on the effects of low-dose dopamine or nesiritide in AHF with renal dysfunction. Prof. Metra presented also other very interesting data comparing the outcome of different phenotypes of AHF hospitalized patients, by highlighting the tight correlation with the SBP levels. In the second part of his presentation, the speaker talked about vasodilators and their effects in these patients and presented very innovative data on ularitide in AHF decompensated



patients and its results on mortality. Finally, the speaker talked about relaxin, that is an insulin-like protein that contributes to maternal hemodynamic adaptations to pregnancy and presented very interesting data demonstrating that this drug produces a significant reduction in the risk of in-hospital WHF of about 47%. Prof. Metra spoke also about the results of the Relax-AHF-2 trial, by highlighting that the drug did not have any effect in people above 75 years old.

- Can we improve treatment of the patients hospitalized for acute heart failure based on the data presented by the speaker?
- What are the key points of the AHF treatment based on the data presented by the speaker?
- What are the main effects of the oral administration of tolvaptan in hospitalized patients with worsening HF?
- What is the mechanism of action of Relaxin based on the data presented by the speaker?

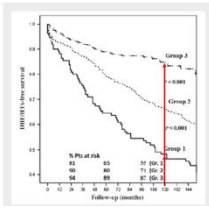
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Dilated Cardiomyopathy. How has changed the evolution of the disease

DCM: CHANGING MORTALITY

853 DCM PATIENTS (1978-2007) - Follow-up: 127,181 months (Merlo M, Sinagra G et al. EHJ 2014)



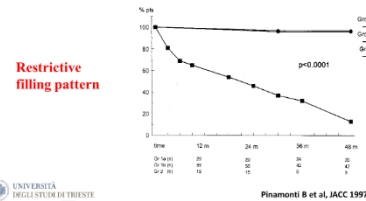
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Prof. Merlo from Trieste (IT), spoke about Dilated Cardiomyopathy. How has changed the evolution of the disease. More in particular, the speaker talked about the DCM mortality, by highlighting that in these year survival is dramatically improved. Going deeper in his lecture Prof. Merlo presented very interesting data on the DCM outcome improvement as well as in knowledge and talked about the etiological

classification, by highlighting that DCM is not a non-ischemic cardiomyopathy. Speaking about the etiologic classification, Prof. Merlo presented very interesting data on HHC, myocarditis, genetic cardiomyopathy and finally on the red flags approach characterized by the clinical, biohumoral, ECG, Echo, CMR evaluations. In the second part of his presentation, the speaker talked about DCM as a dynamic disease and the prognostic role of the medical therapy by highlighting the need for go beyond the left ventricular examination. The speaker presented very interesting data on the hemodynamic reverse remodelling involving the mitral regurgitation, the right ventricle, the diastolic dysfunction, all of them impacting on outcome. Finally, the speaker talked about specific DCM aspects like the need for a continuous

HEMODYNAMIC REVERSE REMODELING DIASTOLIC DYSFUNCTION



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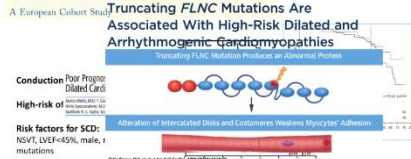
Pianomonti B et al, JACC 1997

ASUTS

reclassification of disease, the tight link between HNDC and DCM, the pediatric cases and open issues like the arrhythmic risk stratification, DCM genetics, the next-generation sequences and the genotype-phenotype correlation, very useful for prognosis and the target therapy in DCM. In conclusion Prof. Merlo pointed out that today the prognosis of DCM is dramatically improved till becoming a benign disease.

GENOTYPE-PHENOTYPE CORRELATION:PROGNOSIS

Risk Factors for Malignant Ventricular Arrhythmias in Lamin A/C Mutation Carriers



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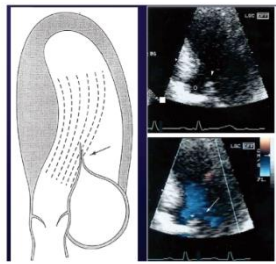
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- What are the main cornerstones of the improvement in outcome from the speaker point of view?
- What are the main topics of the red flags approach presented by the speaker?
- What are the main comorbidities in DCM patients based on the data presented by the speaker?
- How important is the familial screening in genetic cardiomyopathy from the speaker point of view?

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Pathophysiology and prognostic implication of Left Ventricular Obstruction in Hypertrophic Cardiomyopathy



Sherrid. J Am Coll Cardiol 2000;36:1344-1354.

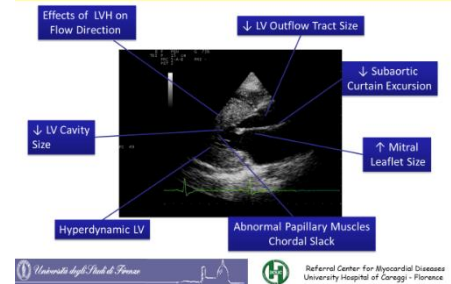


Referral Center for Myocardial Diseases
University Hospital of Careggi - Florence

The Pathophysiology and prognostic implication of Left Ventricular Obstruction in Hypertrophic Cardiomyopathy, was the topic discussed by Prof. Olivotto from Florence (IT), more in particular the speaker talked about the main echocardiographic pictures of this disease. Going deeper in his lecture, Prof. Olivotto talked about pathophysiology and highlighting the particular role played by flow velocity in the determination of the disease. The speaker talked

about also the role played by the reduction of the aortic valve size and the related higher pressure gradient. In the main part of his talk Prof. Olivotto presented very interesting imaging data on the role played by some abnormalities like the reduction of the LV cavity, the abnormal papillary muscles chordal slack, the increase of the mitral valve size, the reduction of the subaortic curtain excursion and finally the reduction of the outflow tract size, by highlighting that all these factors can contribute to the dysfunctional hemodynamic pattern more than the hypertrophy itself. The speaker talked also about the midventricular obstruction as a very rare abnormality, characterized by the worsening of the outcome more

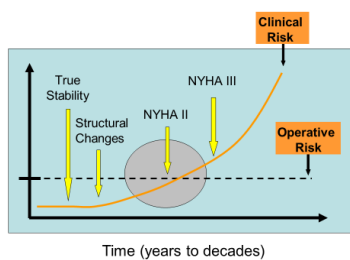
LVOT Obstruction in HCM: a Conspiracy of Mismatches



Referral Center for Myocardial Diseases
University Hospital of Careggi - Florence

in particular where associated with the presence of atrial fibrillation. In the last part of his lecture, Prof. Olivotto presented very interesting data on the effects of myectomy on the outcome of the HCM patients, by highlighting the need for the surgical approach to these patients in order to improve prognosis. Finally, Prof. Olivotto spoke about new therapeutic options aiming to the correction the underlying causal defects like MYK-461 and 491 and presented very preliminary clinical pharmacological data.

Surgical Timing



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University Hospital of Careggi - Florence

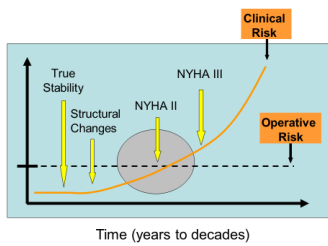
- What is the best surgical timing for HCM patients based on the data presented by the speaker?
- What's about the role played by the LV outflow in the evolution of the disease based on the data presented by the speaker?
- What are the main abnormalities leading to the LVOT obstruction in HCM patients from the speaker point of view?
- What's about the midventricular obstruction presented by the speaker?

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Surgical approach to obstructive Hypertrophic Cardiomyopathy

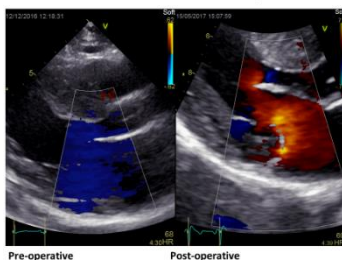
Surgical Timing



Università degli Studi di Firenze
 Referral Center for Cardiomyopathies
 University Hospital of Careggi - Florence

that the abnormalities of the mitral valve apparatus play a central role in the determination of the LV outflow obstruction and deeply discussed the main techniques applied in myectomy,

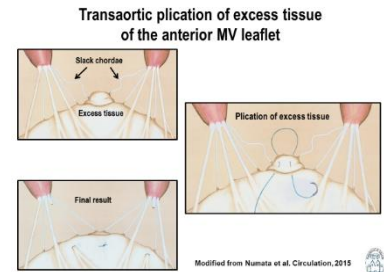
Transthoracic echocardiogram



Pre-operative Post-operative



Prof. Ferrazzi from Monza (IT), presented very interesting and impressive data on the surgical approach to obstructive Hypertrophic Cardiomyopathy. More in particular the speaker talked about the results of extended myectomy in obstructive class 3-4 HCM patients. Going deeper in his lecture, Prof. Ferrazzi, presented very interesting imaging data demonstrating



by highlighting that these procedures have to be tailored for any specific patient thanks the use of MRI. In the main part of his talk, Prof. Ferrazzi presented very interesting videos on the main parts of this operation and very interesting imaging data on patients after 3 months follow-up. In conclusion, the speaker pointed out that the mitral valve repair moves the mitral valve posteriorly, enlarges the LV outflow tract and contributes to abolish the outflow gradient.

- What's about the MRI pictures before and after myectomy in HCM patients based on the data presented by the speaker?
- What are the main procedures associated with the transaortic MV chordal cutting, presented by the speaker?
- What is the surgical approach presented by the speaker?
- What's about the results of the extended myectomy in obstructive HCM patients based on the data presented by the speaker?

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Microvascular dysfunction and Cardiomyopathies

Study	Method	n	MBF (ml/min/100g)	MBF (ml/min/100g)	CFR	Reference
Normal controls						
PET		3484	0.22±0.06	2.85±1.25	0.35±1.26	Gould et al ¹⁶
Primary LHM						
LVH		10	0.74±0.17	2.85±1.15	0.26±0.10	Reibel et al ¹⁷
PET		14	0.8±0.2	NA	6.1±1.0	Toni et al ¹⁸
Doppler		92	10.6±3.1 (normal)	81.2±17.8 (normal)	8.0±1.2	Holm-Guth et al ¹⁹
Secondary LHM						
PET		245	0.88±0.10	1.87±0.23	1.84±0.38	Gould et al ¹⁶
PET		25	1.2±0.36	1.8±0.26	1.5±0.44	Cocchi et al ²⁰
PET		21	1.54±0.21	1.5±0.22	1.0±0.10	Cocchi et al ²⁰
PET		81	—	1.8±0.09	—	DiCorleto et al ²¹
COF		25	0.58±0.25	1.87±0.26	2.28±0.5	Neglia et al ²²
PET		67	0.88±0.21	1.53±0.29	2.2±0.58	Neglia et al ²²
Myocardial infarct						
PET		25	0.88±0.13	1.87±0.23	NA	Neglia et al ²²
PET		25	1.2±0.36	1.8±0.26	2.1±0.74	Reibel et al ¹⁷
Aortic stenosis		20	1.19±0.25	1.8±0.28	1.9±0.5	Poligian et al ²³
PET		20	1.19±0.25	1.8±0.28	1.9±0.5	Poligian et al ²³
Doppler		28	23.3±10.1 (normal)	37.8±11.3 (normal)	1.7±0±0.5	Holm-Guth et al ¹⁹
Myocardial hypertrophy						
Doppler		81	28±8 (normal)	38±11 (normal)	2.1±0.5	Altaba et al ²⁴

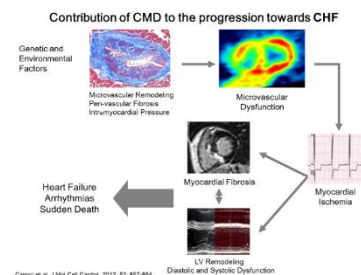
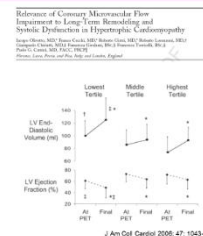
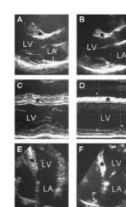
CFR, coronary flow reserve; COF, cardiac output; LVH, hypertrophic cardiomyopathy; LV, left ventricle; LVMI, left ventricular mass; MBF, myocardial blood flow during hyperventilation; MBF, myocardial blood flow at rest; NA, not available; PET, positron emission tomography; myocardial blood flow during hyperventilation; MBF, myocardial blood flow at rest; NA, not available; PET, positron emission tomography.

Lazzaroni E, Remaldi G and Camici P. Circulation 2015

The main topic at the core of Prof. Camici presentation, was the microvascular dysfunction and Cardiomyopathies. The speaker, coming from Milan (IT), presented very interesting data on the coronary microvascular dysfunction in primary cardiomyopathies. Going deeper in his lecture, Prof. Camici talked about the link between coronary ATS and myocardial ischemia and presented very interesting imaging data at a resolution less than 500 mm.

on the coronary tree, the one involved in the microvascular disease. In the main part of his talk, the speaker discussed the major pathophysiological mechanisms leading to the CM dysfunction, more in particular Prof. Camici, pointed out that there is no in vivo technique for the imaging of the coronary microcirculation and as a consequence, the maximum myocardial blood flow is an index of the microvascular function. The speaker presented very interesting data on the main defects of the myocardial blood flow in hypertrophic cardiomyopathy patients and talked about the vascular and the extra-vascular mechanisms

Severity of coronary microvascular dysfunction predicts adverse LV remodelling in HCM



Camici et al. J Mol Cell Cardiol. 2012; 52:857-864.

leading to the coronary microvascular dysfunction. In the second part of his lecture, Prof. Camici presented very interesting data on the relationship between the microvascular dysfunction and the HCM prognosis and the relationship between hypertension, LVH and the reduction on MBF. Finally, the speaker talked about the contribution of CMD to the progression towards CHF and presented a very interesting model explaining the pathway leading to CHF starting from hypertension through LVH.

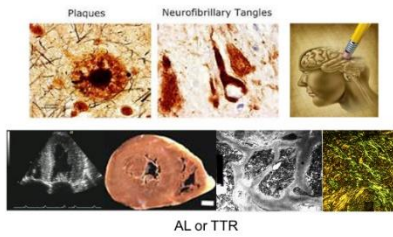
- What's about the model explaining the pathway leading to CHF starting from hypertension, presented by the speaker?
- What are the key points of the CMD contribution to the progression towards CHF?
- What's about the relationship between microvascular dysfunction and the HCM prognosis?
- What are the main mechanisms of the coronary microvascular dysfunction presented by the speaker?

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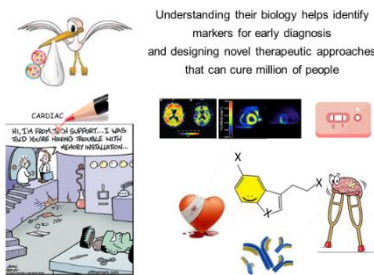
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Mind the Heart: Cardiomyopathy and Alzheimer's - a tangled web

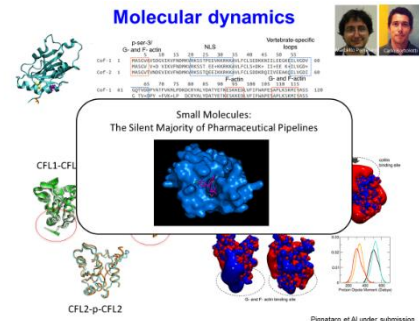
Amyloidosis



Prof. Del Monte talked about the main diseases linked with the production of amyloid like Alzheimer disease or others like cardiomyopathy, by highlighting that these diseases are not rare diseases, for amyloid is not rare at all, but are very important chronic diseases. In the main part of her lecture, Prof. Del Monte, presented very interesting data on those proteins that aggregate in DCM, like cofilin, its presence as cofilin 2 that is the inactive compound present in animal models and



Mind the Heart: Cardiomyopathy and Alzheimer's - a tangled web was the topic of Prof. Del Monte presentation. The speaker, coming from Boston (USA), presented very interesting data, starting from the predicting conformation from sequence and the energy landscape theory that explains the production of amyloid. Going deeper in her lecture



in human heart diseases like DCM. Finally, the speaker talked about the molecular dynamics of CFL2 studied with the intention to find new compounds able to counteract the effect of this protein and presented very interesting data on patients affected by Alzheimer disease and their connection with the diastolic dysfunction. In conclusion, Prof. Del Monte pointed out that understanding the biology of these two diseases helps to identify markers for the early diagnosis and the design of novel therapeutic approaches potentially

available for millions of people.

- Is amyloid a protein or a structure, based on the data presented by the speaker?
- What is the goal standard for the CFL2 detection in myocardial cells, based on the data presented by the speaker?
- What's about the correlation between Alzheimer disease and Heart alterations?
- What are the origins of the Alzheimer disease and the DCM from the speaker point of view?

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Autophagy and Inflammation in Dilated Cardiomyopathy



Dr. Beltrami from Udine (IT) spoke about Autophagy and Inflammation in Dilated Cardiomyopathy. Going deeper in his lecture the speaker presented very interesting data on a study designed to evaluate if proteostasis, autophagy and inflammasome activation may be involved in the pro-inflammatory status that characterizes DCM. More in particular Dr. Beltrami discussed the

main data of his study, starting from the patients' population, the presence of fibrosis and hypertrophy in DCM cardiomyocytes, the loss of proteostasis, the raise of the polyubiquitinated proteins, the presence of the aggresomes, the autophagy phenomena, the lysosomal dysfunction, the mitochondrial autophagy and dysfunction, the oxidative stress and the DNA damage in the DCM myocytes, the IL1 levels, the inflammasome activation and finally, the correlations of all these elements in DCM patients. In conclusion, Dr. Beltrami pointed out that a vicious circle of cell senescence characterizes DCM and the metabolomic data show a switch in the oxidative to glycolytic metabolism in these patients, therefore, the pharmacological stimulation of autophagy may have a positive impact in the progression of DCM

- What is the aim of the study presented by the speaker?
- What's about the patient population presented by the speaker?
- What's about autophagy?
- What's about the inflammasome activation based on the data presented by the speaker?

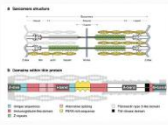
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Genetics in Cardiomyopathies. From bench to bedside

Titin in Dilated Cardiomyopathy

Titin (TTN) is the largest known protein in biology, spanning half the cardiac sarcomere and, as such, is a basic structural and functional unit of striated muscles. It is essential for heart development as well as mechanical and regulatory functions of the sarcomere. Next-generation sequencing (NGS) in clinical DCM cohorts implicated truncating variants in titin (TTN) as major disease alleles, accounting for more than 25% of familial DCM cases, but these variants have also been identified in 2-3% of the general population.



Ali M. Tabish et al 2017

Genetics in Cardiomyopathies. From bench to bedside was the topic at the core of Prof. Severini presentation. The speaker coming from Trieste (IT), talked about the genetic cardiomyopathies and the ACC/AHA classification. Going deeper in his lecture, Prof. Severini presented

very interesting data about hypertrophic and dilated cardiomyopathy and TTN that is the largest known protein in biology spanning half of the cardiac sarcomere with its truncating mutations accounting for more than 25% of familial DCM. In the main part of his lecture, the speaker talked about the HCM genetic diagnosis starting from the data published in 1989 and presented very interesting data on the main techniques developed for the diagnosis of the mutations leading to the clinical expression of the cardiomyopathies. More in particular Prof. Severini talked about the NGS technology and the different available platforms. In conclusion, Prof. Severini pointed out that the genetic testing can be useful in the diagnosis and in the screening and management of those family members like children and adolescents, who may not have yet manifested the clinical features of disease but may be at risk for sudden death.

Different NGS Platforms and Technology

Platform	Quantity	Read Length	Run Time	Sample/Run	Advantage	Disadvantage
SGS (Sanger)	Typical sequencing	~900	~9.5	~0.1M	Long read length	High error rate, expensive
454 (Pyro)	Typical sequencing	~100	~20-30	~1M	Long read length	High error rate, expensive
Roche (MSP)	Paired-end sequencing	~100-200	~4-10	~1.5M	Short read length	High error rate, low coverage
Ion Torrent	Paired-end sequencing	~100	~2-4h	~20M	Short read length	High error rate, low coverage
Roche/454	Paired-end sequencing	~100-1,000	~7-8 days	~1M	High throughput	Long run time
Roche/454	Paired-end sequencing	~100-1,000	~6 days	~200M	High throughput	Expensive, long run time

Genetic variants identification with PAN Cardiomyopathy Panel

TOTAL PATIENTS:	Pathogenic and likely Pathogenic	Genes	VUS	No mutations
36	16		31	9
	4	TTN		
	3	DSC2		
	2	MYBPC3		
	4	ELNC		
	1	NEXN		
	1	MYH7		
	1	SCN5A		
	1	DSG2		

- What are the main platforms used for the cardiomyopathies diagnosis?
- What's about the Trieste PAN CMP panel presented by the speaker?
- What are the main clinical NGS implications based on the data presented by the speaker?
- What are the main tools for the genetic variant interpretation presented by the speaker?

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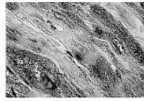
Left ventricular remodeling in Cardiomyopathies and Heart Failure: the heart break of success

Cardiac myocyte changes in LV remodeling and in its reversal

1. Cell hypertrophy
2. Reactivation of fetal genes
3. β -adrenergic desensitization
4. Loss of myofibrils and progressive disarray of the cytoskeleton
5. Changes in excitation contraction coupling
6. Modifications of myocyte metabolism

Remodeling

Loss of sarcomeric cross striation in the failing heart



De Jager et al. J Am Coll Cardiol. 2005; 46: 2028-2035

Washington University School of Medicine in St. Louis

Center for Cardiovascular Research

The Left ventricular remodeling in Cardiomyopathies and Heart Failure: the heart break of success was the topic at the core of Prof. Adamo presentation. The speaker coming from St. Luis (USA), talked about the heart remodelling in response to injury, the cardiac remodelling and its reversal at the cellular level, the myocardial remission compared to the myocardial recovery and finally about the clinical perspective. Going

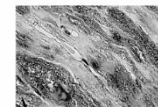
deeper in his lecture, Prof. Adamo presented very interesting data on the LV remodelling by highlighting that it is reversible in case of removal of the noxious stimulus or of initiation of therapy and these phenomena are characterized by changes at the cellular, histological and macroscopical level. In the main part of his lecture, the speaker talked about these changes in the cells, in the myocardial tissue and in the left ventricle and finally He pointed out that the reverse remodelling leads to an apparently

Cardiac myocyte changes in LV remodeling and in its reversal

1. Cell hypertrophy
2. Reactivation of fetal genes
3. β -adrenergic desensitization
4. Loss of myofibrils and progressive disarray of the cytoskeleton
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Remodeling

Loss of sarcomeric cross striation in the failing heart



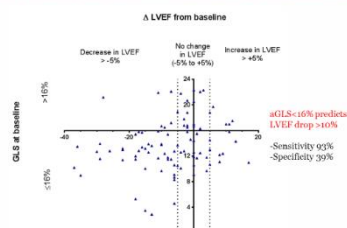
De Jager et al. J Am Coll Cardiol. 2005; 46: 2028-2035

Washington University School of Medicine in St. Louis

Center for Cardiovascular Research

normal heart, but this is not true. In the second part of his talk, Prof. Adamo presented very interesting data given by a clinical study running on patients with recovered LVEF, aimed to investigate if the global longitudinal strain of this patients can be indicative for myocardial recovery or remission. In conclusion, the speaker pointed out that patients with apparently healed heart must remain on therapy and under close surveillance and GLS may help in stratifying patients with fully recovered LVEF.

In patients with recovered LVEF, Global Longitudinal Strain correlates with increased chance of dropping LVEF during follow-up



Washington University School of Medicine in St. Louis

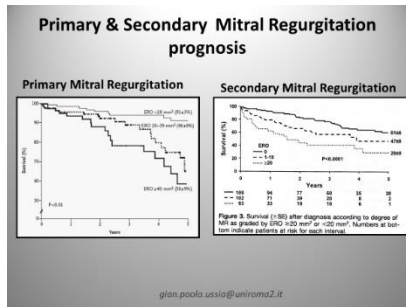
Center for Cardiovascular Research

- Is a reverse remodelled LV normal, and healed, based on the data presented by the speaker?
- What's about the model of the myocardial remission and myocardial recovery presented by the speaker?
- What are the main myocardial tissue changes in the LV remodelling and their reversals?
- What's about the cardiac myocyte changes in LV remodelling and its reversal from the speaker point of view?

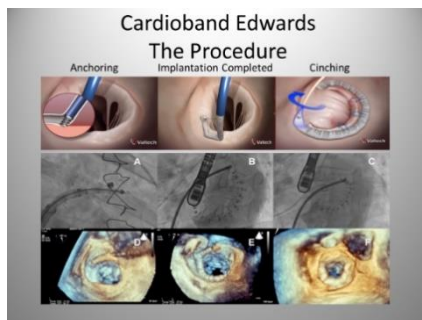
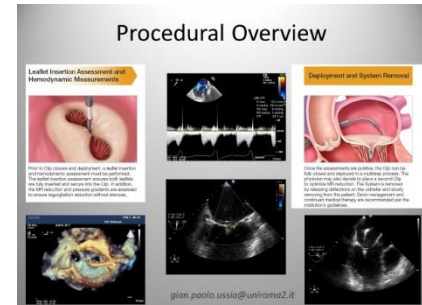
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Mitraclip in Heart Failure. Pitfalls and future perspectives



The Mitraclip in Heart Failure. Pitfalls and future perspectives was the topic of Prof. Ussia presentation. The speaker coming from Rome (IT), talked about the burden of this valve disease. Going deeper in his lecture, Prof. Ussia, presented very interesting data on aetiology and more in particular on the primary type characterized by the mixomatous or fibroblastic prolapse of the mitral valve and on the secondary mitral regurgitation, characterized by the valvular dysfunction without any anatomical lesion. In the main part of his lecture, the speaker talked about the functional MVR as a ventricular disease, its prognostic implications, prognosis and surgical treatment.



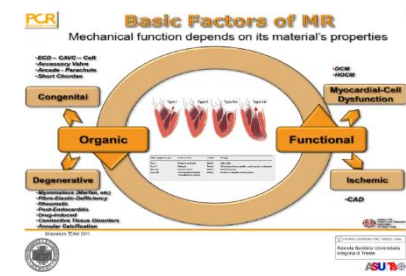
Prof. Ussia, presented very interesting data on the mytraclip system and its main skills, given by clinical studies running in patients submitted to mitral valvular repair. In the second part of his presentation, the speaker talked about the ESC/EACTS guidelines on the management of the valvular heart diseases and highlighted the advantages and pitfalls related to this procedure. Finally, Prof. Ussia talked about the future perspectives and more in particular about the Cardioband Edwards and the Pascal repair system procedures.

- What are the main the advantages of the mitraclip procedure presented by the speaker?
- What's about the pitfalls of the mitraclip procedure?
- What's about the mitraclip procedure in Europe based on the data presented by the speaker?
- What are the main results of the Everest II trial?
- What is the main mitraclip procedure presented by the speaker?

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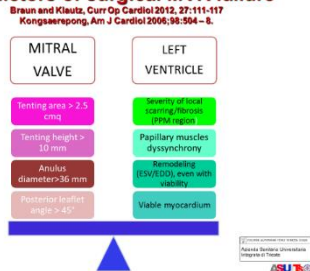
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Patients' predictors of efficacy for MitraClip. The best candidate for the best result.



data given by clinical studies on the long-term prognosis of medically treated patients with functional MR and LVD. More in

Predictors of surgical MVR failure



Patients' predictors of efficacy for MitraClip. The best candidate for the best result was the topic Dr. Stolfo talked about. The speaker coming from Trieste (IT), at the beginning of his talk spoke about the basic factors of mitral regurgitation and its prevalence in HF patients. Going deeper in his lecture, Dr. Stolfo presented very interesting data given by clinical studies on the long-term prognosis of medically treated patients with functional MR and LVD. More in particular the speaker talked about the goals of the mitraclip procedures and about the candidate patients and presented very interesting data on the results obtained in patients affected by primary and secondary MR. Finally, Dr. Stolfo highlighted that thanks to the mitraclip procedure there is a possible improvement in QoL and in reducing hospitalization but not in survival and in reverse remodelling.

Potential targets of interventional MR management

- Relief of symptoms
- Reduce hospitalizations
- LV reverse remodeling
- Hard endpoints (Death/HT)



- What are the main targets of the interventional MR management?
- What are the main predictors of MVR failure?
- What is possible to do after clip from the speaker point of view?
- What's about the evolution of the percutaneous valve repair therapy, based on the data presented by the speaker?

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FFR in multivessel disease and left ventricular dysfunction

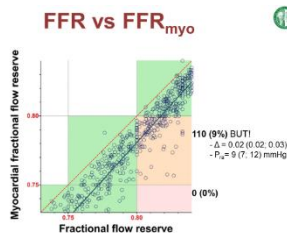
Fractional Flow Reserve 

$$\frac{P_{\text{distal}}}{P_{\text{aortic}}} = \text{FFR}$$


...during maximal hyperaemia


Thiele, May 2017

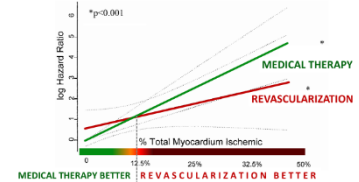
FFR in multivessel disease and left ventricular dysfunction was the topic Prof. Tóth talked about. The speaker coming from Graz, (A), spoke about FFR and its definition. Going deeper in his lecture, Prof. Tóth presented very interesting data on the complex cases and the FFR application and its applicability in HF patients. Talking about the complex clinical cases, the speaker discussed 3 very interesting clinical cases characterized by the presence of myocardial ischemia and main risk factors like hypertension, hypercholesterolemia, diabetes and others and presented very interesting data on the comparison between the results of medical therapy and FFR-guided PCI. In the second part of his presentation, Prof. Tóth talked about the



Thiele, May 2017

 application of FFR in HF patients and addressed the audience with this question: does the incorporation of the right atrial pressure have any impact on the calculation of the fractional flow reserve? In order to find a comprehensive answer, the speaker presented very interesting data on a clinical study running in HF patients with FFR measurements and highlighted that even in these patients FFR measurements are accurate.

Ischemia beats everything? 



Thiele, May 2017


Hellmuth et al. Circulation 2013

- What's about the FFR formula presented by the speaker?
- What's about the use of FFR in complex cases, based on the data presented by the speaker?
- What is the applicability of FFR in HF patients, based on the data presented by the speaker?
- How to estimate the stenosis diameter based on the data presented by the speaker?

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The effect of pre-hospital triage and therapy on the incidence of heart failure in STEMI patients who are candidates for primary angioplasty




Time Delays

	Transportation Delay	System Delay
Netherlands	25 min	85-110 min
Denmark	28 min	97-139 min
Ireland	30-60 min	145 min


**Delay due to Transportation only
20-30% of Total System Delay**

* APEX AMI trial, N=5745, >200 centers



Prof. van't Hof talked about the effect of pre-hospital triage and therapy on the incidence of heart failure in STEMI patients who are candidates for primary angioplasty. The speaker coming from Maastricht (NL), at the beginning of his lecture, talked about the components of delay in STEMI patients and the ideal intervals for intervention. Going deeper in his lecture, Prof. van't Hof presented very interesting


data on the time delay in transportation to hospital and its relationship with the system delay, by highlighting that the problem is not the transportation but the system delay. More in particular, the speaker talked about a Triage model characterized by the direct transportation to a PCI center when the health operators working on ambulance detect the presence of an ischemic lesion in their patients. Prof. van't Hof presented very interesting data comparing system delay and mortality and on the primary PCI and the intravenous

Primary PCI and Pharmacotherapy

Acute (Pre-Hospital) Phase

- Intravenous agents
 - Fibrinolytics
 - 2b/3a blockers
 - Heparin / Bivalirudin
 - Beta blockers?



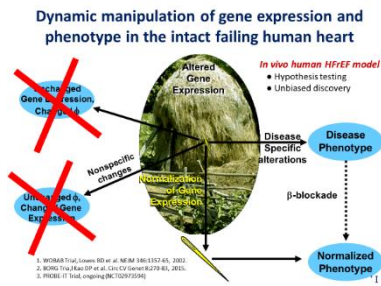
agents to be administered in these patients. Finally, the speaker talked about the early administration of oral drugs in these patients during ambulance transportation, like ticagrelor and beta-blockers. In conclusion, Prof. van't Hof pointed out that the time to treatment is the most important element able to reduce the infarct size and the evolution to HF and that the effective and safe “drugs” in the prehospital phase to improve ST resolution are GPI's, Ticagrelor and Beta-blockers

- What are the main components of delay in STEMI based on the data presented by the speaker?
- What are the main characteristics of the field triage presented by the speaker?
- What's about the correlation between system delay and mortality, based on the data presented by the speaker?
- What are the main intravenous agents, the speaker talked about to be administered in the acute pre-hospital phase?

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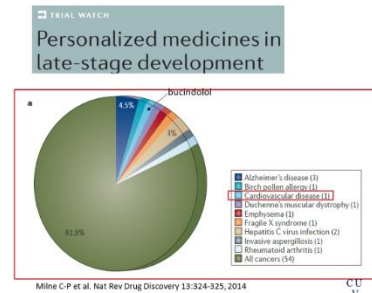
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Treatment of chronic Heart Failure: Future perspective from 30 years of knowledge.

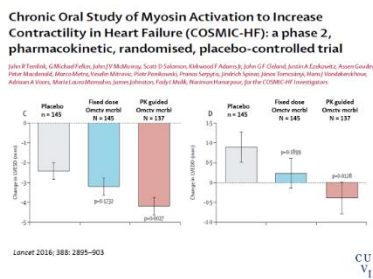


Treatment of chronic Heart Failure: Future perspective from 30 years of knowledge, was the topic at the core of Prof. Bristow presentation. The speaker coming from Anschutz (USA), at the beginning of his lecture, spoke about the rules of engagement of the treatment of CHF. Going deeper in his talk, Prof. Bristow presented very interesting data on the conceptual models of HF starting from the 60th till now and on the FDA drug approvals from 2000 to 2016. In the main part of his

lecture, the speaker talked about the possible approaches to improve the drug development success in HF. More in particular he presented a huge amount of data on 4 critical topics, like identifying and targeting central control nodes of biologic networks involved in remodelling, identifying and targeting therapeutic phenotypes in HF subgroups, investigating the basis of the non-response to the most effective therapies and finally identifying novel, druggable targets for the development of new effective therapies.



Speaking about central control nodes for remodelling, Prof. Bristow presented very interesting data suggesting the possibility to act on remodelling through the gene expression. In the second part of his lecture, Prof. Bristow talked about the identification and the targeting of therapeutic phenotypes in HF subgroups as a true precision medicine approach and presented very interesting data on the approaches to pharmacogenetic targeting like the GWAS and the candidate gene variant. In the last part of his lecture, Prof. Bristow talked about novel targets and the related drugs in development for the treatment of HFrEF, by highlighting that many of them are already in phase 2 and 3 clinical development and more in particular presented very interesting data on Omecamtiv mecarbil targeting the myosin activation.



- How extensive is the $\beta 1$ -AR signaling network for remodelling, based on the microarray data presented by the speaker?
- What is the general mechanism of the β -blocker induced improvement in myocardial function and reversal of remodelling, based on the data presented by the speaker?
- What are the precision medicine methods for identifying patients likely to respond to a cardiovascular treatment?
- What are the main criteria for the therapeutic targeting of individual gene/protein germline polymorphisms?
- What are the main characteristics of the Beta-1 adrenergic receptor polymorphisms presented by the speaker?

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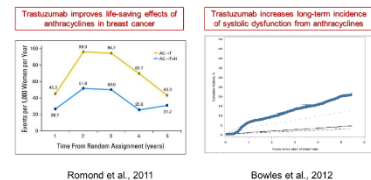
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Cardiotoxicity of chemotherapeutic drugs



Prof. Minotti talked about the Cardiotoxicity of chemotherapeutic drugs. The speaker coming from Rome (IT), presented very interesting data on cardio-oncology. Going deeper in his lecture, Prof. Minotti talked about the main cardiotoxic effects of anthracyclines also at quite low doses, believed to be safe for the heart till now. In the main part of

his lecture, the speaker presented very interesting data on the onset of diastolic dysfunction in patients treated with chemotherapy and on the new oncologic drugs that make the cardiac cells weaker and the old drugs more toxic. More in particular Prof. Minotti talked about trastuzumab, its mechanism of action, its receptors present in the hearth as well



as in the other organs. In the second part of his talk, the speaker presented very interesting data on other new drugs causing cardiotoxicity but not HF like ibrutinib, a BTK inhibitor, responsible for the onset of atrial fibrillation or ponatinib, a BCR-ABL T315I mutant inhibitor that causes an higher rate of arterial thrombosis in CML patients. In the last part of his

presentation Prof. Minotti presented other very interesting data on the anti VEGF drugs causing dose-dependent hypertension and on the tyrosin kinase inhibitors responsible for the QT prolongation. In conclusion, Prof. Minotti pointed out that the cancer patients need for a very effective long-term medical care program in order to avoid the consequences of the cardiotoxic effects of chemotherapy.

TKI and QT prolongation

Tyrosin kinase inhibitor	Delta QTc >80 ms (% of patients)	QTc >500 ms (% of patients)	Torsade de pointes (% of patients)
Bosutinib	0.34	0.2	n.a.
Crizotinib	3.5	1.3	n.a.
Dasatinib	0.6-3	<1.4	n.a.
Lapatinib	11	6.1	n.a.
Nilotinib	1.9-4.7	<1.2	n.a.
Ponatinib	n.a.	2	<0.3
Ponatinib	n.a.	n.a.	n.a.
Sorafenib	n.a.	n.a.	n.a.
Sunitinib	1-4	0.5	<0.1
Vandetanib	12-15	4-3.3	reported (unknown %)
Vemurafenib	1.6	1.6	reported (unknown %)

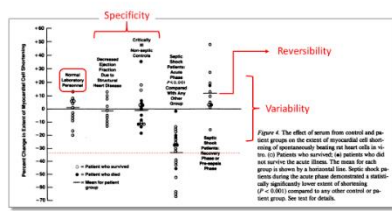
- What is the mission of cardio-oncology from the speaker point of view?
- When do rise diastolic dysfunction in patients with chemotherapy?
- What is the mechanism of action of Trastuzumab on the hearth from the speaker point of view?
- What are the main characteristics of ibrutinib based on the data presented by the speaker?
- How targeted are the targeted drugs based on the data presented by the speaker?

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Inflammation and Heart Failure

Interleukin-1 and the Heart

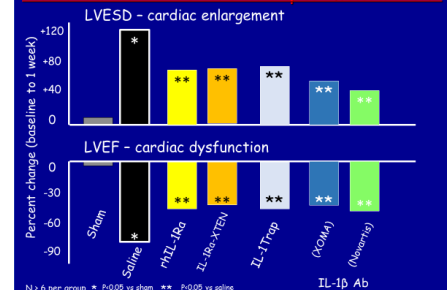


J Clin Invest. 1985 Oct;76(4):1539-53.

Inflammation and Heart Failure was the topic Prof. Abbate talked about. The speaker coming from Richmond (USA), introduced his talk by presenting very interesting data on the link between inflammation and the heart. Going deeper in his lecture, Prof. Abbate talked about interleukin-1 and its correlation with heart failure starting from its depressive effect on the hearth. In the main part of his lecture, the speaker

presented very interesting data on three main topics like the role of IL-1 in the development of HF, its role in acute HF and finally on its role in CHF. Speaking about IL-1 and the onset of HF, Prof. Abbate presented very interesting data on the role of IL-1 receptor antagonist as a sensitive marker of instability in patients with CAD and on the VCU-Anakirna remodelling trial. In the second part of his lecture, Prof. Abbate talked about the effect of IL-1 on the acute heart failure and presented very interesting data on animal studies

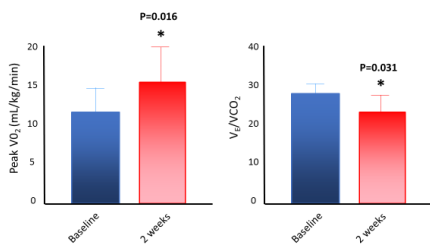
Interleukin-1 blockers in experimental AMI



N ≥ 6 per group * P<0.05 vs sham ** P<0.05 vs saline

running in mice demonstrating the acute effect of IL-1 on HF and the opposite effect of Anakirna. Finally, Prof. Abbate talked about IL-1 and CHF and presented very interesting data on the effects of Anakirna in patients with systolic heart failure discharged by hospital and stable within two week from discharge. In conclusion, Prof, Abbate pointed out that the IL-1 blockade may improve exercise capacity in patients with systolic HF.

The role of IL-1 in chronic Heart Failure



Van Tassel BW et al. PLoS One. 2012

- What is the main effect of IL-1 on the hearth based on the data presented by the speaker?
- What is the effect of Anakirna in CAD patients based on the data presented by the speaker?
- What are the main results of Anakirna on the quality of life in the REDHART study presented by the speaker?

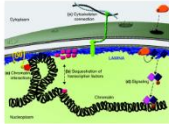
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Biomechanical characteristics of cardiomyocytes in Inherited Cardiomyopathies

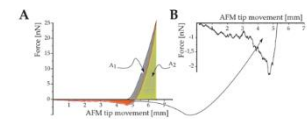
LAMINOPATHIES

- Mutations in lamins and other nuclear envelope proteins can cause a variety of diseases (laminopathies), some of which also have cardiac phenotypes
- LMNA mutations account for 5% to 10% of inherited dilated cardiomyopathies characterized by poor outcome and high risk of sudden death
- Given its prevalence and clinical impact, detailed understanding of LMNA function in health and disease will fulfill an unmet clinical need and will lead to advancement in the treatment of heart failure



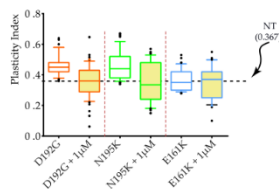
Prof. Sbaizero talked about the biomechanical characteristics of cardiomyocytes in Inherited Cardiomyopathies. The speaker coming from Trieste (IT), presented very interesting data on molecular biology, cell biology and patients. Going deeper in his lecture, Prof. Sbaizero talked about a couple of concepts like laminopathies and mechanotransduction with the

Atomic Force Microscopy (AFM)



intention to present his study aiming to investigate the ways through which lamin mutations alter the biochemical properties of the cardiac cells and the mechanism linking the altered biomechanical properties to the increase of the stress vulnerability. In the main part of his lecture, the speaker presented his study starting from the

Rescue Plasticity Index



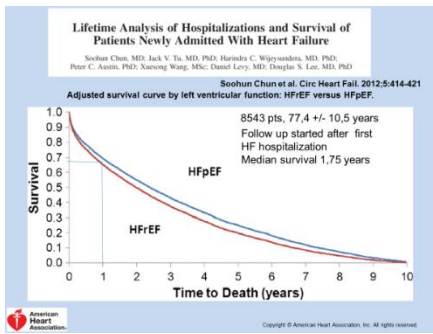
applied methodologies and the three LMNA mutations studied. Finally, Prof. Sbaizero presented the results of his study, not only on the effects of mutations but also on the effects of the p38-MAPK inhibitor. In conclusion, the speaker pointed out that the lamin mutations alter the biochemical properties of the cardiac cells and the p38 inhibitor can rescue these biochemical properties.

- What are the methodology applied by the speaker in his study?
- What are the three LMNA mutations studied by the speaker?
- What are the main results on morphology and elasticity?
- What's about the plasticity index presented by the speaker?

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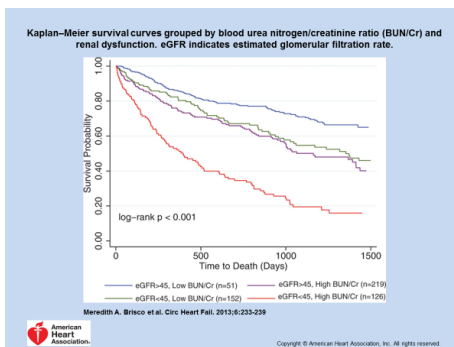
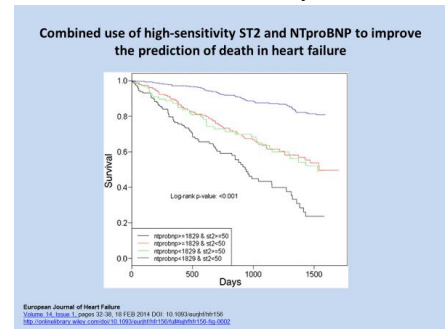
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Prognostic markers in patients with Heart Failure



The prognostic markers in patients with Heart Failure was the topic Prof. Goncalvesova talked about. The speaker coming from Bratislava (SVK), presented very interesting data on the hospitalization and survival rate of new patients affected by HF with preserved EF. Going deeper in her lecture, Prof. Goncalvesova talked about prognosis/identification of risk factors from the patients, the researchers and the physicians point of view.

Speaking about the individual patient decision making, the speaker, presented very interesting data on the heart transplant listing and the VAD implantations. In the main part of her presentation, Prof. Goncalvesova talked about the available risk's markers in routine outpatient and bedside visits and presented very interesting data on the



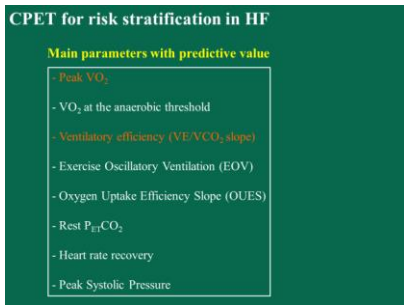
predictors of all-cause mortality at 1 year for AHF and CHF patients. In the second part of her talk, the speaker presented the main models for the mortality/hospitalization risk calculation together with the data on their predictive power. Finally, Prof. Goncalvesova talked about a clinical algorithm to guide early referral to advanced heart failure centers. In conclusion, the speaker pointed out that high BUN, low BP, hyponatremia are the strongest predictors of poor outcomes in the models.

- What are the main independent predictors of one year mortality presented by the speaker?
- What is the predictive power of the mortality/hospitalization models presented by the speaker?
- What are the key points of the clinical algorithm presented by the speaker?
- How do the models work, based on the data presented by the speaker?

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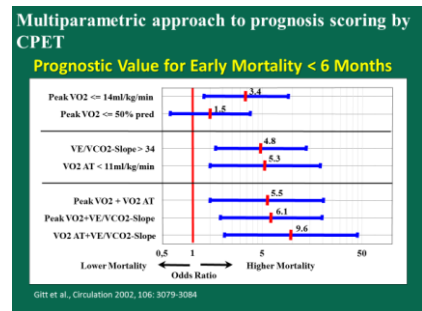
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Cardiopulmonary exercise testing in heart failure and cardiomyopathies. Heart and Lung: CPET from physiopathology to clinical decisions

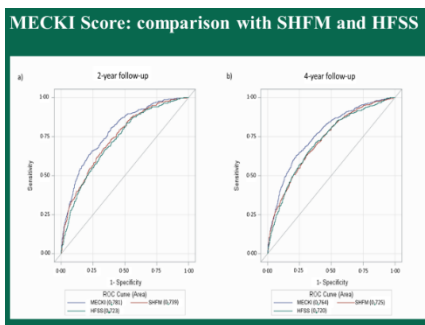


Cardiopulmonary exercise testing in heart failure and cardiomyopathies. Heart and Lung: CPET from physiopathology to clinical decisions was the topic Prof. Contini talked about. The speaker coming from Milan (IT), presented very interesting data on the role of CPET in HF patients. Going deeper in his lecture, Prof. Contini talked about the CEPT derived parameters and those ones

used for the risk stratification in HF. In the main part of his talk, the speaker presented very interesting data on Peak $\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ slope applied in the CHF prognosis. Prof. Contini talked also about the multiparametric prognosis assessment in HF and presented very interesting data on a multivariable score composed by 5 parameters. In the second part of his lecture, the speaker talked about the main prognostic scores used for the HF



mortality prediction like the Seattle HF model and the HF survival score and presented a very innovative score, the so called MECKI score characterized by the match of the metabolic exercise with cardiac and kidney indexes. Prof. Contini talked about the methodology used for the development of this score, starting from the selection of the more significant and robust parameters and the algorithm identification and presented very interesting data on its



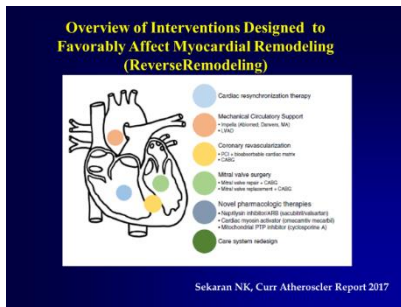
validation.

- What are the 5 parameters composing the multivariable score presented by the speaker?
- What are the clinical Italian center taking part of the MECKI score group presented by the speaker?
- What are the main characteristics of the ADHD in older patients?
- What's about MECKI validation?

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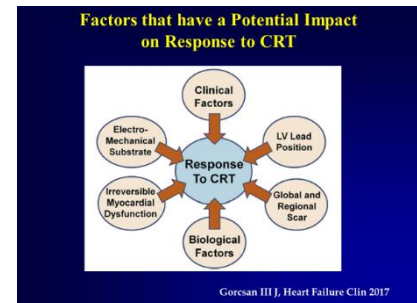
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Prognostic evaluation of LV Reverse Remodeling, LV recoil and Functional Mitral Regurgitation

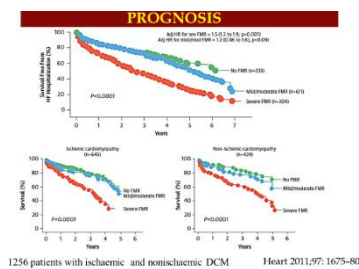


The prognostic evaluation of LV Reverse Remodeling, LV recoil and Functional Mitral Regurgitation, was the topic Prof. Carerj talked about. The speaker coming from Messina (IT), presented very interesting data on ventricular remodelling and on an overview of interventions designed for the best approach to the myocardial remodelling. Going deeper in his lecture, Prof. Carerj talked

about the cardiac resynchronization therapy and the novel pharmacological therapies, by presenting very interesting data given by the main clinical trials running in patients with ventricular remodelling. In the main part of his lecture, the speaker presented very interesting data on the coronary revascularization therapy and more in particular on PCI together with bio absorbable cardiac



matrix and on CABG. Prof. Carerj talked also about the potential benefits of the coronary revascularization and the imaging modalities used to test the myocardial viability. In the second part of his talk, the speaker presented many data on the mitral valve surgery divided into valve repair and replacement, implemented in patients affected by SMR and talked about the main therapeutic considerations, like goals and guidelines.

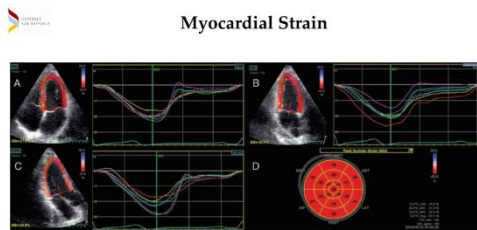


- What are the main results of the concurrent inhibition of ACEi and Neprilysin in experimental heart failure?
- Why is important to study together with the LV also the LA from the speaker point of view?
- What are the main factors that have a potential impact on response to the cardiac resynchronization therapy presented by the speaker?
- What are the goals of the mitral valve surgery in patients affected by secondary mitral regurgitation, presented by the speaker?

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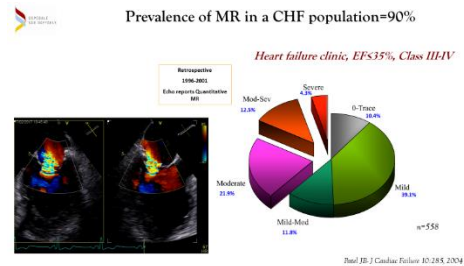
<http://www.fondazione-menarini.it/Home/Eventi/Advances-in-Heart-Failure-Cardiomyopathies-and-Pericardial-Diseases/Video-Slide> ... and, after having logged in, enter in the multimedia area.

Strain, Speckle Tracking, 3D Echocardiography: which role in the assessment of heart failure?

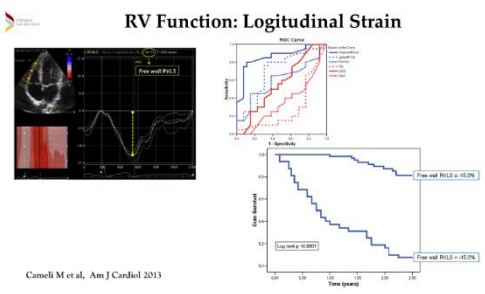


Strain, Speckle Tracking, 3D Echocardiography: which role in the assessment of heart failure? was the topic Prof. Agricola talked about. The speaker coming from Milan (IT), presented very interesting data on these topics starting from what is necessary to know about LV dimensions, systolic function and shape, diastolic function, tissue

characterization, MV involvement, Dyssynchrony, RV function, TV involvement, Pulmonary congestion and LA volume. Going deeper in his lecture, Prof. Agricola talked about the theoretical advantages of the 3D echo and presented very interesting imaging data on myocardial strain, its application in staging, phenotyping and verifying the therapy effect in HF patients. In the main part of his presentation, the speaker talked about the multimodality cardiac imaging used for guiding



CRT, its use in the myocardial tissue characterization and finally about the detection and the follow-up evaluation of valve diseases like the mitral regurgitation. Finally, Prof. Agricola presented very interesting data on the application of the longitudinal strain and the 3D technique for the RV functional evaluation together with the pulmonary congestion evaluation.

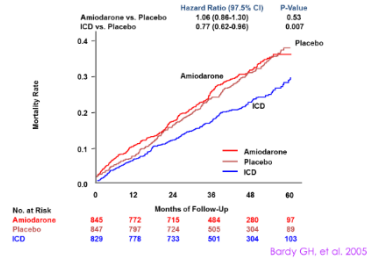


- What are the main characteristics of the myocardial strain?
- What are the main data on the myocardial strain application in HFpEF patients, based on the data presented by the speaker?
- What is the non-responder rate to CRT, based on the data presented by the speaker?
- What are the main applications of the myocardial strain in the patients affected by clinical and sub-clinical myocardial diseases?
- What is the therapeutic approach to HF patients with MR, based on the data presented by the speaker?

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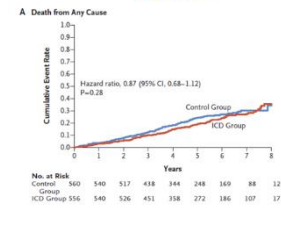
Cardiac resynchronization and sudden death risk prevention: a critical overview

SCD-HeFT Mortality Rate: Overall Results



Cardiac resynchronization and sudden death risk prevention: a critical overview was the topic Prof. Cappato talked about. The speaker coming from Bergamo (IT), presented very interesting data on the implantable cardioverter defibrillator and the cardiac resynchronization. Going deeper in his lecture, Prof. Cappato talked about ICD, by highlighting that with this technology it is possible to reduce mortality of about 23%. The speaker presented also very interesting data given by many studies, the Danish one included.

DANISH



In the main part of his presentation, Prof. Cappato talked about the subcutaneous ICD and the cardiac resynchronization therapy for CHF patients. More in particular the speaker discussed the main considerations comparing CRT and ICD. In conclusion, Prof. Cappato pointed out that ICD therapy is effective and that CRT works in patients with desynchronization cardiomyopathy.

CRT & ICD Therapy in CHF: A Critical Overview

Conclusions

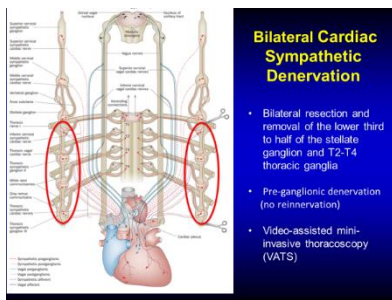
- ICD therapy effective
- CRT works in patients with desynchronization cardiomyopathy (super-responders)
- These patients cannot be distinguished from the remaining population (substrate disease) at time of implant
- Too many patients receive CRT

- What are the main characteristics of the Danish study population, based on the data presented by the speaker?
- What are the main randomized clinical trials on ICD presented by the speaker?
- What are the main considerations sustaining CRT against ICD based on the data presented by the speaker?
- What's about the effect of CRT on survival from the speaker point of view?
- What are the main results of the resynchronization therapy in advanced HF patients from the speaker point of view?
- What's about the comparison between CRT and ICD in CHD patients, based on the data presented by the speaker?

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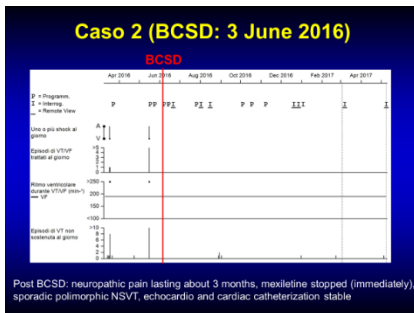
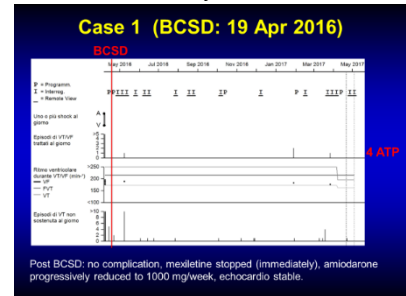
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Cardiac sympathetic denervation in structural heart disease: single center experience



Cardiac sympathetic denervation in structural heart disease: single center experience was the topic Dr. Dusir talked about. The speaker coming from Pavia (IT), presented very

interesting data on this new technique performed on two patients affected by ventricular tachycardia the first one and ventricular fibrillation the second one, after the administration of amiodarone without the resolution of their clinical conditions. In the main part of her talk the speaker presented the data on these two patients after the sympathetic denervation execution, showing that after one year these two patients are stable, without any complication.

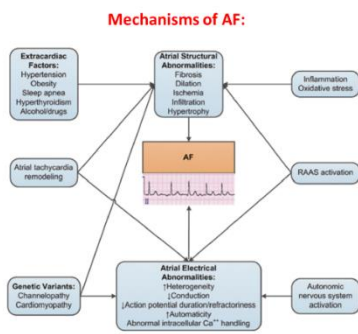


- What are the main characteristics of the sympathetic denervation, presented by the speaker?
- How many patients with electrical storms are hospitalized in the center the speaker coming from?
- What is the survival free rate from ICD shock, based on the data presented by the speaker?

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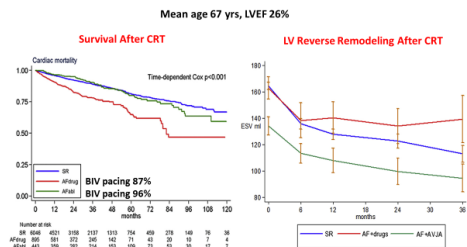
Ablation of atrial fibrillation in end-stage heart failure and cardiomyopathies



Ablation of atrial fibrillation in end-stage heart failure and cardiomyopathies was the topic Dr. Proclemer talked about. The speaker coming from Udine (IT), presented very interesting data on the mechanism of AF. Going deeper in his presentation, Dr. Proclemer talked about the main data published on Atrial fibrillation linked to Heart Failure. In the main part of his talk,

the speaker, presented very interesting data on the long-term outcomes of catheter ablation in AF patients also in comparison with medical therapy based on amiodarone administration. More in particular Dr. Proclemer talked about the catheter ablation in paroxysmal AF patients and

Cardiac Resynchronization Therapy in Patients With Atrial Fibrillation



Gasparini et al. J Am Coll Cardiol HF 2013;1:500-7



Ablation of atrial fibrillation in end-stage heart failure and cardiomyopathies

Conclusion

- ✓ AF ablation should be considered in not too old and sick patients who deteriorate early after the onset of AF despite adequate rate control.
- ✓ For patients in whom the relationship between AF and symptoms of HF is less evident, a period of sinus rhythm after ECV may help to identify candidates to AF ablation.
- ✓ AF ablation by means of large PVI and AV nodal ablation with CRT can be useful for selected and different sub-groups of HF patients with refractory AF.

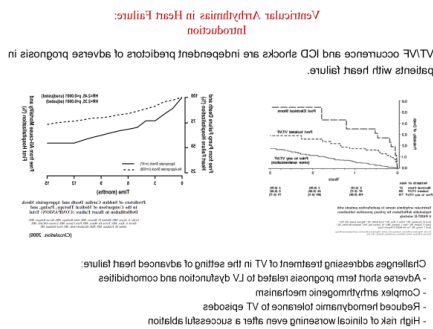
about the wide antral ablation procedure, by presenting very interesting data given by the Udine clinical experience. In the second part of his presentation, the speaker discussed the algorithm published by Anselmino et al. on the catheter ablation of atrial fibrillation in CHF patients and presented very interesting data on the cardiac resynchronization therapy in AF patients. In conclusion, the speaker pointed out that AF ablation should be considered in not too old and sick patients who deteriorate early after the onset of AF.

- What are the main points of the Algorithm presented by the speaker on the catheter ablation of atrial fibrillation?
- What's about the wide antral ablation from the speaker point of view?
- What are the long-term outcomes of catheter ablation in AF patients presented by the speaker?
- What are the key points of the treatment of patients with HF with reduced EF?

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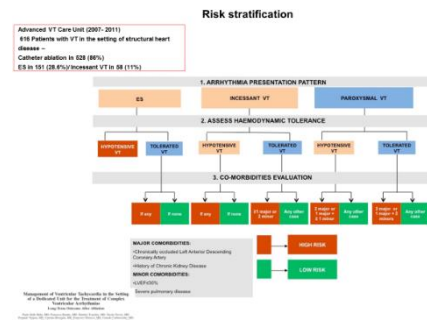
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Ablation of ventricular arrhythmias in end-stage heart failure and cardiomyopathies

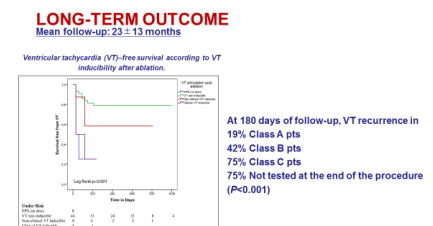


Ablation of ventricular arrhythmias in end-stage heart failure and cardiomyopathies was the topic Prof. Della Bella talked about. The speaker coming from Milan (IT), presented very interesting data on ventricular arrhythmias in HF patients. Going deeper in his lecture, Prof. Della Bella talked about the impact of the catheter ablation on short and long-term survival in advanced HF patients, the risk

stratification, the ventricular tachycardia in advanced HF patients and finally about the role of the hemodynamic support. Talking about catheter ablation in HF advanced patients, the speaker presented very interesting data on survival, by highlighting that the patients in NYHA class II and III have the same outcomes. More in particular he discussed the data given by the clinical experience of his center and the related risk patient stratification. Prof. Della Bella presented also very interesting data on the propagation



patterns during VT mapping performed on HF patients in his clinical center. Finally, the speaker presented very interesting data on the hemodynamic support in VT ablation and on the post-operative course. In conclusion, Prof. Della Bella pointed out that the rhythm stabilization by catheter ablation is not futile as it prevents recurrences of electrical storm, allows recovery of cardiac function and bridges to heart transplantation of LVAD.



- What are the main key points of the risk stratification of HF patient with ventricular arrhythmias treated at the San Raffaele hospital?
- What's about the mapping/ablation approach presented by the speaker?
- What is the procedure outcome in the HF patients, presented by the speaker?
- What is the recurrence rate of the patients treated by the speaker?
- What's about mortality and FU survival?

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Percutaenous implantation of VA ECMO in the cath lab in hands of interventional cardiologist

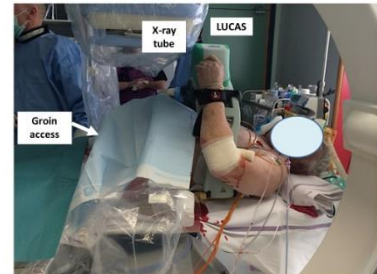
ONCE WIRES AND ARTERIAL/VENOUS SHEATHS ARE IN CORRECT POSITION...



Percutaenous implantation of VA ECMO in the cath lab in hands of interventional cardiologist was the topic Prof. Noc talked about. The speaker coming from Ljubljana (SLO), presented very interesting data on indications for VA via ECMO. Going deeper in his lecture, Prof. Noc talked about the main steps of this procedure for the selection

of the best site available for the ECMO implantation the main problem and the related solutions. In the main part of his lecture the speaker presented very interesting data on the anterograde sheath implantation and the usefulness of the retrograde angiography for the placement of the distal perfusion sheath. More in particular the speaker addressed the audience on the possible

CATH LAB SETTING DURING VA ECMO IMPLANTATION FOR REFRACTORY CARDIAC ARREST



arterial spasms raising in patients treated with too high dosage of vasopressors. In the second part of his presentation, Prof. Noc talked about the ECMO implantation in patients affected by cardiogenic shocks, by highlighting that it is of high importance to choose the right artery at the first attempt. In conclusion, the speaker pointed out that VA ECMO may be followed by coronary angiography, PCI or other procedures.

IMPLANTATION-RELATED COMPLICATIONS OF VA ECMO-REFRACTORY CARDIAC ARREST IN THE CATH LAB (n=12)

Distal perfusion after arterial cannula	4 (42%)
Arterial cannula, Fr	20±2
Venous cannula, Fr	24±2
ECMO duration, days	3±3
ECMO Implantation complications	
Vessel dissection/rupture	0 (0%)
Infection at cannula site	0 (0%)
BARC 3+5 bleeding	1 (8%)
Ipsilateral limb ischemia	5 (42%)

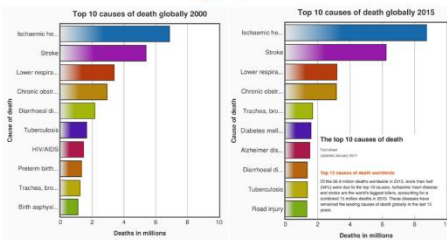
Eurointervention 2016;12:1465-72

- What are the main problems linked with ECMO implantation?
- What's about the solution proposed by the speaker?
- What's about the ECMO implantation in cardiogenic shock
- What is to do in case of severe iliofemoral spasm based on the data presented by the speaker?

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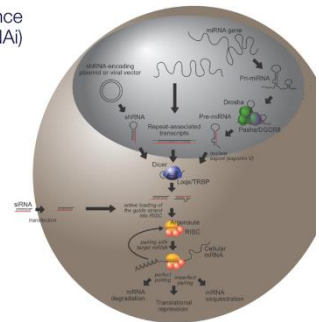
The future is now? Regenerative Medicine and Gene therapy as effective treatment for Heart Failure



"We keep discussing how much we have progressed among our subspecialty, yet the paradox is that the disease state remains the number 1 killer in the world"
Valentin Fuster, MD, PhD, JACC Editor-in-Chief

The future is now? Regenerative Medicine and Gene therapy as effective treatment for Heart Failure was the topic Prof. Giacca talked about. The speaker coming from Trieste (IT), presented very interesting data on the ICGEB that is an international organization working in the United Nations system on the global status of health with the aim to collect a lot of data on children mortality, but also on

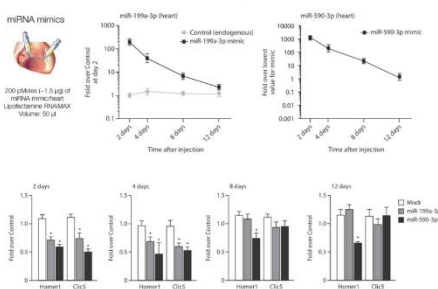
RNA interference (RNAi)



cardiovascular diseases outcomes. In the main part of his lecture, Prof. Giacca talked about the top 10 causes of global mortality in 2015 where Ischemic heart diseases are at top of the ranking. More in particular the speaker presented very interesting data on the new drugs in clinical development and on the most common degenerative conditions without affordable drugs available for their cure till now. In the second part of his lecture, Prof. Giacca talked about the biological drugs for the degenerative conditions and highlighted that there are many of these drugs for cancer but not for the cardiological diseases. The speaker presented also a huge amount of data on the therapeutic nucleic acids for the

somatic gene therapy able to block the RNA function, or to suppress a STOP codon or to form a triple helix. In the last part of his presentation Prof. Giacca talked about microRNA therapeutics and their effects on fibrosis, angiogenesis, cardiomyocyte regeneration demonstrated in animal studies performed in mice but also in pigs and presented very interesting data on the effects of the miRNA mimics on the myocardial repair after MI in pigs.

Prolonged effect of miRNA mimics after intracardiac injection



- What are the main topics of the CVD burden presented by the speaker?
- What's about the biotherapeutics for degenerative conditions presented by the speaker?
- What are the main advantages of the informational drugs based on the data presented by the speaker?
- What are the main effects of miRNA mimics in pigs presented by the speaker?

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These are only some of the topics addressed in the congress's sections

For a deeper knowledge on these topics, please visit the International Menarini Foundation web site where You can find all the speeches in their full version.

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