HIGHLIGHTS

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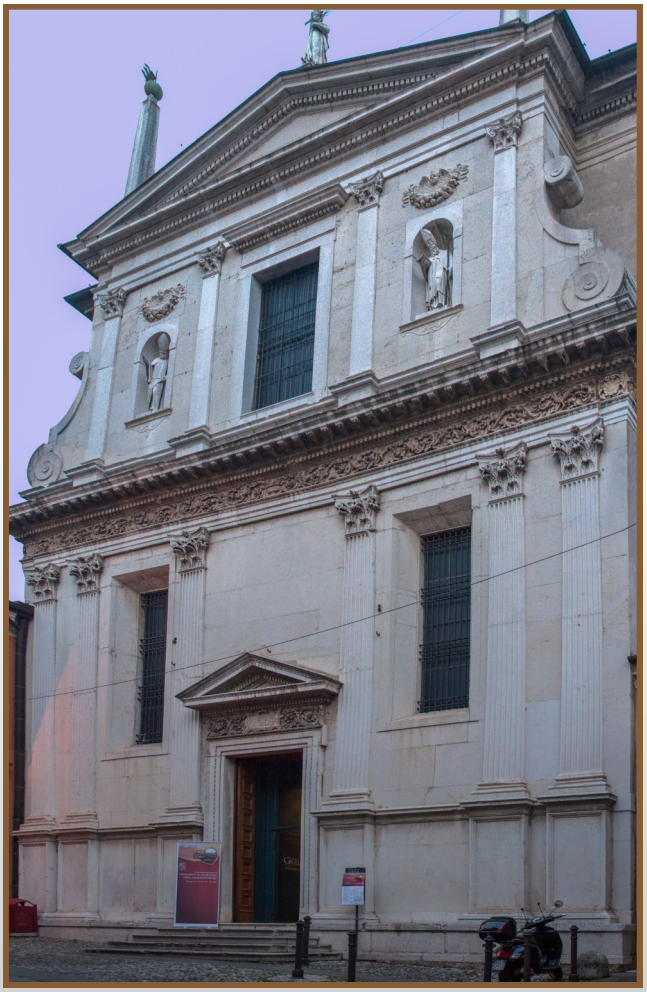
Welcome to Brescia!

Marco Metra (Brescia, Italy)

Professor Metra, Chairman of the congress, opened the conference recalling the importance of the School of Cardiology in Brescia. He thanked the high-level researchers attending the event and presenting their study based upon years of cardiological research activity. Professor Pecorelli, the Rector of the University of Brescia, talked about the importance of prevention in the context of cardiovascular diseases. He expressed the wish that this congress would further examine this important issue representing the future of the Medicine applied to public health.



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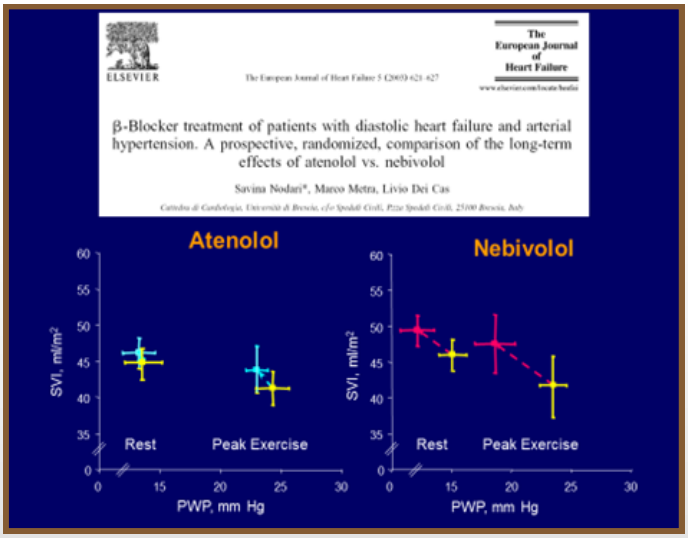
Innovation in Cardiology: the contribution from Brescia. A story that began 40 years ago

Livio De Cais
(Brescia, Italy)

Professor Livio Dei Cas from Brescia reported in his presentation a series of data demonstrating the valuable contribution of the School of Cardiology in Brescia to improve the study of cardiovascular diseases. Ischaemic cardiomyopathy and heart failure represent one of the main public health problems. To grasp the enormity of the problem, just think of the number of deaths due to cardiovascular diseases in European countries corresponding to 50% of the major causes of ill health. What is the contribution coming from the Cardiology of Brescia? It acts on two fronts: journals editing and clinical research on heart failure treatment. Since it was founded in 1983, the Cardiology Unit in Brescia has grown into one of the major worldwide Cardiology in terms of clinical research and patients' health management. In all these years, one of the key objectives of Cardiology has been the cardiovascular prevention to reduce hospitalisations and to increase preventive strategies. The Cardiology of Brescia has been the first in Italy to promote the use of β-blockers to treat heart failure. Another area where the School of Cardiology in Brescia has been outstanding is in scientific journal editing, nearly 500 publications in about 15 years.

Official journal of the Italian Federation of Cardiology. Editor-in-Chief: Livio Dei Cas



What is the impact of the cardiovascular disease in terms of mortality?
What are the main scientific journals published by the Brescia School of Cardiology?
What are the main clinical research lines on Cardiology developed by the researchers from the Brescia School of Cardiology?

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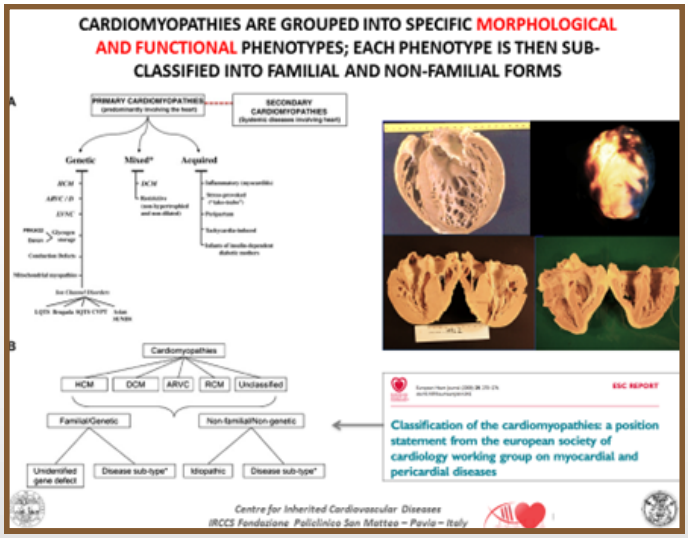
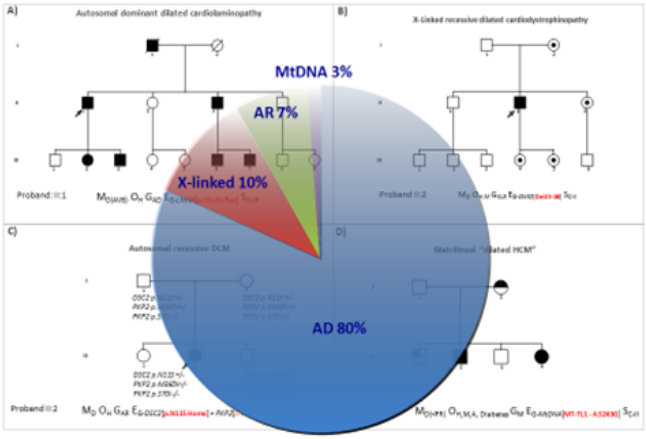
Genetics and other causes of Cardiovascular disease. Genetics of Cardiomyopathies

Eloisa Arbustini
(Pavia, Italy)

Professor Arbustini from Pavia discussed in her presentation the relationship between genetics and cardiomyopathies. Both are mostly characterised by a specific genetic aetiology where the alteration of a gene may be combined with other different phenotypic manifestations. Their diagnosis is therefore based on genetic and phenotypic family screening study. An anatomic marker of cardiomyopathy is the left ventricular no-compaction cardiomyopathy that presents a not compacted left ventricle with trabeculae. This is one of the main genetic forms of cardiomyopathy and currently clinically and genetically studied in order to discover the causes and to identify the most appropriate therapeutic protocols. The speaker submitted also data regarding the different phenotypic manifestations stemming from a single genetic mutation. 80% of the total amount of the familiar dilated cardiomyopathies are autosomal dominant diseases. A core aspect for the diagnosis and for the treatment of cardiomyopathies is the clinical family screening that should always be implemented before each genetic analysis. The synthesis of the data produced by the clinical analysis on patients with cardiomyopathies and those data produced by the genetic analysis are essential for the diagnosis of specific cardiomyopathy forms.





What is the relationship between cardiomyopathies and genetic alterations?
What is the relationship between genetic-based and non-genetic-based cardiomyopathies?
What is the best diagnostic strategy for a cardiomyopathy clinical family screening?
When to perform genetic tests on family members?

To answer these and other questions click on this link: www.fondazione-menarini.it/... and after having logged in, access the multimedia material.

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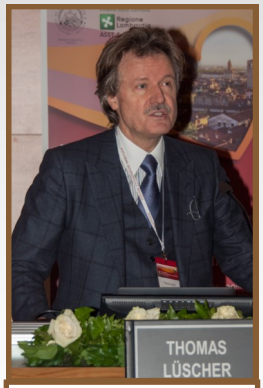
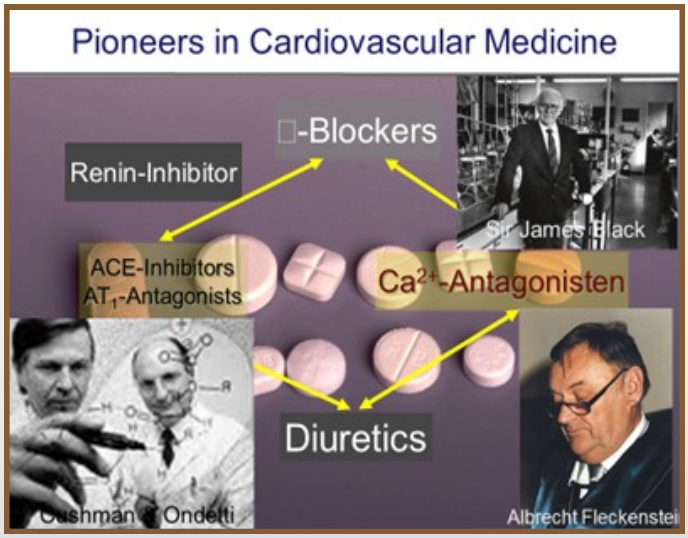
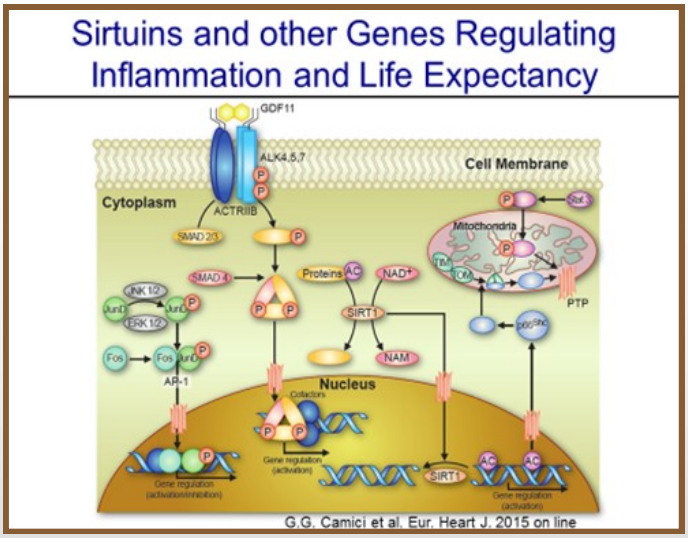
Cardiovascular Disease today: from Pathophysiology to New Prevention Modalities



Thomas Luscher
(Zurich, Switzerland)

Professor Lüsher from Zürich submitted very recent data on cardiovascular prevention strategies based on the study of the main pathophysiological mechanisms of the disease. In the evolution history only the human race developed high plasma levels of LDL (Low Density Lipoprotein). Therefore, atherosclerosis is a typical human race disease and it is not present in other animal species. Recent data noted the inhibition of the expression of PCSK9 (Proprotein convertase subtilisin/kexin type 9) protein as the triggering factor for the LDL synthesis in the liver. CEPT (Cholesteryl ester transfer protein) is another enzyme that plays an important role in LDL synthesis via VLDL (Very-low-density lipoprotein) and it acts in the liver. Several attempts have been made to synthesize drugs capable of inhibiting the activity of this enzyme. However, these efforts have not yet produced the expected positive results. Advances in medicine have led to a dramatic reduction of cardiovascular events from the 1950s until today. Further progresses are currently underway, particularly in terms of identifying the main pro-inflammatory factors responsible for the inflammatory cascade leading to atherosclerosis. One of these factors is Interleukin-6. The new frontier is to study the relationship between atherosclerosis and aging to analyze the underlying inflammatory conditions and to discover the development of new treatments based on the use of stem cells.





What are the main mechanisms underlying cardiovascular disease?
What are the main pro-inflammatory phenomena?
What is the role Interleukin-6 plays in causing such inflammatory phenomena?
Which is the phenomenon causing the deletion of SIRT1 gene?

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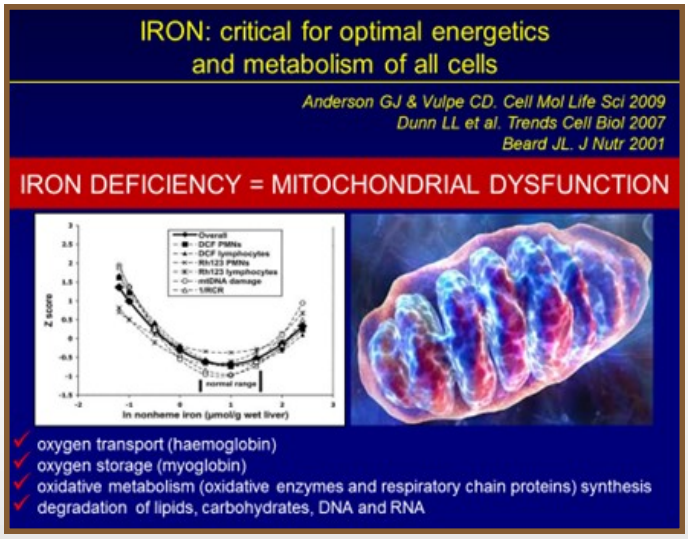
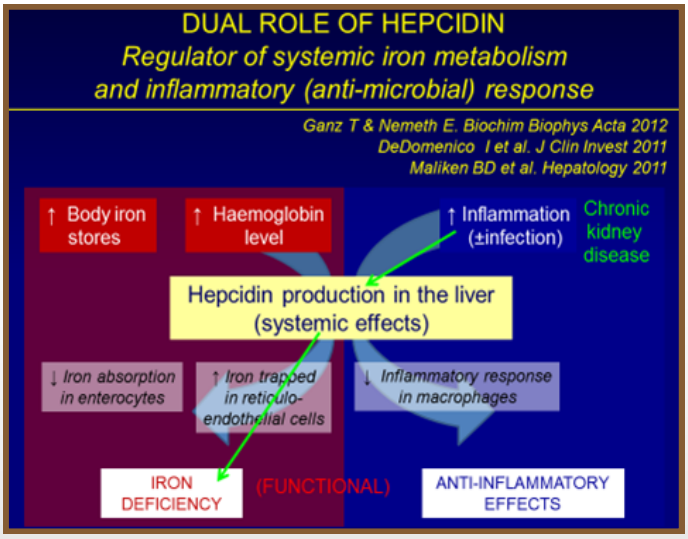
Iron in Cardiovascular Disease

Ewa Jankowska
(Wroclaw, Poland)

Professor Jankowska from Wroclaw examined the connection between iron and cardiovascular disease in detail. Iron deficiency is one of the main factors of heart failure and is often linked to specific inflammatory factors. Iron deficiency is also associated with increased all-cause mortality in cardiac patients. These data show the strategic importance of iron, not only for the phenomena linked to erythropoiesis, but also for other metabolisms: iron deficiency induces, in fact, a mitochondrial dysfunction. A protein that is deeply involved in iron metabolism is Hepcidin. The increase of its production in the liver, mediated through inflammatory phenomena, causes iron deficiency. Iron deficiency is furthermore a negative prognostic factor also for patients suffering from coronary artery disease and type II diabetes. The Speaker has finally presented interesting data on the role of iron therapy in managing heart failure patients.





What are the main connections between iron deficiency and cardiovascular disease?
What is the new definition of iron deficiency in patients with heart failure?
What is the role of Hepcidin in the control of iron metabolism?
What is the role of iron therapy in patients suffering from hearth failure?

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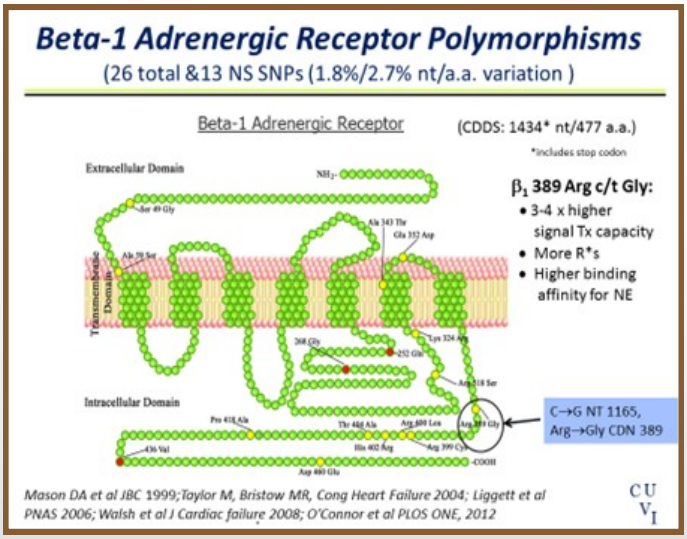
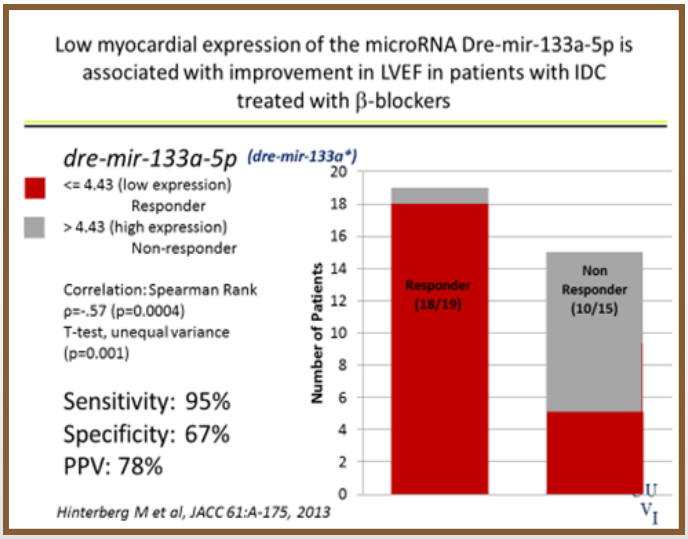
Genetics for Drug Selection

Michael Bristow
(Denver, USA)

Professor Bristow from Denver examined in detail the relationship between patients' phenotyping with genomic biomarkers and the identification of the targeted therapy. Although this strategy presents potentially significant advantages, it seems not to be applicable to all models of disease. However, the use of pharmacogenomics seems to be particularly effective in some clinical conditions or in specific pathological situations. Examples include the discovery of genetic variations influencing the hepatic metabolism of Warfarin or even the identification of specific therapies for genetic variations affecting the primary or secondary target of some medications as Warfarin and Bucindolo. This application presents significant advantages in the fields of neoplastic and congenital heart diseases. An example is the low myocardial expression of the microRNA Dre-mir-133a-5p, which is associated with improvement in LVEF (left ventricular ejection fraction) in patients with IDC (Implantable Cardioverter Defibrillator) and treated with β-blockers. On a patient with heart failure, the molecular phenotyping could lead to significant therapeutic effects, particularly when biomarkers present a high-level value of sensitivity and specificity. The test must be easy to perform and easy for sampling.





What are the main diseases that can benefit from phenotyping using genomic biomarkers?
What are the conceptual models for heart failure management related to genomics?
What are the acute and chronic effects of β1 – AR receptor stimulation?

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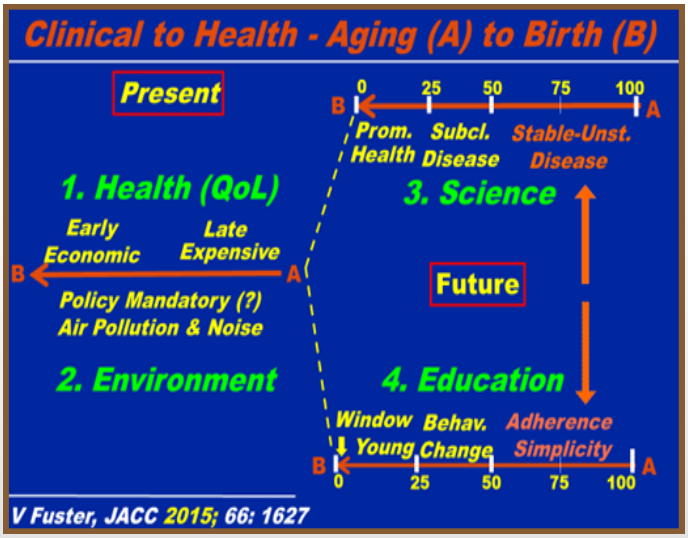
The Heart and The Brain
Cardiovascular prevention: a new frontier

Valentin Fuster
(New York, USA)

Professor Fuster from New York talked about the correlation between cardiovascular health and aging. He delved into three aspects regarding the qualitative and quantitative assessment of the mental health in different age. First, the adherence to cardiovascular therapy with drug overuse and underuse representing a typical problem of adult/old age. Second, the burden of disease that is a problem both for patient and for family members. Last, the wellbeing defined as the complete physical, intellectual and social well-being, and not just the absence of any illness and disease. The Speaker dwelt in particular on the difference between current and public health strategies to implement in a next future. The healthcare policies approach, based on treatment of diseases and not on their prevention, and the high costs resulting from it effectively caused the collapse of the health system. These strategies are particularly affected by global pollution, as air pollution and noise are pro-inflammatory factors underlying the development of various pathologies. As an alternative to this model, Professor Fuster presented a whole series of data derived from population studies. They were conducted with scientific methodology and based on a new model contemplating educational and scientific strategies along with health and environmental strategies, which were applied to different ages, from childhood to old age.



What are the new possible health policy strategies to be implemented in the next future?
How to approach the typical senile and presenile multidrug therapy problem?
What are the most effective educational models for the different human ages?
How strongly cardiovascular disease influence the degeneration of brain tissue?

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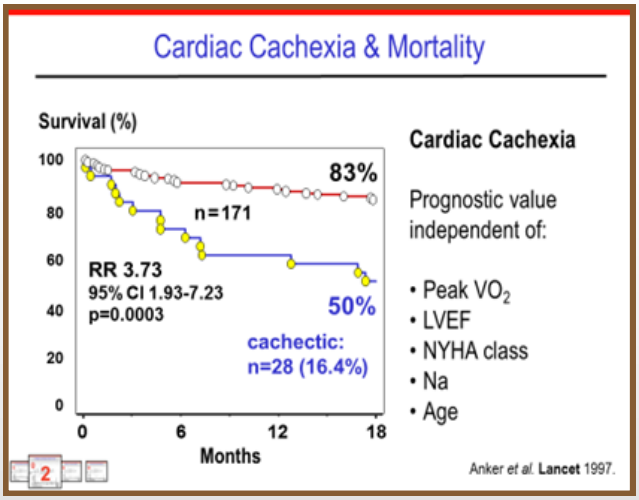
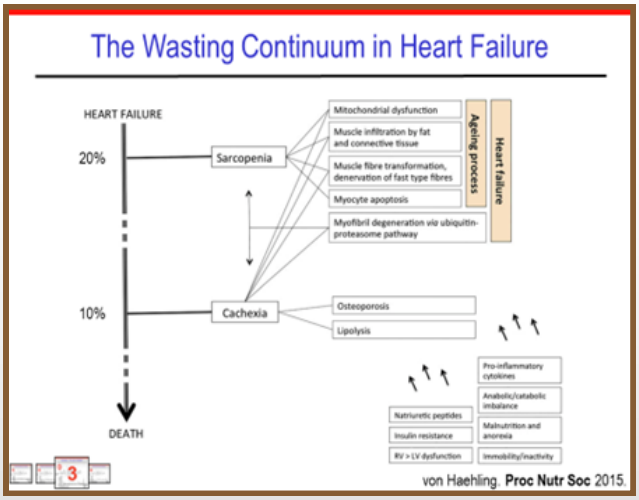
Cachexia and other Co-morbidities

Stephan von Haehling
(Gottingen, Germany)

Professor von Haehling from Göttingen analysed the issue of cachexia and other co-morbidities affecting those patients suffering from left ventricular dysfunction. The concept of cachexia should be differentiated from other seemingly similar terms such as sarcopenia and frailty. The patient with cachexia loses both body weight and muscle mass as a consequence of specific chronic diseases. This phenomenon is also seen in patients with heart failure where the peripheral perfusion deficiency and the decreased activity, together with other metabolic and inflammatory factors, lead to the onset of systemic myopathy and loss of fat body mass. Generally, the loss of muscle mass anticipates the loss of body fat and, when the phenomena are both present, patients begin to lose weight. This factor could also be seen in patients still in overweight conditions and it may mislead the doctor at diagnosis. In patients with heart failure Cachexia worsens significantly the prognosis. Sarcopenia means loss of muscle mass not associated with weight loss. Frailty is the increased likelihood of dependency in carrying out normal activities and affects the patient with cachexia or sarcopenia. Then, for one reason or another, patients suffering from heart failure are intended to lose muscle mass. How to deal with this phenomenon? Physical exercise increases anabolic factors and reduces the catabolic phenomena in the muscle metabolism by inhibiting proteasome activity. Other approaches provide the reduction of catabolic or inflammatory factors and there is a whole range of new molecules acting on these phenomena.





What are the main features of cachexia? How to differentiate diagnosis of cachexia from diagnosis of sarcopenia? What are the new molecules in the development stage determining the reduction of the catabolic factors connected to the loss of muscle mass? What are the new molecules in development leading to a reduction of the inflammations due to the loss of muscle mass?

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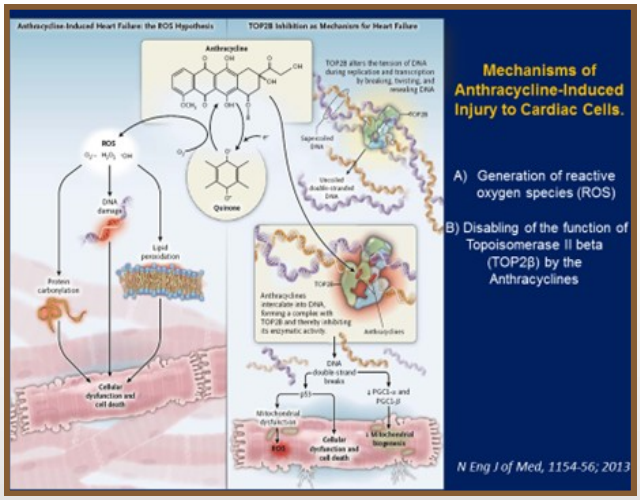
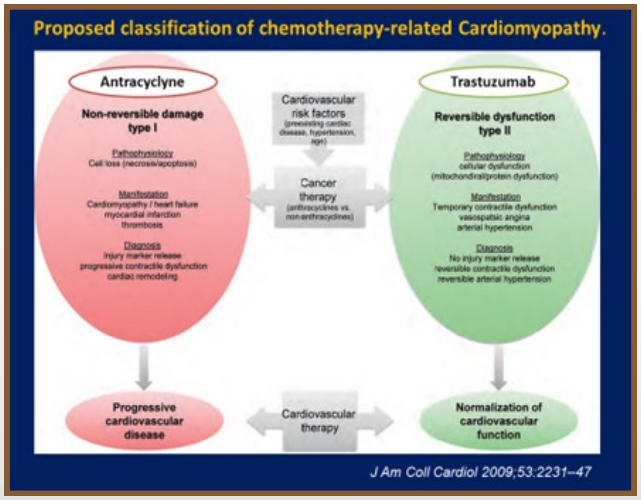
Heart and cancer therapy

Savina Nodari
(Brescia, Italy)

Professor Nodari from Brescia talked about the problem of the cardiotoxicity induced by cancer treatments. Two are the main mechanisms inducing the cardiac toxicity: the generation of reactive oxygen species (ROS) and the inactivation of topoisomerase II beta with consequent alteration of mitochondrial metabolism. All these processes have a greater impact in the longer run than in the acute period. As regards treatment protocols, the group of drugs more involved is anthracyclines. In addition, enhancing drugs affect the heart adversely. However, recent published data appear to indicate that this organ damage could be reversible. Evaluation of LV ejection fraction (LVEF) is the traditionally preferred prognostic method by cardiologists and guidelines for assessing cardiac function during anticancer treatment. Are we sure that this is the right parameter? Probably not, as ejection fraction does not represent an indicator of the decline in myocardial contractile function. For the early detection of cardiotoxicity it is important to use novel echocardiographic techniques as the global strain. Among cardiac biomarkers, Troponin produced contradictory results. The real problem is that these patients are not sufficiently monitored and they often meet the cardiologist only when they are in a symptomatic condition. The problem is clearly due to the lack of an early clinical evaluation. Furthermore, also cardioprotective agents were recently studied and the trials indicated that the treatment with ACE inhibitors or statins should be used in primary prevention protocols to obtain significant improvement of the prognosis of the patients.





What are the main effects of anthracyclines on myocardial cells?
What are the main cardioprotective drugs? Are all these drugs safety?
How to identify patients at higher risk of cardiotoxicity?
How important is the genetic variants in predicting patient's sensibility to anthracyclines?

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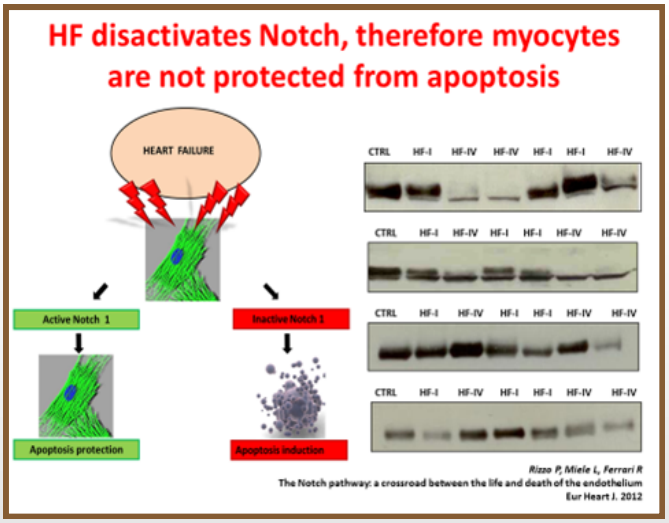
Life and Death of the Myocyte as a cause of remodelling

Roberto Ferrari
(Ferrara, Italy)

Professor Ferrari from Ferrara submitted extremely interesting data on the biological mechanisms involved in the myocardial remodelling. First, what is meant by remodelling? There are various definitions based on clinician, biologist and pathologist point of view and there are many different treatment options for the cure. However, how to explain this phenomenon? To answer this question we need to plunge into the mystery of life inevitably tied to birth and death. The embryonic myocytes are connected to the life/death cycle. It is not the same for adult myocytes dying from necrosis. During remodelling, myocytes reacquire the potential they lost in the post-embryonic phase and again they can reproduce the cellular life/death cycle phenomena. In the myocardial remodelling, we notice apoptosis and hypertrophy phenomena. Both are the consequence of neuroendocrine tissue activation following a myocyte stretch. These factors demonstrate a down-regulation towards the embryonic myocardial tissue where the apoptosis and hypertrophy phenomena are able to maintain a seemingly well-balanced equilibrium for sufficiently long time-periods. As time goes by, however, apoptosis phenomena prevail over those with hypertrophy. Notch is a system that adjust the balance between these phenomena. In hearth failure patients, it is interesting to see that this system is inhibited by allowing apoptosis to prevail over hypertrophy.

- Life and death are integrating parts of the universe
- Express opposite concepts, but are aspects of the same design
- Two entities programmed from the nuclei

  - Life → Reproduction
  - Death → Apoptosis



What are the characteristics of embryonic myocytes?
How to define apoptosis?
Are there drugs using apoptosis and hypertrophy factors?
What is the mechanism of action of Cytokine IL6?
Are there drugs using the mechanism of action of Interleukin IL6?

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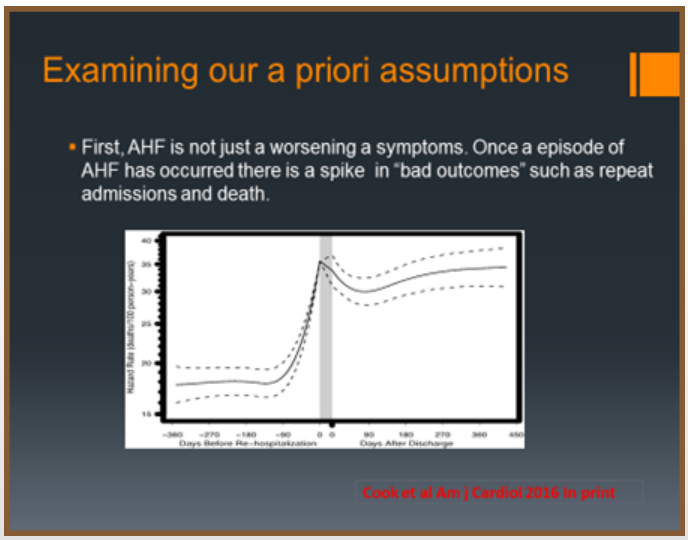
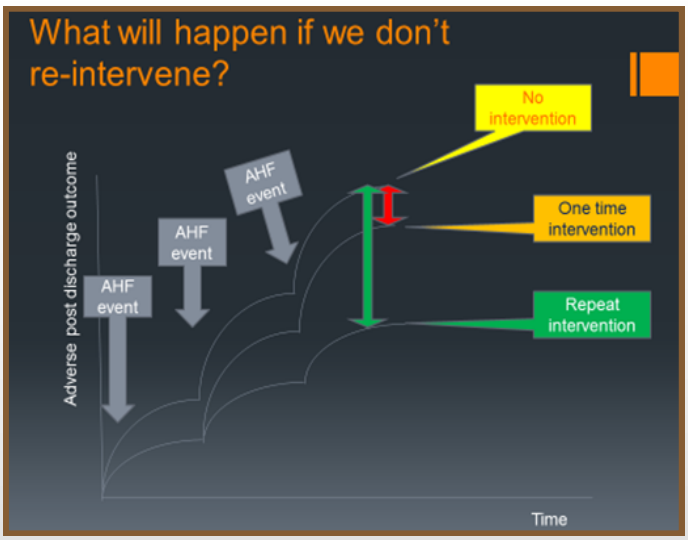
Affecting post discharge outcomes in Acute Heart Failure: a proposal for a new frame work

Gad Cotter
(Durham, UK)

Professor Cotter from Durham submitted very interesting data on this current issue regarding therapeutic protocols for patients with AHF (acute heart failure). Are we sure that the therapeutic approach for hospitalized patients with AHF is correct? Professor Cotter opened his speech with this question and answered to it clearly by submitting data taken from the literature. The therapeutic approach of these patients is limited to rebalance their hemodynamic status but it is encumbered by a whole series of relapses marked by a spike in mortality for every new hospital admission. Despite these considerations supported by incontrovertible clinical data, we do not see any sign of change in the conventional therapeutic approach. Professor Cotter presented new treatment strategies that, together with the action on the hemodynamic balance, are also aimed at limiting neuro-hormonal, metabolic and pro-inflammatory derangements damaging other organs, kidney in particular. This alternative therapeutic strategy is based on the consideration that AHF is a systemic disease characterized by the simultaneous presence of neuro-hormonal, inflammatory phenomena and organ damages. Another topic is based on the attempt to act on the diseases underlying HF with the intention to improve the clinical conditions of the patient. In addition, the implement of socio-economic strategies may help to improve the conditions of those patients discharged from hospital. The opportunity to intervene on them in a complementary way can significantly improve their long-term diagnosis whenever they are hospitalized for the onset of acute disease.





What are the results of the conventional therapeutic approach in patients with heart failure? What are the effects of Serelaxin on the outcome of these patients? What are the main socio-economic factors influencing hospital readmissions during early post-discharge?

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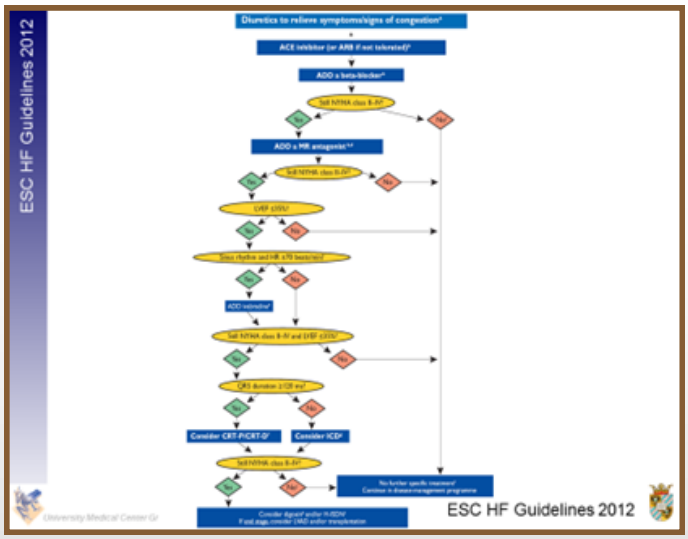
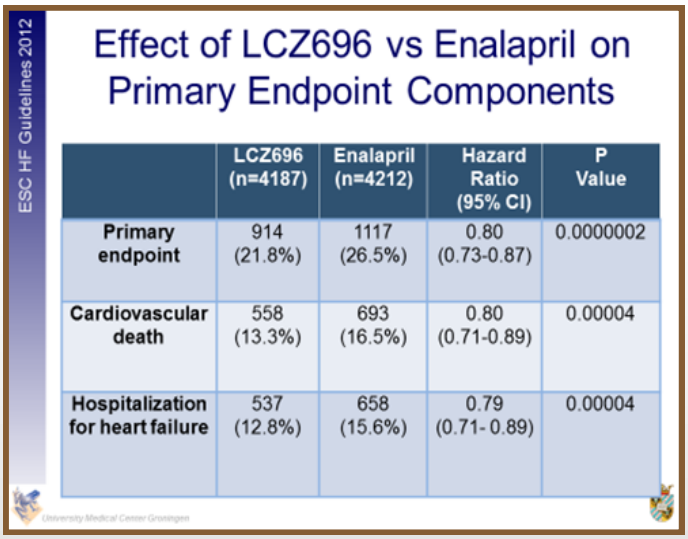
An Alogorithm for Medical Treatment of HFrEF

Adriaan A. Voors
(Groningen, Netherland)

Professor Voors from Groningen submitted very interesting data on the medical treatment options for patients with heart disease and particularly for those with reduced ejection fraction (HFrEF). Professor Voors examined in detail the algorithm suggested by the European Society of Cardiology (ESC) in the heart failure Guidelines of 2012 and submitted literature data for each indicated drug. He began by describing the diuretics to get up to the most recent drugs such as the inhibitors of natriuretic peptide in association with ACE inhibitors. As for the diuretics, he stressed the need to use them at the lowest adjusted achievable dose to restore the correct dry body weight of the patient and to avoid the risk of dehydration leading to hypotension and renal dysfunction. The risk of the intensive use of these drugs is hypotension. ACE inhibitors and angiotensin II inhibitors are the reference medications for the treatment of these patients. They are more effective in combination with β-blockers to relieve disease symptoms and reduce the risk of mortality. In case of persisting symptoms despite treatment, Guidelines recommend the use of β-blockers combined with Muscarinic acetylcholine receptor antagonists. For worsening heart failure it is recommended the combination with Ivabradine. At this point, the speaker presented data on a new drug, the LCZ696, that is an ACE-inhibitor combined with a natriuretic peptide inhibitor that can improve the symptoms of these patients and significantly reduce their mortality.





	LCZ696 (n=4187)	Enalapril (n=4212)	Hazard Ratio (95% CI)	P Value
Primary endpoint	914 (21.8%)	1117 (26.5%)	0.80 (0.73-0.87)	0.0000002
Cardiovascular death	558 (13.3%)	693 (16.5%)	0.80 (0.71-0.89)	0.00004
Hospitalization for heart failure	537 (12.8%)	658 (15.6%)	0.79 (0.71- 0.89)	0.00004

What are the difficulties related to the use of diuretics?
How to combine ACE inhibitors and β-blockers?
What is the effect of β-blockers on mortality?
What are the results of the trials conducted with Ivabradine?
What are the results of the Paradigm trial?

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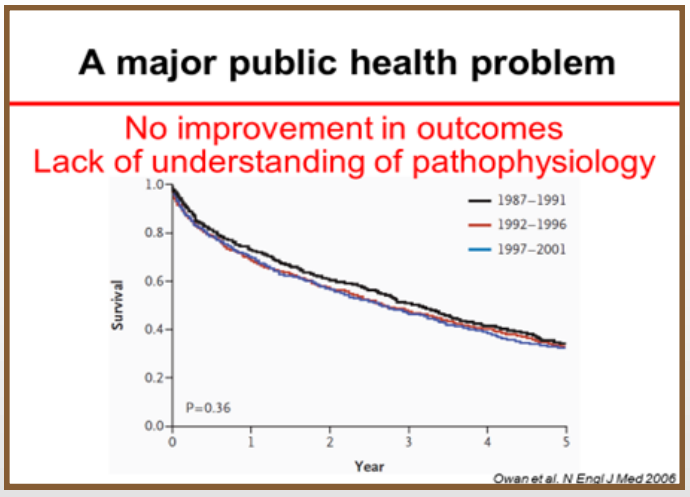
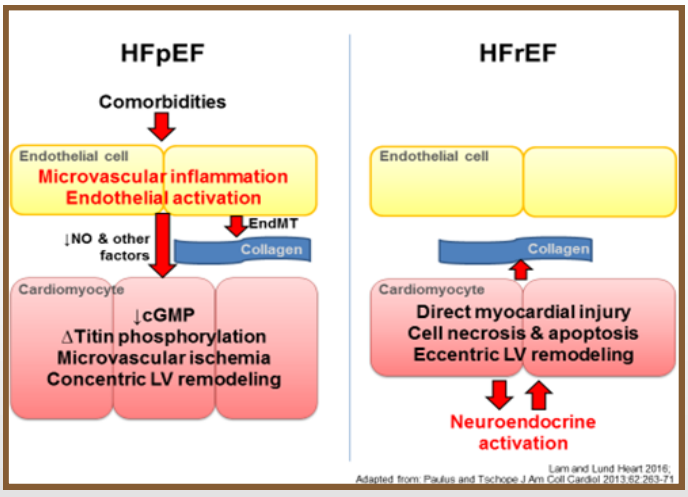
The centre & periphery: mechanisms for HFpEF

Carolyn Su Ping Lam
(Singapore, Rep. of)

Professor Carolyn Su Ping Lam from Singapore examined in detail the issue of HFpEF (heart failure with preserved ejection fraction) submitting very interesting clinical and pathophysiological data. The prevalence of heart failure with preserved ejection fraction is rapidly increasing in the world and it mainly affects female gender. The problem of this disease is the lack of therapeutic protocols to monitor its evolution, probably because it is necessary to increase the knowledge of the related pathophysiological mechanisms. So far, patients with heart failure with preserved ejection fraction have been treated with the same protocols used for hearth failure with reduced ejection fraction (HFrEF) and the results have been unsatisfactory. The HFpEF pathophysiological basis are radically different from HFrEF. HFpEF is characterized by the presence of pulmonary hypertension and right ventricular systolic dysfunction. In this kind of disease, comorbidities, often affecting the prognosis significantly, are consistently observed. Another typical aspect of this type of heart failure is its association with coronary artery disease.
All these features suggest that there is a different pathophysiological mechanisms at the base of the two types of heart failure. In the conventional HFrEF prevails the cardiac remodelling due to myocardial cellular necrosis damage while, in the HFpEF, microvascular inflammation and endothelium dysfunction are prominent and they are in turn responsible of micro-vascular deficiency and oxidative stress in the endocardial tissue. Furthermore, they lead to a deficiency of the enzymatic cGMP-PKG that is responsible for the reduced cardiomyocytes's contractility.





What are the main hemodynamic differences between the two types of hearth failures treated with vasodilators?
How strongly comorbidities influence the outcome of patients with HFpEF?
How important are endothelial dysfunction factors in treating this kind of heart failure?

To answer these and other questions click on this link: www.fondazione-menarini.it/... and after having logged in, access the multimedia material.

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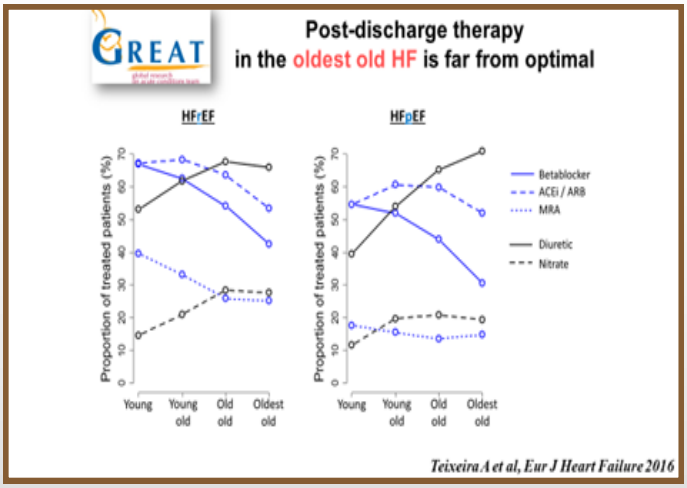
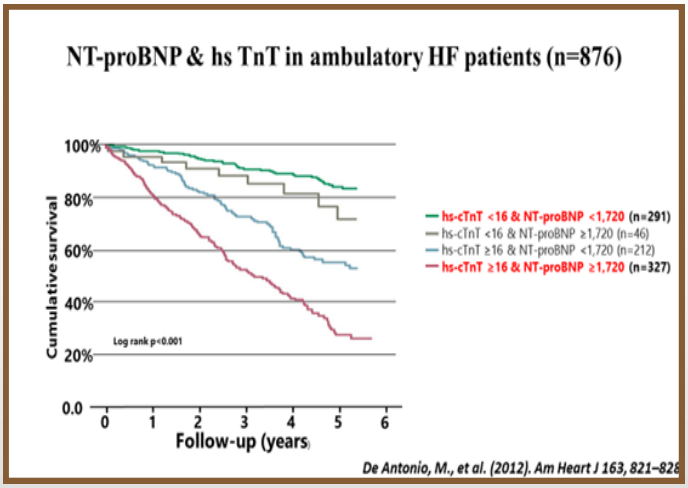
Chronic heart failure and biomarkers

Alexandre Mebazaa
(Paris, France)

Professor Mebazaa from Paris submitted really interesting data on the diagnostic biomarkers applied to prognosis and to the monitoring of the evolution of heart failure. There are various and well-known therapies for patients with heart failure but they are far from optimal, in particular in the oldest old HF patients. Indeed, as the age of the patients rises, the percentage of those in drug treatment decreases. Even more alarming is the statistic showing that less than 20% of treated patients take the target drug dose set out in the Guidelines. Summarising the results, less than 70% of patients with heart failure are pharmacologically treated and less than 20% of them take the target dose. A possible solution to this problem is the use of cardiovascular biomarkers to assess the severity of the disease, to assess the benefits of therapies and to provide information on the need to intensify the drug treatments. Modern cardiovascular biomarkers can give such indications. Natriuretic peptides and troponin are strong markers of severity of heart failure.
The reduction of their haematic levels is strongly connected to the evolution of the disease. Such reduction is a particular reliable indicator to establish prognosis or disease severity in chronic HF. BNP was successfully used in several trials to achieve the targed dose to be used to treat heart failure patients. At this point, Professor Mebazaa presented unpublished data regarding the use of natriuretic peptides in the management of patients with cardiogenic shock. Even in this high-risk condition, the use of biomarkers proved to be of great help for the optimal management of the pharmacological treatment. Biomarkers, until now used for diagnosis of heart failure, are therefore a valuable support for the treatment optimisation.





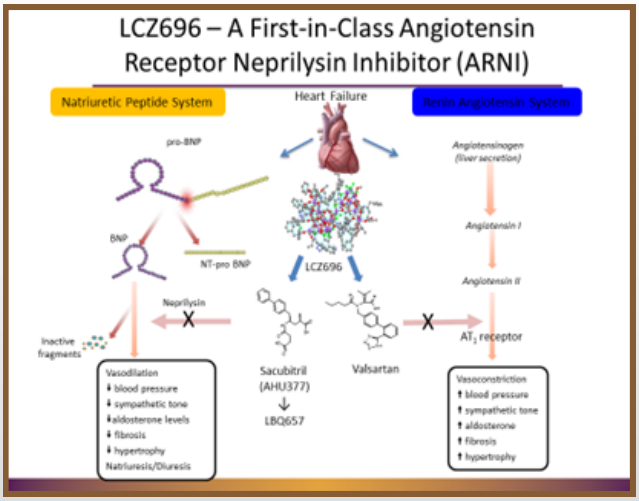
What are the primary biomarkers that have been studied to manage the HF therapy?
Can we monitor the effect of pharmacological treatment with biomarkers?
What are the indications for the use of biomarkers in the management of patients with cardiogenic shock?

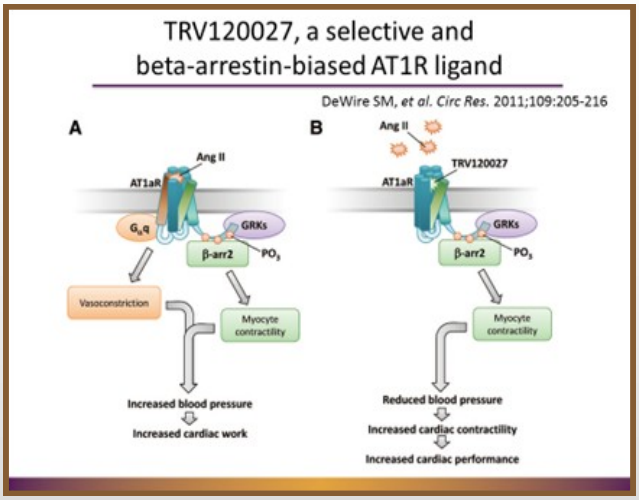
HIGHLIGHTS

New Tools for Heart Failure Treatment: Clinical Data

John Teerlink
(San Francisco, USA)

Professor Teerlink from San Francisco presented the new tools for heart failure treatment showing the analysis of the main clinical trials conducted on patients with heart failure. Some of them are still in early stage of development. Among these new tools, some medications result from well-known pharmacological classes but they present highly innovative characteristics. It is the case of the combination treatments of ACE inhibitors and natriuretic peptides inhibitors presenting, so far, very interesting data. Spironolactone is another well-known molecule that has recently been studied in the treatment of heart failure. Compared to the response observed in the placebo group, TOPCAT (Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist) trial data demonstrate that the drug is effective in reducing the events particularly in populations enrolled in America and not in those randomized from Russia and Georgia. More generally, Spironolactone did raise to a series of doubts about safety effects. Finerenone is another drug in the same class: it has demonstrated superior selectivity and therefore a better safety profile and it maintains a good efficacy profile. TRV027, a new selective biased-ligand angiotensin receptor antagonist of AT1R has no vasoconstrictive effects and stimulates cardiac contractility. It is currently used in phase 1 and phase 2 studies and, according to preliminary data, it seems to have promising characteristics. In the pipeline are molecules with vasodilating effects as Ularitide, a natriuretic peptide synthesized in the kidneys, resistant to endopeptidases with more powerful vasodilating effects. Vericiguat, a soluble guanylate cyclase stimulator that, from literature data, appears to reduce fatal cardiovascular events compared with placebo. Serelaxin is a very interesting drug: it is a protein similar to insulin, the so called pregnancy hormone. Ongoing studies show that it significantly improves survival when compared with placebo. Another kind of agents with vasodilating properties are the Nitroxyl donors. They are potent vasodilators and stimulate RyR2 release enhancing calcium release. Another really interesting drug is Omecamtiv Mecarbil (OM). It is a novel selective cardiac myosin activator that in the ongoing studies showed some really interesting effects, particularly on increasing contractility in acute heart failure.





What are the main combination characteristics of ACE inhibitors and natriuretic peptides inhibitors? Why TOPCAT trials demonstrate differing significant drug effects on different populations? What are the main characteristics of Serelaxin?

HIGHLIGHTS

These are some of the topics addressed during the congress talks. For more in depth information please visit the website of the **Fondazione Internazionale Menarini**, which contains the full versions of the congress talks.

To view the talks by the lecturers of this event click on this link: www.fondazione-menarini.it/... and, after having logged in, access the multimedia material.

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