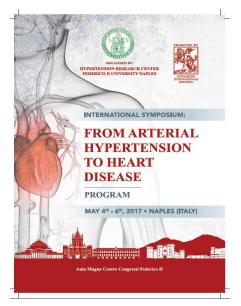
International Symposium: FROM ARTERIAL HYPERTENSION TO HEART DISEASE Naples (Italy), May 04-06, 2017 Highlights

Introduction



Prof. de Simone, chairman of the symposium, opened the congress, with the endorsement of the ESC Council of Hypertension and the European Society of Cardiology. This congress focused on the new strategies for reducing the cardiovascular risk due to the onset of the arterial hypertension, among the main topics discussed in this congress stood out issues on epidemiology, pathophysiology, coronary artery disease, cardiovascular disease and heart failure. One of the main session has been spent in the relationship between sex specificity and the cardiovascular disease. More than 100 cardiologists, specialists in hypertension and young physicians attended the symposium, coming from Italy and all the world.

Giuseppe Schillaci Memorial Lecture

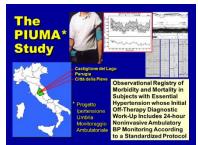


Prof. Verdecchia from Assisi (IT), spoke about Prof. Giuseppe Schillaci in his memorial lecture. Going deeper in his talk, the speaker presented the main steps of Prof. Schillaci splendid career from the beginning in 1985 when he was graduated in Medicine

at the University of Palermo till his last lecture during a meeting held in Perugia, a few days before his departure. Prof. Verdecchia talked

also about the PIUMA Study and other studies coordinated by Prof. Schillaci. More in particular he presented data on the Perugia score, an ECG-LVH score, developed by Giuseppe

Schillaci himself. In a few years Prof.



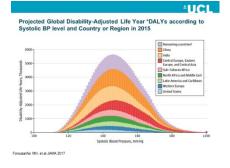
Perugia Score

- At least 1 of the following:
- Typical LV strain
- R wave in aVL + S wave in V₃ > 20 mm (women) or 24 mm
- (men) • Romhilt-Estes score ≥ 5 points

Schillaci evolved by the role of Researcher to the role of Associate Professor, taking care of young physicians not only from the science but also from the human point of view, Prof. Verdecchia highlighted. In conclusion, the speaker pointed out that Giuseppe Schillaci was a very brilliant Researcher as well as a wonderful Physician and a Man signed by a very deep humanity.

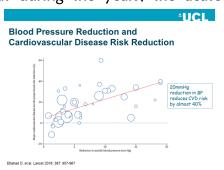
- What's about the main characteristics of the PIUMA study, presented by the speaker?
- What's about the Perugia score, developed by Giuseppe Schillaci?
- What are the main topics of the academic career of Prof. Schillaci, presented by the speaker?
- What are the main publications of Prof. Schillaci presented by the speaker?

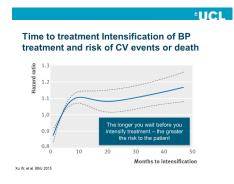
Opening lecture: World impact of arterial hypertension



The World impact of arterial hypertension, was the topic at the core of the lecture of Prof. Williams. At the beginning of his talk the speaker, coming from London (UK), presented very interesting data derived from clinical, registries and epidemiological studies on the effects of high blood pressure in the world-wide population. Going deeper in his lecture, Prof. Williams, pointed out that the largest number of systolic blood pressure-related deaths were due to ischemic heart disease, haemorrhagic stroke and ischemic stroke. In

the main part of his lecture, the speaker highlighted that during the years, the acute complications of hypertension like ischemic heart disease and stroke have been decreased, but have been increased the chronic complications like heart failure, atrial fibrillation, CKD and dementia. In the second part of his speech, Prof. Williams, talked about therapy, starting from the data of the Veteran Administration Cooperative Study published in 1967, where without any statistical analysis the authors stated that the evidence provided by these earlier studies





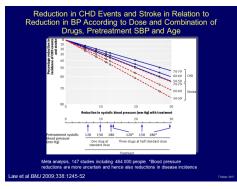
leaves little doubt as to

the value of antihypertensive drug therapy. The speaker went deeper in in this issue, by highlighting that 50 years later, there is still a high proportion of hypertensive patients not well controlled and presented very impressive data on the major problems that dramatically reduce the effect of therapy, like patients' non-adherence, deficiencies of the healthcare systems in approaching the chronic diseases like hypertension and the physician's inertia in starting the treatment.

- Why is high blood pressure the leading global contributor to premature death, from the speaker point of view?
- What do we mean by control, based on the data presented by the speaker?
- What's about the time to the treatment intensification of BP therapy and the risk of CV events or deaths, based on the data presented by the speaker?
- What are the main problems with the global hypertension treatment strategies, presented by the speaker?
- How many are the life years lost due to hypertension, based on the data presented by the speaker?

Pathophysiology of hypertension

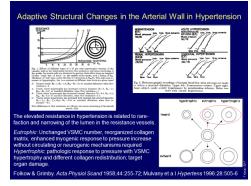
hemodynamic burden of arterial

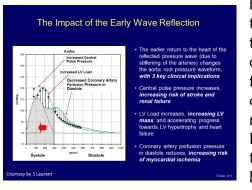


The pathophysiology of the hemodynamic burden of arterial hypertension was the topic at the core of the lecture discussed by Prof. Kahan. The speaker, coming from Stockholm (SE), introduced his talk by presenting data on the reduction of the CHD events in correlation with the BP levels. Going deeper in his lecture, Prof. Kahan spoke about hypertension as an hemodynamic syndrome with three

major components characterized by structural and genetic

factors and a very heterogeneous etiology. In the main part of his talk, the speaker presented very interesting data on the role played by the kidney in the etilogy of hypertension, pointing to the deep relationship between the kidney diseases and the onset of hypertension. Prof. Kahan spoke also about the neurohormonal cardiovascular control, by highlighting that there is very deep correlation between sympathetic activation, the kidney and the onset of

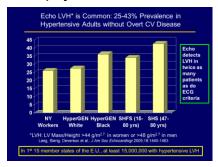




hypertension. In the second part of his lecture, the speaker talked about the role played by remodelling at the level of the small arteries in the early onset of hypertension, the role of the aortic compliance on the blood pressure levels and finally, about the impact of the early wave reflection on the central pulse and LV pressure, leading to the increase of the risk of stroke, renal failure and heart failure. In conclusion, Prof. Kahan pointed out that all these data strongly indicate that hypertension is not a simple disease but primarily an hemodynamic syndrome.

- What is hypertension from the speaker point of view?
- What's about the intrinsically heterogeneous etiology of hypertension, from the speaker point of view?
- What are the main adaptive changes in the arterial wall in hypertensive patients, based on the data presented by the speaker?
- What's about the impact of the early wave reflection, based on the data presented by the speaker?
- What is the role of the aortic compliance on blood pressure and the effect of aging, from the speaker point of view?
- What are the main adaptive structural changes in the arterial wall in hypertension?

Early pre-clinical manifestations of the cardiovascular impact

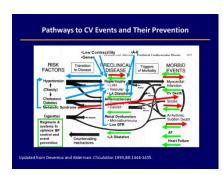


The early pre-clinical manifestations of the cardiovascular impact was the topic of Prof. Devereux presentation. The speaker, coming from New York (USA), talked about the role played by hypertension in the onset of the CV disease, by

> ss Predicts Hypertension in Adults Optimal BP: The Strong Heart St

highlighting its double dimension as risk factor and preclinical disease, leading to the main CV events like MI, Stroke, LVH. Going deeper in

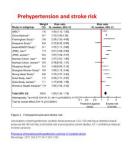
his lecture, Prof. Devereux, presented very interesting data on the role played by the LV hypertrophy in the hypertensive patients, by highlighting that the LV mass is a very strong predictor of hypertension, CV events and also mortality. In



the main part of his talk, Prof. Devereux presented many data on the correlation between the regression of the LVH and the risk-reduction of new-onset diabetes. Finally, the speaker talked about obesity and its role in the development of hypertension and LVH. In conclusion, Prof. Devereux pointed out that through the prevention of hypertension and the reduction of the LVH it is possible to reduce the prevalence and the incidence of the major cardiovascular events.

- Why care about LVH based on the data presented by the speaker.?
- Is the preclinical CV disease reversible, from the speaker point of view?
- Is the LVH regression beneficial from the speaker point of view?
- What's about the association between the carotid atherosclerosis and the LV mass, based on the data presented by the speaker?

Atherosclerosis and progression of hypertension



Atherosclerosis and progression of hypertension, was the topic of Dr. Brguljan presentation. The speaker, coming from Ljubljana (SI), talked about Atherosclerosis from a clinical perspective point of view, about pre-hypertension and hypertension and finally about the early stages of organ damage. Going deeper in her lecture, Prof. Brguljan presented very interesting data on the synergism between hypertension and hyperlipidemia in the onset

of atherosclerosis and on the

correlation between the incidence of pre-hypertension and the stroke risk. The speaker presented a lot of data given by the main clinical trials running in hypertensive patients, like the Framingham and the TROPHY study and finally she spoke about IDACO, that is the International Database on Ambulatory blood pressure in relation to Cardiovascular Outcomes study and presented very impressive data on the main abnormalities linked with the presence of hypertension, like the ones in the retinal microcirculation, or at the coronary artery level. Prof. Brguljan

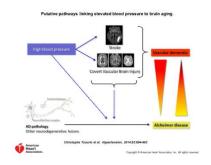


IDACO HRs (vs. NT) associated with masked HT No. of No. of subjects events No. of No. of subjects events

spoke also about the correlation between microalbuminuria and hypertension and the prognostic value of the microalbuminuria and on the correlation between the presence of obstructive sleep apnea and atherosclerosis. In conclusion, the speaker pointed out that the correlation between atherosclerosis and the progression of hypertension is very strong based on also the data of the IDACO study.

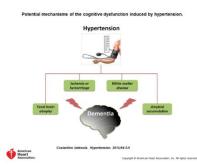
- What are the main mechanisms linking hypertension and hyperlipidemia in the pathogenesis of the atherosclerosis, based on the data presented by the speaker?
- What's about the relationship between the abnormalities of the retinal microvascular structure and the risk of mortality from IHD and stroke?
- Is it the time to measure the microalbuminuria in the hypertensive patients?
- What is the prognostic value of the microalbuminuria, based on the data presented by the speaker?

Cerebrovascular impairment and cognitive dysfunction



Prof. Sierra coming from Barcelona (ES) spoke about cerebrovascular impairment and cognitive dysfunction, by presenting very interesting data on the most common types of brain lesions, like white matter hyperintensities, silent infarcts and microbleeds. Going deeper in her lecture, Prof. Sierra spoke about the correlation between high blood pressure and cognitive decline, by highlighting that the strongest evidence comes from observational studies and about the relationship between hypertension and Alzheimer

disease. In the main part of her lecture, the speaker talked about the main mechanisms linking elevated BP levels to the cognitive impairment or dementia and presented very interesting data on functional, structural, pharmacologic and stroke-related factors. In the second part of her talk, Prof. Brguljan, spoke about some unsolved issues and more in particular she presented some data on the effect of the antihypertensive therapy on the microvascular brain damage and the cognitive impairment, by highlighting that,



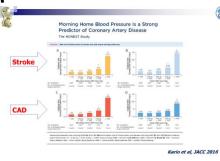
based on the majority of the observational studies, an association between mid-life HTN and late-life cognitive decline or dementia has been found, but this correlation has not been

	Summary of Main Longitudinal Studies on the
Ì	Relationship between Antihypertensive Drug Use and
	Risk of Dementia
•	An association between mid-life HTN and late-life cognitive decline or dementia has been found in the majority of observational studies
•	Observational studies point to some benefit of anti-HTN treatment or risk of dementia
•	The longer the duration of treatment, the stronger the preventive effect. Treatment seems more effective in the youngest old than in the oldest persons
•	Few studies suggest a greater effect of some classes of antihypertensive therapy, but evidence to date is limited and subject to bias
•	Meta-analyses neither prove nor disprove the efficacy of anti- hypertensive treatment on dementia risk

proved or disproved by the main meta-analyses based on the main randomized clinical trials performed on this topic. In conclusion, Prof. Brguljan pointed out that emerging evidences suggest that hypertension may also play an important role in the development of cognitive decline, Alzheimer disease and vascular dementia, but the BP levels that should be targeted for achieving an optimal perfusion while preventing the cognitive decline are still being debated.

- Are there cognitive domains specifically affected by the microvascular brain damage?
- What is the effect of the antihypertensive therapy on the microvascular brain damage and on the cognitive impairment?
- What's about the relationship between the arterial structure and function with the vascular cognitive impairment from the speaker point of view?
- What are the main potential mechanisms of the cognitive dysfunction induced by the hypertension?

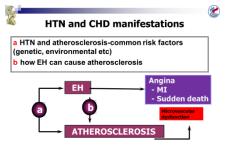
Angina pectoris and other CHD manifestations in hypertensive patients



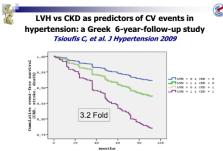
Angina pectoris and other CHD manifestations in the hypertensive patient was the topic at the core of Prof. Tsioufis presentation. The speaker, coming from Athens (GR), presented very interesting data given by clinical studies on epidemiology, pathophysiology, screening for CAD and finally on the treatment of hypertension. Going

deeper in his lecture, Prof. Tsioufis spoke about the correlation

between hypertension and the CV events in untreated patients and presented very interesting data on the relationship between pulse pressure and the risk of CV events in patients affected by atherosclerosis, by highlighting that the morning home blood pressure is a very strong



predictor of coronary artery disease. In the main part of his lecture, the speaker talked about the CHD pathophysiology in hypertensive patients, by highlighting the central role played by

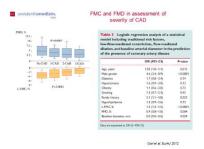


the endothelial dysfunction. Speaking about the screening of CAD in hypertensive patients, Prof. Tsioufis presented very interesting clinical cases of hypertensive patients affected by CAD and highlighted that in intermediate and high risk patients a diagnostic stress test should be considered. Finally, the speaker talked about treatment and presented very interesting data on the best BP targets to be achieved in HTN and CAD patients based on the

2013 ESH/ESC guidelines, the SPRINT and the more recent meta-analyses, by highlighting that for any 10 mmHg fall in SBP, irrespective of the baseline BP there is a 20% of major CVD risk reduction.

- What are the key points of the interaction between hypertension, dyslipidemia and the risk of atherosclerosis?
- What's about the parallel structural and functional adaptations of the coronary arteries in the hypertensive patients, from the speaker point of view?
- What's about the stroke risk reduction for any 10 mmHg fall in SBP, based on the data presented by the speaker?
- What's about the diagnosis of CAD in HTN patients, from the speaker point of view?
- How to diagnose CAD in a hypertensive patient, from the speaker point of view?
- What are the main mechanisms of abnormal CFR in LVH hypertensive patients?

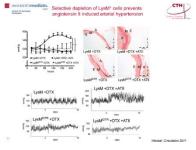
Endothelial dysfunction



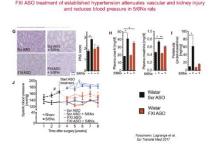
The endothelial dysfunction was the topic discussed by Dr. Wenzel. The speaker, coming from Mainz (DE), presented very interesting data about the biological mechanisms leading to the onset of the endothelial dysfunction and its clinical consequences. Going deeper in his lecture, Prof. Wenzel spoke about the correlation between

the impaired endothelial function and the onset of CV events in

patients with stable CAD, by presenting very interesting data on the flow-mediated dilation that measures the state of the endothelial function in human, and its predictive value in CAD patients. In the main part of his lecture, the speaker talked about the relationship between hypertension, angiotensin II,



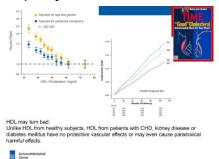
inflammation and CVD, by highlighting that, based on pre-clinical studies running in mice, there is a tight correlation between angiotensin II, the onset of inflammation at the vascular



level and the onset of MI,. Finally, Prof. Wenzel, presented very interesting data on the involvement of the main coagulation factors in the development of the endothelial dysfunction and spoke about the role of specific FXI-targeted anticoagulants as antithrombotic agents in mice affected by CVD. In conclusion, Prof. Wenzel pointed out that vascular inflammation is casually involved in the pathogenesis of hypertension.

- What is the main source of superoxide based on the data presented by the speaker?
- What's about the inflammatory paradigm of atherogenesis?
- What's about the interaction between immune cells, platelets, coagulation factors and the vessel wall from the speaker point of view?
- How to measure the endothelial function in humans?
- What's about the correlation between FMD and CVD in primary prevention?
- What's about the correlation between FMD and FMC (flow mediated constriction) from the speaker point of view?

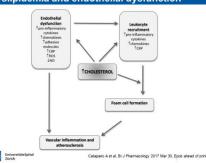
Dyslipidemia and arteriosclerosis



Prof. Sudano from Zurich (CH), spoke about dyslipidemia and arteriosclerosis. At the beginning of her lecture, the speaker presented very interesting data on the major atherosclerotic cardiovascular risk factors by highlighting the role played by the LDL-Cholesterol. In the main part of her lecture, Prof. Sudano talked about cholesterol and its metabolism, by highlighting that also the HDL, more in particular those ones in Dyslipidemia and endothelial dysfunction particular those ones in

patients affected by MI,

can induce endothelial dysfunction and presented very interesting data on the main mechanisms leading to the onset of the major lesions of the endothelium leading to the onset of the endothelial dysfunction. Prof. Sudano pointed out that we have to think about LDL-Cholesterol in the same way of smoking, two different risk factors, that share one characteristic: to be cumulative over time during the life. The speaker presented very interesting data on the combined effects of LDL-C and

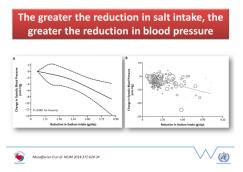


fatal MI or St fourier 19% RRR 33% RRR HR 0.67 (95%CI 0.59-0.77) P+0.00001 R 0.81 (95%CI 0.70-0.93 Stroke onfatal MI 4% atal or 2% Univer Zürich

high SBP on the onset of the cardiovascular events. In the second part of her lecture, the speaker talked about therapy, by highlighting the role played by statins and PCSK9 inhibitors taken in combination for the reduction of the CV events in hypercolesterolemic patients. In conclusion, Prof. Sudano, pointed out that the efficacy of the main antidyslipidemic drugs, takes advantages of their combination and helps patients in understanding how important is to use them continuously.

- What's about the role of HDL in patients with CHD, from the speaker point of view?
- Wat's about the correlation between dyslipidemia and endothelial dysfunction, based on the data presented by the speaker?
- What is the risk effect of LDL-C over time from the speaker point of view?
- What's about the combined effect of LDL-C and SBP on cardiovascular events from the speaker point of view?

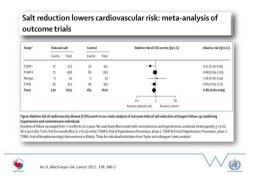
The salt affair



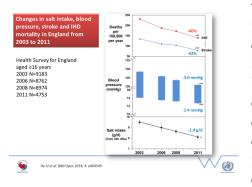
The salt affair, was the topic discussed by Prof. Cappuccio. More in particular the speaker, coming from Coventry (UK) presented very interesting data on the role played by salt on blood pressure, by highlighting that any metaanalysis performed on the correlation between salt intake and the blood pressure levels, shows that the reduction in salt intake causes a direct reduction in blood pressure.

Going deeper in his lecture, the speaker highlighted that

there are limited data given by randomized clinical trials on the effects of the salt reduction on the prevalence of the cardiovascular risk and this is the reason for the onset of the new salt debate, characterized by two questions, the first one about the presence or not of something like a J-curve effect between sodium intake and CV events and the second



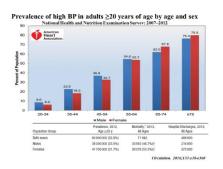
one about the possibility of any harm in reducing the salt intake towards the recommended



targets. In the main part of his lecture, Prof. Cappuccio presented very interesting data in order to find comprehensive answers to these two questions and highlighted that the published studies on the correlation between the salt reduction intake and the raise of the cardiovascular events present methodological errors that determine the loss of any affordability in their results. In conclusion, Prof. Cappuccio pointed out that the population salt reduction programmes are feasible, effective, powerful and lifesaving.

- What's about the role of the salt in ours diet?
- What is the relationship between a higher salt intake and the risk of stroke, based on the data presented by the speaker?
- What's about the common methodological errors of the studies demonstrating a relationship between the reduction of the salt intake and the raise of the cardiovascular events?
- What's about the cost-effectiveness of the salt-intake reduction from the speaker point of view?
- What are the main components of a strategy to reduce the population saltintake, presented by the speaker?

Sex differences in arterial hypertension

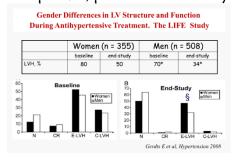


Prof. Cifkova from Prague (CZ), spoke about the sex differences in arterial hypertension. In her talk, the speaker presented very interesting data on the epidemiology of hypertension, the BP levels around the menopause, the large clinical trials in hypertension, the relationship between oral contraception and BP and finally on the target organ damage in hypertension. Going deeper in her lecture, Prof. Cifkova spoke about epidemiology, by highlighting that there are differences

between men and women in the prevalence of hypertension and the related outcomes. The speaker talked also about the onset of hypertension in postmenopausal women and the related predisposing factors. More in particular, Prof. Cefkova presented very interesting data on the relationship between aging, BMI and the increase in BP levels in postmenopausal women and spoke about the correlation between the ovarian failure and the onset of hypertension. Speaking

Calcium antagonists	↑ Peripheral edema in wome
ACE inhibitors	↑ Cough in women

about the large clinical trials, Prof. Cifkova highlighted that have been recruited more men than women and this factor can introduce a bias in the event rate detection divided for sex. The speaker, presented also very interesting data on the different effects in men and women



of the main antihypertensive drugs. Finally, Prof. Cefkova talked about the target organ damage in hypertension, by highlighting that the data given by the main clinical trials running in hypertensive patients, demonstrate that female gender is more exposed than male to target organ damage. In conclusion, Prof. Cefkova pointed out that women develop target organ damage in hypertension more often than men and its regression is less common.

- Does the BP increase in relation to menopause in women?
- What's about the correlation between the concomitant increase in age and BMI and the raising of the prevalence in hypertension in postmenopausal women?
- What's about the correlation between the ovarian failure and the rise of blood pressure, based on the data presented by the speaker?
- Should hypertension be treated differently in men and women, based on the data presented by the speaker?
- What's about the gender-related differences in adverse events due to the antihypertensive treatment?
- Why is RAS important in pregnancy?

Sex differences in incidence and outcome of coronary heart disease

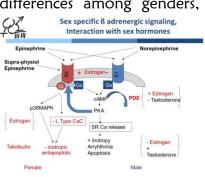


The sex differences in incidence and outcome of coronary heart disease was the topic discussed by Prof. Regitz-Zagrosek in her lecture. The speaker, coming from Berlin (DE), presented very interesting data given by european projects and an europeaan network on Gendermedicine. Going

deeper in her talk, Prof. Regitz-Zagrosek spoke about the GenCAD project and more in particular, about the classical

and the novel risk factors and their prevalence in men and women, by highlighting that gender is one of these novel risk factors for CHD. In the main part of her lecture, the speaker presented very interesting data on Pathophysiology and the differences among genders, by highlighting that coronary

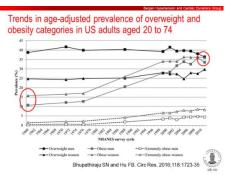
	Age dep	endent CAD manifest	ations in won	nen
atherosclerosis	premenopause low risk	45-65 years low-intermediate risk	older age high risk obstructive CAD	
→ total burden atherosclerosis		less obstructive CAD than in men coronary microvascular disease Coronary dissections	, non-obstructive CAD	
	EUGENMED stud	y group, EHJ, 2015	→ ageing	GenCAD



spasms, dissections and dysfunction are more frequent in women than in men. In the second part of her lecture, the speaker talked about the sex-differences in the sudden cardiac death prevalence, by highlighting that men present a doubled risk compared to women and presented very interesting data on the protective factors present in women but not in men. In conclusion, Prof. Regitz-Zagrosek pointed out that women have a later onset of atherosclerosis, but an equal number of deaths.

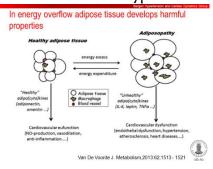
- What's about the differences between sex and gender, from the speaker point of view?
- What's about the GEnCAD project, based on the data presented by the speaker?
- What is the prevalence of Tako tsubo in women, based on the data presented by the speaker?
- What's about the sex-differences in ACS manifestations, based on the data presented by the speaker?
- What are the main triggers and inhibitors of the sudden cardiac death?

Obesity and metabolic syndrome

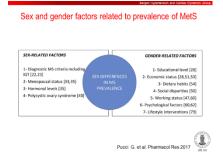


Prof. Gerdts from Bergen (NO), presented very interesting data on obesity and the metabolic syndrome. At the beginning of her lecture, the speaker talked about the prevalence of obesity and diabetes in the USA population and their relationship with the prevalence of LV hypertrophy. Going deeper in her lecture, Prof. Gerdts presented very interesting data on the effect of obesity on the LV function and on the cardiac benefit of the antihypertensive treatment, by highlighting that obesity reduces the cardiac benefit of the antihypertensive

treatment and impair the LV function. In the main part of her presentation, the speaker talked about the pathophysiology of the LV hypertrophy in obese patients and presented a lot of very interesting data on all the associated factors like the harmful properties of the adipose tissue in obese patients and the obesity-related genes and their sex-differences due to aging. In the second part of her lecture, Prof. Gerdts talked about the relationship between metabolic syndrome and obesity, by highlighting that there



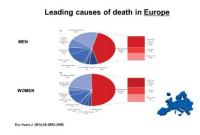
are many sex and gender related factors that influence the metabolic syndrome prevalence



and presented very interesting data on the effect of DM and MetS on the myocardial energetic efficiency and on the correlations between fitness and MetS in obese patients. Finally, the speaker presented other data on the correlation between fitness and hypertensive subtypes in hypertensive patients. In conclusion, the speaker pointed out that obesity reduces the cardiac benefit of the antihypertensive treatment and that the abdominal adiposity influences the heart more in women than in man.

- What's about the harmful properties of the adipose tissue in case of energy overflow?
- What's about the effect of diabetes mellitus and MetS on the myocardial energetic efficiency, from the speaker point of view?
- What are the main sex and gender factors related to the prevalence of MetS, based on the data presented by the speaker?
- What is the prevalence of MetS in women compared to men?

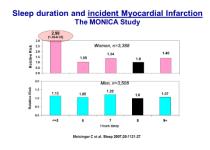
Is sex-difference real or due to co-factors



The main topic at the core of Prof. Stranges presentation, was the role of co-factors in the onset of the sex-differences in the outcome of CVD and hypertension. The speaker, coming from Ontario (CA), talked about the epidemiological data, the

Bradford-Hill Criteria and about the supportive evidence for sex difference in CVD in primary prevention. Going

deeper in his lecture, Prof. Stranges, presented very interesting epidemiological data on the prevalence of CVD and about the residual risks for CVD and other disease in women and in men, by highlighting that women present a higher prevalence and residual risk than men. In the main part of his lecture,



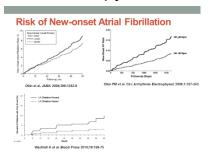
Prof. Stranges spoke about the main supportive evidence for sex differences in CVD and presented very interesting data on the sleep problems as an emerging risk factor for CVD and



on the ischemic heart disease pathophysiologic model in women, by highlighting that it is time to recognize the presence of a female specific Ischemic Heart Disease. Finally, the speaker talked about the topics to be implemented for a better detection of the sex differences in CVD. In conclusion, Prof. Strangers pointed out that there are a lot of differences between women and men also in the way to see themselves.

- What are the main study populations on the sex differences presented by the speaker?
- What are the main topics to be implemented from the speaker point of view for a better detection of the sex differences in CVD?
- What is the model of ischemic heart disease pathophysiology in women, presented by the speaker?
- What's about poor sleep as a new risk factor for CVD, based on the data presented by the speaker?
- What's about the sleep duration and the incidence of Myocardial Infarction in the MONICA study?
- What are the main sex differences in CVD, based on the data presented by the speaker?

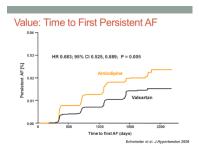
Arterial hypertension and atrial fibrillation



The main topic at the core of Prof. Wachtell presentation, was the relationship between arterial hypertension and atrial fibrialltion. The speaker, coming from Oslo (NO), presented very interesting data, on the role of atrial fibrillation in

increasing the risk of stroke and the cardiovascular morbidity and mortality. Prof. Wachtell spoke also about the correlation

between hypertension and atrial fibrillation and the further increase in cardiovascular risk. Going deeper in his lecture, the speaker presented very interesting data on the relationship between the RAA system and the atrial fibrillation and on the



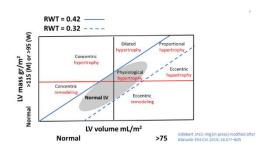
The cardiovascular continuum



main principles for the AF management, with a particular focus on drugs, like ACEi, ARB and beta-blockers. Finally, Prof. Wachtell, spoke about the relationship between AF and the prevalence of HF and raised the question about AF as a target organ damage disease. In conclusion, the speaker, pointed out that in the cardiovascular continuum there is a preferred linkage between atherosclerosis, hypertension and atrial fibrillation.

- Is atrial fibrillation a target organ damage, based on the data presented by the speaker?
- What's about the correlation between atrial fibrillation and heart failure?
- What's about the relationship between the antihypertensive treatment and the risk of atrial fibrillation, from the speaker point of view?
- What's about the prevention of AF by RAS-inhibition?
- Do Beta-blockers prevent atrial fibrillation beyond its blood pressure lowering properties?
- Can beta-blockers prevent AF if the patient is in sinus rhythm?

The importance of preclinical left ventricular dysfunction



Prof. Gillebert from Gent (BE) spoke about the importance of the preclinical left ventricular dysfunction in hypertensive patients. Going deeper in his lecture the speaker talked about the echocardiographic parameters with prognostic significance, like LV mass, the LA volume index, the EF and other novel parameters like the myocardial strain, the strain rate and the arterial wave reflections. More in particular Prof. Gillbert

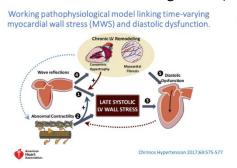
presented very interesting data on LV mass and geometry, by highlighting the role of the LV volume from a diagnostic but also prognostic point of view. Speaking about the systolic function, Prof. Gillbert highlighted that EF is a ratio, so it works only if the numerator and the denominator go in opposite directions. Going deeper in his talk, the speaker presented very interesting data on the global longitudinal strain, that is a more valuable marker



JACC Img 2017 (in pre

LA systolic strain or reservoir function

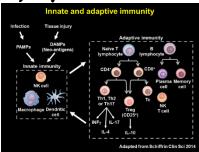
of the left ventricular function than EF. Prof. Gillebert presented also data on the left atrial function and on the afterload and its capacity to induce diastolic dysfunction in a hypertension and obese setting. Finally, the speaker talked about the peak longitudinal strain,



by highlighting that there is an inverse correlation between contractility and systolic wall stress and presented very interesting data on the working pathophysiological model linking time-varying myocardial wall stress and diastolic dysfunction. In conclusion, Prof. Gillebert pointed out that well established prognostic indices are LVMI, RWT, LAVI, TR velocity and e/e'.

- Is Ejection Fraction a friend or a foe, from the speaker point of view?
- What's about the reflection magnitude and its capacity to be an independent predictor of the onset of heart failure?
- What are the key point of the working pathophysiological model linking timevarying myocardial wall stress and diastolic dysfunction?
- What's about the impaired systolic function and the afterload in CABG patients?
- What are the main characteristics of the Late-systolic wave reflections?

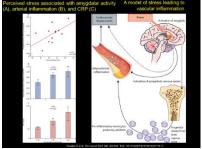
Inflammation and the immune system in the hypertensive injury



Inflammation and the immune system in the hypertensive injury, was the topic at the core of Prof. Schiffrin presentation. The speaker coming from Montreal (CA), at the beginning of his lecture talked about the innate and the adaptive immunity

with a particular focus on the role played by the Т lymphocytes in the production of the inflammatory cytokines.

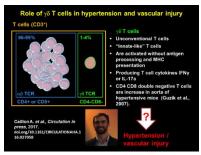
Prof. Schiffrin presented also very interesting data on the role of the monocyte/macrophages in the determination of the vascular damage in a hypertensive setting. In the main part of his lecture, the speaker presented very interesting data given by



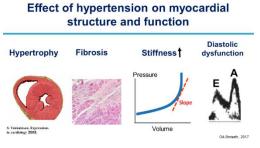
preclinical studies on the role

played by T cells in the onset of hypertension, by highlighting the tight correlation between immune system, inflammation and hypertension. In the second part of his presentation, Prof. Schiffrin talked about the role played by T cells in the onset of the human hypertension, by highlighting the correlation between immune system and hypertension also in humans and the xð T lymphocytes as the main cellular line involved.

- What's about the role of the T cells in hypertension and vascular injury, from the speaker point of view?
- What's about the correlation between angiotensin II and T lymphocytes?
- What's about the role of the T regulatory cells in angiotensin II-induced hypertension and vascular injury?
- What's about the model of stress leading to vascular inflammation presented by the speaker?
- What's about the study of patients with grade I essential hypertension treated with mycophenolate mofetil, based on the data presented by the speaker?



Mechanisms of HFpEF in arterial hypertension



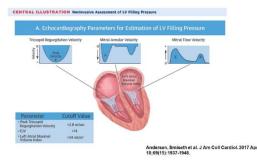
The mechanisms of HFpEF in arterial hypertension was the topic at the core of Prof. Smiseth presentation. The speaker coming from Oslo (NO), at the beginning of his lecture talked about the effects of hypertension on the myocardial structure and function starting from hypertrophy till the late onset of diastolic dysfunction. Going deeper in his lecture, Prof. Smiseth talked about

the three

hallmarks of the diastolic dysfunction, like the impaired relaxation, the loss of restoring forces and the increased diastolic stiffness, all of them producing the elevation of the LV filling pressure as a compensatory process. In the main part of his lecture, the speaker presented very interesting data on the prognostic significance of the left ventricular

Effect of hypertension on myocardial
structure and functionHypertrophyFibrosisStiffnestDiastolic
dysfunctionImage: Stiffnest of Colspan="3">Diastolic
dysfunctionImage: Stiffnest of Colspan="3">Image: Stiffnest of Colspan"Image: Stiffnest

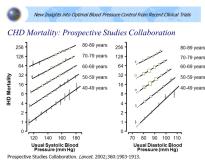
diastolic dysfunction in essential hypertension and on the determinants of e', one of the most important measures for the detection of the diastolic dysfunction. In the second part of his lecture, the speaker talked about the assessment of the systolic function like the longitudinal strain and presented very interesting data on the fast and slow mechanisms of circulatory congestion. Finally, Prof. Smiseth spoke about the obese phenotype very common in HFpEF patients, by highlighting that this is a syndrome characterized by the presence of a lot of



inflammatory processes in liver and other organs than simply the heart and presented very interesting data on the main phenotypes linked with the onset of HFpEF. In conclusion, the speaker pointed out that the concept that HFpEF is due to high blood pressure causing diastolic dysfunction is too simplistic, because in most cases HFpEF has an heterogeneous etiology.

- What's about the assessment of the diastolic dysfunction based on the data presented by the speaker?
- What are the main measures for the assessment of the systolic function, from the speaker point of view?
- What's about the fast and the low mechanisms of circulatory congestion?
- What are the main extracardiac mechanisms involved in the HFpEF onset, from the speaker point of view?
- What are the main echocardiographic parameters for the estimation of the LV filling Pressure, from the speaker point of view?

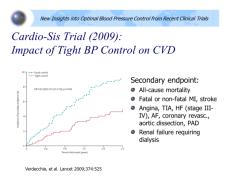
New insight into optimal blood pressure control from recent clinical trial



The new insight into optimal blood pressure control from recent clinical trial was the topic Prof. Levy talked about. The speaker coming from Framingham (USA), focused his talk on hypertension as a risk factor for CVD, the key clinical trials and finally on the implications for the BP guidelines. Going deeper in his lecture, Prof. Levy presented very interesting

data on the roots of CVD prevention starting from

the first assumptions in 1961, when the main established risk factors were age, sex, blood pressure, Cholesterol level and LVH, by highlighting that also today age, sex and BP levels are the main risk factors for CHD mortality without any J-curve effect. In the main part of his talk, Prof. Levy talked about the results of the key clinical trials and presented very interesting data given by the SHEP,



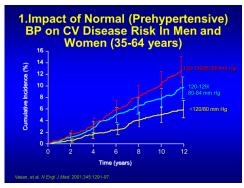
Syst-Eur, Cardio-Sis and SPRINT trials on the major endpoints linked with SBP and DBP reduction, by highlighting that in all these studies the lower BP levels the better in CVD risk

resholds for Initiation of Drug Treatment:					
fferences in Guidelines					
Guideline	Age ≥60 years				
JNC 8	150/90				
ASH/ISH	140/90				
ASH/ISH ESH/ESC	140/90 140/90				

reduction were achieved. In the second part of his lecture, the speaker discussed about the discrepancies among the different Guidelines recommendations on the BP threshold for initiating the drug treatment. In conclusion, Prof. Levy, pointed out that, based on the data of the main clinical trials presented, for patients with hypertension, the optimal target of treatment should be a systolic BP lower than 120 mmHg.

- Does hypertension treatment effect, match the observational data, based on the data presented by the speaker?
- What's about the impact of a tight BP control on LVH and CVD, based on the Cardio-Sis trial data?
- What are the main results of the SPRINT trial in the elderly, based on the data presented by the speaker?
- What's about the implications for BP guidelines, from the speaker point of view?
- What are the main predictions of Prof. Levy about the future blood pressure guidelines?

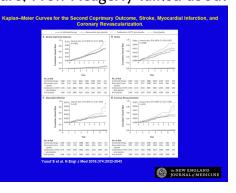
Should we treat early and lower?



"Should we treat early and lower?" was the topic of Prof. Heagerty presentation. The speaker coming from Manchester (UK), at the beginning of his lecture presented very interesting data on the impact of the normal BP on the CVD risk in men and women, by highlighting that for any BP level there is a risk for CVD. Going deeper in his lecture, Prof. Heagerty talked about

SBP and its raising with aging due to the

increasing stiffness of the arterial tree and presented very interesting data given by the Framingham study, demonstrating that to treat earler is better. In the main part of his lecture, the speaker talked about the need for starting earlier the antihypertensive treatment and presented very interesting data on the best BP threshold



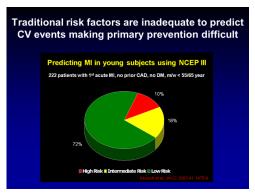
Condenation + Hydrochianedrainik (N=4200) 240 (4.1)	Plander (N=1345)	Hased Rate (MNLO)	
	279 (4.4)	8.93 (0.09-1.10)	0.45
312(4.9)	328 (5.2)	8.85 (8.81-111)	0.51
19-3.5	144(5.7)	6.5713.75-1.001	0.36
110.20	94(3.2)	581-031-1381	0.54
101-02-40	139(2.7)	89138.0-1.01	0.48
52(0.0)	62:0.00	0.8+(0.50-1.22)	0.34
26416	418.0	0.33 (0.07-1.40)	0.18
21.00.0	2418.51	0.7218-0-1.271	0.26
64-(1.0)	74(5.2)	8.84 (0.42-1.23)	0.39
51-00-00	44-13-23	8.N(831-186)	0.35
36.6.6	141-0.5	0.0010-0.00	0.78
200,0070(4.0)	20,004 (1.1)	1.07(0.00-1.2%)	0.46
110-05-05	BRICK.B.	84648-110	0.45
112	118		
54	18		
	14.		
184		88738.06-1.03	0.88
	19 (12) 19 (24) 20	N(22) H(23) 159,84 199,25 20,941 40,28 20,441 40,28 20,441 40,28 20,441 40,28 20,441 40,28 20,441 40,28 20,444 40,28 20,444 40,28 20,444 40,28 20,444 40,28 20,444 40,28 20,444 40,28 20,444 40,38 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24 20,444 24	D/2.0 M/2.0 R0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 240.0 40.0 0(0.70-10) 240.0 40.0 0(0.70-10) 240.0 40.0 0(0.70-10) 240.0 40.0 0(0.70-10) 240.0 40.0 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10) 100.0 0(0.70-10) 0(0.70-10)

to be achieved in

order to reduce the CVD events. Prof. Heagerty talked also about the data given by the HOPE-3 study, running in intermediate risk patients with normal BP, by pointing out that the events decreased and were significant only in patients with an entry pressure below 140 mmHg. In conclusion, the speaker highlighted that we can treat earlier with a short-term use of statins and lower at least to 130 mmHg.

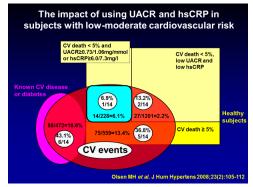
- What are the main effects of treating early from the speaker point of view?
- What's about the BP threshold to be achieved for the best effect on the CVD risk reduction, from the speaker point of view?
- What are the main characteristics of the HOPE-3 trial, based on the data presented by the speaker?
- What's about the data of the TROPHY study, from the speaker point of view?
- What's about the impact of normal BP on CVD risk in men and women, based on the data presented by the speaker?

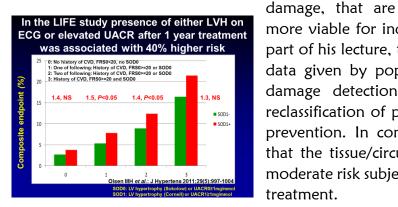
Biomarkers and risk stratification in cardiovascular prevention



Prof. Olsen talked about biomarkers and the risk stratification in cardiovascular prevention. The speaker, coming Holbaek (DK), focused his lecture on the risk stratification and individualized intervention divided in prevention and treatment. Going deeper in his talk, Prof. Holbaek presented very interesting data on the score calculation for the detection of the CV death risk in the population and highlighted that should be important to use the relative risk instead of the absolute risk. Talking about the score calculation for the detection

of the CV event risk, the speaker, pointed out that this score underestimates the CV events in subjects younger than 60 years and especially in women. In the main part of his lecture, Prof. Holbaek talked about the limits of the traditional risk factors, by highlighting that the individual susceptibility is too high and there is a need of too many years of exposure. The speaker presented very interesting data on the markers of subclinical CV

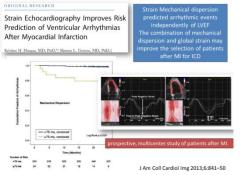




more viable for individualized treatment. In the second part of his lecture, the speaker presented very interesting data given by population studies on the target organ damage detection and highlighted the need for a reclassification of patients in order to raise the effect of prevention. In conclusion, Prof. Holbaek pointed out that the tissue/circulating markers of TOD can identify moderate risk subjects that might benefit from preventive treatment.

- What's about the impact of using UACR and CPR in subjects with low-moderate cardiovascular risk from the speaker point of view??
- What's about the markers of subclinical cardiovascular damage based on the data presented by the speaker?
- What are the main problems of the traditional CV risk factors from the speaker point of view?

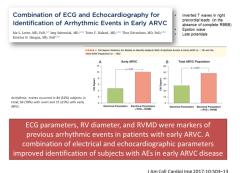
Can new imaging technology improve primary prevention and address therapy?

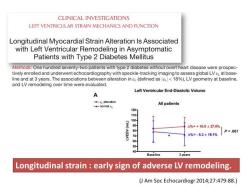


The new imaging technology and its application in primary prevention was the topic Prof. Donal talked about. The speaker coming from Rennes (FR), in his lecture talked about cardio-oncology, about the use of imaging in diabetic and in hypertensive patients, in patients at risk for HF and SD and finally in asymptomatic patients looking for coronary artery disease. Going

deeper in his lecture, Prof. Donal presented

very interesting data given by imaging studies running in CVD patients with the aim to demonstrate that imaging is useful in asymptomatic patients in order to improve outcomes. In the main part of his lecture, the speaker talked about the strain echocardiography and the late gadolinium enhancement applied to subclinical patients in





primary prevention

and presented very interesting data, demonstrating that these new imaging tools are able to improve primary prevention in many field of Medicine. In conclusion, Prof. Donal pointed out that despite the lack of demonstration in the field of the ischemic heart disease, imaging technology looks promising and tools like global longitudinal strain look valuable in hypertensive and diabetic patients.

- What's about longitudinal strain and the early signs of adverse LV remodelling based on the data presented by the speaker?
- What are the main echo diagnostic criteria in chemo-induced cardiomyopathies, based on the data presented by the speaker?
- What's about the relationship between global longitudinal strain and the risk of asymptomatic HF from the speaker point of view?
- What's about the combination of ECG and Echocardiography for the identification of arrhythmic events in ARVC patients?

What are the crucial differences in hypertension across the Atlantic?

	JNC8		JNC7	
	Targets	Drugs	Targets	Drugs
>80 y.o		No-black:	< <u>140</u> /90	<u>Tiazidas</u> If not: ACEs, ARBs, CCBs, Bbloq.
60-80 y.o	<150/90	ACEs, ARBs, tiazida, CCBs. Black: Tiazida or CCBs.		
<60 y.o	<140/90			
DM	<140/90	ACEs, ARBs, tiazida, CCBs.	< <u>130/80</u>	ACEs, ARBs, tiazidas, CCBs, Bbloq
CKD NO proteinuria		ACES or ARBs		ACEs or ARBs

Prof. Zamorano in his lecture talked about the crucial differences in hypertension across the Atlantic. The speaker coming from Madrid (ES), started his talk, by presenting the main differences in methods, definitions, recommendations, objectives, drug treatment and entity between JNC7 and 8. Going deeper in his lecture, Prof. Zamorano presented very interesting data given by the major clinical trials in hypertensive patients, on the main JNC8 recommendations about the conditions for starting

the treatment in different populations from adults to youngers with comorbidities like CKD and diabetes and belonging to different races. In the main part of his lecture, Prof. Zamorano talked about the 2013 ESH/ESC guidelines for the management of hypertension and the classes of recommendations and the levels of evidence. More in particular the speaker presented very interesting data on some topics of these guidelines like the epidemiological aspects on hypertension, the definition

Definitions of hypertension by office and out-of-office blood pressure levels (mmHg) Category Systolic Diastolic Office BP ≥140 and/or ≥90 Ambulatory BP - Daytime (or awake) ≥135 and/or ≥85 and/or - Nighttime (or asleep) >120 >70 - 24-hour ≥130 and/or 280 Home BP ≥135 and/or 285 COPEAN ELECTRAN Eur Heart J, 2013; 34: 2159-2219 Hypertens, 2013; 31: 1281-1357 Blood Programs, 2013: 192-279

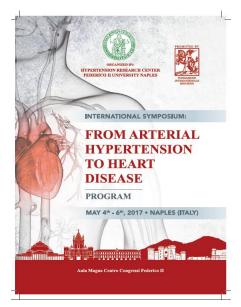
and the classification of the office blood pressure levels, the out-of-office measurements, the assessment of the cardiovascular risk, the treatment strategies like lifestyle changes and the choice of drugs in monotherapy or in combination. Prof. Zamorano talked also about the

	JNC8		ESC2013		
	Targets	Drugs	Targets	Drugs	
>80 y.o		No-black: ACEs, ARBs, tiazida, CCBs Black:	<150/90		
60-80 y.o	<150/90		<150/90 < <u>140</u> /90	Bbloq, ACEs, ARBs, tiazida,	
<60 y.o	<140/90	Tiazida or CCBs.	<140/90	CCBs.	
DM	<140/90	ACEs, ARBs, tiazida, CCBs.	<140/ <u>85</u>	ACEs or ARBs	
CKD No proteinuria		ACEs or ARBs	<140/90	ACEs or ARBs	

treatment strategies in special populations like the young adults, the elderly, the women and the patients affected by diabetes, heart and cerebrovascular diseases and nephropathy. Finally, the speaker discussed about the main differences in targets and drugs between JNC8 and the ESC2013, based on age and on the presence of comorbidities like DM and CKD. In conclusion, Prof. Zamorano, pointed out that guidelines improve knowledge, but it is necessary to apply them to real

patients with their individual settings.

- How to assess the risk from the speaker point of view?
- What are the main differences between JNC8 and JNC7 based on the data presented by the speaker?
- When and how treat the hypertensive patients, based on the data presented by the speaker?
- What's about the definition and the classification of the office blood pressure levels?
- What's about the ambulatory and the home blood pressure measurements based on the data presented by the speaker?



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.