2nd Branislav "Brano" Radovancevic Heart Failure Forum

Innovations and New Treatment Strategies in Heart Failure

Programme and abstract book

24th - 26th September 2009,
Portorož, Slovenia

TEXAS HEART INSTITUTE
at St. Luke's Episcopal Hospital

Advanced Heart Failure and Transplantation Programme
Department of Cardiology
University Medical Center Ljubljana, Slovenia

Innovations and New Treatment Strategies in Heart Failure

Second International Branislav Radovančević Heart Failure Forum

Final Programme and Book of Abstracts

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Dear friends and colleagues,

We would like to once again welcome you to our second Branislav " Brano" Radovancevic Heart Failure Forum on "Innovations and New Treatment Strategies in Heart Failure" in Portoroz, Slovenia.

Last year the first conference was a tremendous success and we have received overwhelming positive feedback from many attendees and faculty. Conferences such as this will allow continuation of Brano's vision and dedication to research in the treatment of advanced heart failure and his enthusiastic work in transplantation and mechanical circulatory support research.

Our 2nd Brano's Heart Failure Forum offers a comprehensive update on the state of the art treatment of the heart failure disease including acute heart failure, heart failure in valve disease, dilated cardiomyopathy, heart transplantation, among others .

The faculty of the Brano's Heart Failure Forum has established international reputation as both clinicians and leaders in their fields. We are anticipating an audience of clinicians treating the basic cardiac disease issues, interventional cardiologists, cardiac surgeons, interventional radiologists and specialists treating the end stage heart failure patients. In addition, we are hopeful this meeting will attract many other professionals, residents and students with special interest in this field. The lively interactive discussion between the presenters, expert international faculty panel and the audience should stand out as the most engaging and beneficial aspect of the meeting.

Although Slovenia may be one of the smallest countries in Europe, it has unique, beautiful and varied landscape with gorgeous surroundings, ranging from the Alps to the crystal Adriatic Sea.

Therefore, we are happy to be able to be able to welcome you to Portorož.

prof. Igor D. Gregorič, MD

prof. Bojan Vrtovec, MD, PhD

Presidential address

Spoštovani,

vse udeležence prisrčno pozdravljam na simpoziju o novih načinih zdravljenja srčnega popuščanja. Zbrali ste se ugledni strokovnjaki iz številnih evropskih držav, Združenih držav Amerike in iz Slovenije. Tako kompetentna sestava udeležencev in vaša številčnost dokazuje, da boste obravnavali obolenje, ki zaradi naraščanja zasluži vso pozornost. Obolenja, ki imajo tako razsežnost morajo biti temeljito raziskana, poskrbeti moramo za razvoj metod in tehnik zdravljenja, sodelovati pa morate tudi pri razvoju novih zdravil in podporne tehnologije.

Kongres ponuja edinstveno priložnost vpogleda v najnovejše dosežke na področju napredovalega srčnega popuščanja in seznanjenja z mednarodno priznanimi pristopi, modernimi smernicami, hkrati z možnostjo izmenjave izkušenj.

Za Slovenijo je ta dogodek priložnost za promocijo varovanja zdravja pri nas in za promocijo Slovenije kot dežele z visokim medicinskimi in socialnimi standardi. Po drugi strani pa pridobivanje novih znanj od svetovno znanih strokovnjakov omogoča, da bo ta kongres pripomogel k razvoju še višjih standardov v terciarnih centrih v Sloveniji.

Spoštovani,

želim vam prijetno bivanje v Sloveniji. Upam, da si boste našli tudi čas, da boste lahko spoznali nekatere zanimivosti naše slikovite in lepe dežele.

dr. Danilo Türk
Predsednik Republike Slovenije

Branislav Radovančević

Branislav ("Brano") Radovancevic, a legend in the international heart-failure community. Born in Osijek, Croatia, completed his medical degree in Belgrade, Serbia and became a Resident in the Cardiovascular Surgery Institute "Dedinje" also in Belgrade. In 1984, his passion for therapy of heart disease brought him to Houston, Texas, where he joined the Cardiopulmonary Transplantation Department of the Texas Heart Institute (THI) at St. Luke's Episcopal Hospital, as research fellow. He quickly became an invaluable member of the transplant program. As an emerging leader in immunosuppression, immunology and transplant research, Brano was highly respected nationally and internationally. His early research into therapies to prevent and manage organ rejection was recognized worldwide. As an integral member of THI's animal research team, Brano designed and preformed studies involving myocardial protection during cardiac operations, as well as temporary and permanent mechanical circulatory assist devices, heart valve prostheses, and synthetic vascular grafts. In addition he authored, and co- authored approximately 300 publications, and oversaw the writing of numerous heart failure and transplant protocols. He lectured and traveled extensively throughout all these years, sharing his knowledge and expertise about mechanical circulatory support, heart transplantation, and other heart failure therapies with colleagues and friends. Owing to his tireless efforts, Brano became Associate Director of Transplant Research in 1998 and Director of the Center for Cardiac Support in 2005.

For the annual THI symposium, he developed and hosted the famous "Rodeo Meeting", bringing top cardiologists and surgeons from around the country to debate topics in transplantation. Introduced in 1993, the Rodeo Meeting still exists, having grown steadily throughout the years.

Until his death in September 2007, Brano continued to make invaluable contributions to the field of heart transplantation, particularly immunosuppression of the transplant patient, and to other areas of cardiovascular research. His commitment to the fight against heart disease and his compassion for his patients helped make THI an unparalleled bastion of cardiovascular research. Indeed Brano exemplified the mission Dr. Denton A. Cooley envisioned in 1962, when he founded THI: to reduce the devastating toll of cardiovascular disease through innovative programs in research, education and improved patient care. Although Brano is no longer physically present, his legacy will continue to inspire his medical colleagues and to benefit heart failure patients everywhere.

Programme at a glance

THURSDAY, 24/9/09

14.00 - 14.30	Welcome Address
14.30 - 16.15	Publications and Innovations in Heart Failure
16.15 - 16.30	Break
16.30 - 17.45	Heart Failure in Children
18.00 - 19.00	Workshop: Scientific Publishing

FRIDAY, 25/9/09

8.30 - 10.00	End-Organ Perfusion in Advanced Heart Failure
10-00 - 10.15	Break
10.15 - 11.45	Heart Failure and Coronary Artery Disease
12.00 - 12.45	Satellite Symposium: AstraZeneca
12.45 - 13.30	Lunch Break
13.30 - 15.00	Heart Failure and Valvular Disease
15.00 - 15.15	Break
15.15 - 16.45	Immune Modulation and Stem Cell Therapy
16.45 - 17.15	Coffee Break
17.15 - 19.00	Acute Heart Failure
19.00 - 19.30	Satellite Symposium: Servier
20.30	Welcome Reception

SATURDAY, 26/9/09

8.30 - 10.15	Reverse Remodelling
10.15 - 10.45	Break
10.45 - 12.00	Devices: Bridge to Transplantation
12.00 - 12.30	Lunch Break
12.30 - 14.00	Devices: Bridge to Recovery
14.00 - 14.15	Coffee Break
14.15 - 15.45	Heart Transplantation
16.00	Closing Ceremony and Meeting Adjournment

Session 1 Thursday, 24/9 14.30 – 16.15	**Publications and Innovations in Heart Failure**		
	Panel	*Igor D. Gregorič, MD* *Marianne Mallia, ELS* *Michael Samardzija, PhD*	*Houston, TX* *Houston, TX* *Houston, TX*
14.30 - 14.45	**Scientific publishing** *Marianne Mallia, ELS (Houston, TX)*		
14.45 - 15.00	**Organization of clinical trials** *Laura Damme (Boston, MS)*		
15.00 - 15.15	**Regulations and audits during research trials** *Michael Belotto, MPH (New York, NY)*		
15.15 - 15.30	**Commercializing your innovations: the challenges and pitfalls** *Michael Samardzija, PhD (Houston, TX)*		
15.30 - 15.45	**Reimbursement for LVAD therapy** *Tina Ivovic (Washington DC)*		
15.45 - 16.00	**Establishing collaborative heart failure programs to increase patient access to advanced care** *Timothy Myers, BS (Houston, TX)*		
16.00 - 16.15	**Discussion**		

Notes:

Thursday, 24th September

Session 2 Thursday, 24/9 16.30 – 17.45	**Heart Failure in Children**		
	Panel	*Charles D. Fraser Jr., MD* *Jeffrey A. Towbin, MD*	*Houston, TX* *Cincinnati, OH*
16.30 - 16.45	**Novel therapeutic strategies in children with heart failure** *Jeffrey A. Towbin, MD (Cincinnati, OH)*		
16.45 - 17.00	**Cardiac resynchronization therapy in pediatric heart failure patients** *Uros Mazič, MD (Ljubljana, SI)*		
17.00 - 17.15	**LVAD therapy in neonates and children with acute heart failure.** *David Mishaly, MD (Tel Hashomer, Israel)*		
17.15 - 17.30	**Surgical treatment for chronic heart failure in children** *Charles. D. Fraser Jr., MD (Houston, TX)*		
17.30 - 17.45	**Discussion**		
18.00 - 19.00	**Workshop: Scientific publishing** *Marianne Mallia, ELS (Houston, TX)*		

Notes:

Session 3
Friday,
25/9
8.30-10.00

End-Organ Perfusion in Advanced Heart Failure

Panel	*Ali El Banayosi, MD*	*Hershey, PA*
	Bas de Mol, MD	*Amsterdam, NL*
	Miran Šebeštjen, MD, PhD	*Ljubljana, SI*

8.30 - 8.45 **Myocardial and end-organ perfusion with continuous flow pumps**
Egemen Tuzun, MD (Houston, TX)

8.45 - 9.00 **Microcirculation in advanced heart failure**
Bas de Mol, MD (Amsterdam, NL)

9.00 - 9.15 **Skeletal muscle tissue oxygenation in advanced heart failure**
Matej Podbregar, MD, PhD (Ljubljana, SI)

9.15 - 9.30 **Anemia and cardiorenal syndrome in advanced heart failure**
Miran Šebeštjen, MD, PhD (Ljubljana, SI)

9.30 - 9.45 **Plasma exchange and immuno-adsorbtion**
Rajko Radovančević, MD (Houston, TX)

9.45 - 10.00 **Discussion**

Notes:

Friday, 25th September

Session 4
Friday,
25/9
10.15-11.45

Heart Failure and Coronary Heart Disease

Panel	*Fabio B. Jatene, MD*	*Belo Horizonte, Br*
	Mirta Koželj, MD, PhD	*Ljubljana, SI*
	Piotr Przybylowski, MD	*Krakow, P*

10.15 - 10.30 **High-risk ablation in heart failure with high intensive focused ultrasound**
Urban Lönn, MD (Zaventem, BE)

10.30 - 10.45 **Endoscopic epicardial ablation**
Borut Geršak, MD, PhD (Ljubljana, SI)

10.45 - 11.00 **Surgical revascularization in advanced ischemic heart failure**
Piotr Przybylowski, MD (Krakow, P)

11.00 - 11.15 **STICH trial**
Siniša Gradinac, MD (Belgrade, S)

11.15 - 11.30 **Parachute trial**
Pranav Loyalka, MD (Houston, TX)

11.30 - 11.45 **Discussion**

12.00 - 12.45 ***AstraZeneca Satellite Symposium:***
Advanced heart failure: CHARM-es beyond the heart
Moderator: Bojan Vrtovec, MD, PhD (Ljubljana, SI)

Comorbidities in heart failure: epidemiological, diagnostic, and therapeutic challenges
Zlatko Fras, MD, PhD (Ljubljana, SI)

Body wasting and cachexia in advanced heart failure: what are our odds?
Mitja Lainščak, MD, PhD (Golnik, SI)

Session 5
Friday,
25/9
13.30-15.00

Heart Failure and Valvular Disease

Panel	*Borut Geršak, MD, PhD*	*Ljubljana, SI*
	Pranav Loyalka, MD	*Houston, TX*
	Richard W. Smalling, MD	*Houston, TX*

13.30 - 13.45 **Percutaneous aortic valve: from design to clinical application**
David Paniagua, MD (Houston, TX)

13.45 - 14.00 **Percutaeous aortic valve repair- clinical trials**
Matjaž Bunc MD, PhD (Ljubljana, SI)

14.00 - 14.15 **Mitral valve disease in heart failure**
Danijel Planinc, MD (Zagreb, CRO)

14.15 - 14.30 **Percutaneous mitral valve repair**
Richard W. Smalling, MD (Houston, TX)

14.30 - 14.45 **Mitral valve surgery in heart failure**
Fabio B. Jatene, MD (Belo Horizonte, Br)

14.45 - 15.00 **Discussion**

Notes:

Friday, 25th September

Session 6
Friday,
25/9
15.15-16.45

Immune Modulation and Stem Cell Therapy

Panel	*Urban Lönn, MD*	*Zaventem, BE*
	Emerson Perin, MD, PhD	*Houston, TX*
	Guillermo Torre-Amione, MD, PhD	*Houston, TX*

15.15 - 15.30 **Anti-inflammatory and immune modulation therapy in heart failure**
Guillermo Torre-Amione, MD, PhD (Houston, TX)

15.30 - 15.45 **Cardiac tissue regeneration**
Marc W. Gerdisch, MD (Indianapolis, IN)

15.45 - 16.00 **Stem cell therapy in dilated cardiomyopathy**
Bojan Vrtovec, MD, PhD (Ljubljana, SI)

16.00 - 16.15 **Stem cell therapy in ischemic heart failure**
Emerson Perin, MD, PhD (Houston, TX)

16.15 - 16.30 **Benefits of aldosterone blockade in the treatment of post-MI patients with heart failure**
Simon G. Williams, MD, MRCP (Manchester, UK)

16.30 - 16.45 **Discussion**

Notes:

Session 7 Friday, 25/9 17.15-19.00	**Acute Heart Failure**		
	Panel	*Marko Noč, MD, PhD* *Biswajit Kar, MD* *George Wieselthaler, MD*	*Ljubljana, SI* *Houston, TX* *Vienna, A*
17.15-17.30	**Inotropic support in acute heart failure** *Vojka Gorjup, MD, PhD (Ljubljana, SI)*		
17.30-17.45	**Primary PCI in acute heart failure** *Marko Noč, MD, PhD (Ljubljana, SI)*		
17.45-18.00	**Impella support in acute heart failure** *Andrew B. Civitello, MD (Houston, TX)*		
18.00-18.15	**Percutaneous circulatory support in acute heart failure - Cardiology** *Biswajit Kar, MD (Houston, TX)*		
18.15-18.30	**TandemHeart use in acute heart failure – Surgery** *Igor D. Gregorič, MD (Houston, TX)*		
18.30-18.45	**Biventricular VAD support in acute heart failure** *Ivan Knezevič, MD (Ljubljana, SI)*		
18.45-19.00	**Discussion**		
19.00-19.30	***Servier Satellite Symposium:*** **Ischemia - the frontdoor to Heart Failure** *Marko Gričar, MD (Ljubljana, SI)*		

Notes:

Saturday, 26th September

Session 8
Saturday, 26/9
8.30-10.15

Reverse Remodelling

Panel	*Emma Birks, MD*	*Harefield, UK*
	Frank W. Smart, MD	*New Jersey, NY*
	J. David Vega, MD	*Atlanta, GA*

8.30 - 8.45 **Histological correlates of left ventricular remodelling**
Ana Maria Segura, MD (Houston, TX)

8.45 - 9.00 **Electrophysiological correlates of left ventricular remodelling**
Vito Starc MD, PhD (Ljubljana, SI)

9.00 - 9.15 **Surgical correction of HOCM with HF**
Konstantin Borisov, MD (Moscow, R)

9.15 - 9.30 **Developing LVA exclusion trial**
Srdjan D. Nikolic, PhD (S. Francisco, CA)

9.30 - 9.45 **ACORN trial**
William E. Cohn, MD (Houston, TX)

9.45 - 10.00 **The impact of selective VDR Activation on the cardio renal system**
Dennis L.. Andress, MD (Chicago, IL)

10.00 - 10.15 **Discussion**

Notes:

Session 9 Saturday, 26/9 10.45-12.00	**Devices: Bridge to Transplantation**		
	Panel	*Igor D. Gregorič, MD*	*Houston, TX*
		Matthias Loebe, MD	*Houston, TX*
		Francis D. Pagani, MD, PhD	*Ann Arbor, MI*
10.45 - 11.00	**Can you afford not to implant devices?** *Frank W. Smart, MD (Morristown, NJ)*		
11.00 - 11.15	**Complications with VADs: acceptable or prohibitive?** *Andrä Wasler, MD (Graz, A)*		
11.15 - 11.30	**Stroke prevention in mechanical circulatory support** *George M. Wieselthaler, MD (Vienna, A)*		
11.30 - 11.45	**LVAD use as a bridge to transplantation: are results equal to heart transplantation alone?** *James W. Long, MD, PhD (Oklahoma City, OK)*		
11.45 - 12.00	**Discussion**		

Notes:

Saturday, 26th September

Session 10
Saturday, 26/9
12.30-14.00

Devices: Bridge to Recovery

Panel	*Roland Hetzer, MD*	*Berlin, D*
	James W. Long, MD, PhD	*Oklahoma City, OK*
	Jaap Lahpor, MD	*Utrecht, NL*

12.30 - 12.45 **Axial vs pulsatile LVADs: Are criteria for recovery different?**
O.H. Frazier, MD (Houston, TX)

12.45 - 13.00 **HARPS trial**
Emma Birks, MD (Harefield, UK)

13.00 - 13.15 **LVAD and stem cells as recovery for ischemic cardiomyopathy**
Francis D. Pagani, MD, PhD (Ann Arbor, MI)

13.15 - 13.30 **3rd generation LVADs**
Ali El Banayosy, MD (Hershey, PA)

13.30 - 13.45 **Small implantable LVADs**
Matthias Loebe, MD (Houston, TX)

13.45 - 14.00 **Discussion**

Notes:

Session 11 Saturday, 26/9 14.15-15.45	**Heart Transplantation**		
	Panel	*O.H. Frazier, MD*	*Houston, TX*
		Sharon A. Hunt, MD	*Stanford, CA*
		Bojan Vrtovec, MD, PhD	*Ljubljana, SI*
14.15 - 14.45	**Candidate selection for heart transplantation** *Sharon A. Hunt, MD (Stanford, CA)*		
14.45 - 15.00	**Prolonged procurement time for heart transplantation** *Roland Hetzer, MD (Berlin, D)*		
15.00 - 15.15	**Eurotransplant: What's new?** *Jaap Lahpor, MD (Utrecht, NL)*		
15.15 - 15.30	**The change in heart allocation policy in the United States: Is it working as designed?** *J. David Vega, MD (Atlanta, GA)*		
15.30 - 15.45	**Discussion**		

Notes:

Use of sildenafil for prevention of right heart failure in heart transplant recipients with pulmonary hypertension

H. Bedanova[1], M. Orban[1], J. Ondrasek[1], P. Nemec[1]

[1] Center of Cardiovascular and Transplant Surgery, Brno, Czech republic

Objectives: The aim of our study is to demonstrate the impact of sildenafil on pulmonary circulation in HTx patients.
Background: Patients with increased pulmonary vascular resistance may suffer from acute right ventricular dysfunction early after heart transplantation (HTx). Recently, some centers increasingly present favorable effects of oral pulmonary vasodilators (sildenafil, bosentan) in treatment of pulmonary hypertension after HTx.

Methods: We present our experience in 8 patients (6 males and 2 females) in whom sildenafil was used to treat right ventricular dysfunction and pulmonary hypertension detected by Swan-Ganz right heart cathetrization and echocardiography early after HTx. In selected graft recipients with pulmonary hypertension and significant tricuspid regurgitation (TR), in addition to conventional inodilator support and alprostan, we administered 1-2mg per kilogram of sildenafil. Hemodynamic measurements were obtained before transplantation and during the ICU hospitalization. The effect of sildenafil (expected regression of dilatation and dysfunction of right ventricle and reduction of TR) was assessed by transthoracic echocardiogram evaluation. Sildenafil treatment was discontinued in three days after significant regresion of TR using stepwise dose reduction.

Results: Mean age was 54±8,8years. Preoperative hemodynamic data showed systolic pulmonary arterial pressure (SPAP) 46,6± 10,1mmHg, cardiac output (CO) 2,9±0,5l/min., transpulmonary gradient (TPG) 18,9±4,9mmHg and pulmonary vascular resistence (PVR) 6,9±1,9WU. Within 24-72 hours after the administration of sildenafil, acute right ventricular dysfunction resolved rapidly in all cases, TPG decreased significantly (18,9mmHg vs. 11mmHg) and so did PVR (6,9WU vs. 2,5 WU). Tricuspid regurgitation decreased from average grade 2,8 to grade 1,2 during the first week after transplantation and was trivial at the time of dismissal. No side-effects or impact on systemic blood pressure were observed during the course of treatment.

Conclusions: Sildenafil may be effectively used for treatment of acute right ventricular dysfunction in heart recipients with pulmonary hypertension.

Chronic heart failure – changes of adipose tissue

N. A. Bylova[1], K.B. Shavgulidze[1], K.A. Rogov[2], G.P. Arutyunov[1]

[1] Russian State Medical University, [2] City Clinical Hospital #4

Background: Patients with end stage of CHF usually develops cardiac cachexia, which associated with very poor prognosis. Adipose tissue in case of CHF and especially cardiac cachexia obtain new qualities, so that we propose, that adipose begins to be a key point of systemic inflammation in patient with CHF.
Objectives: I phase: to evaluate weight, BMI, levels of leptin in blood, % of adipose tissue in patients with CHF I-IV FC NYHA. II phase: to evaluate histology changes in adipose tissue in CHF I-IV FC (autopsy material).

Methods: I phase: 50 patients with CHF of ischemic genesis: 8 patients – I, 8 – II, 14 - III, 20 – IV FC. Height, weight, BMI, lean mass, % of adipose tissue (Durnin methodic) and leptin in plasma was evaluated.
II phase: 32 autopsies with CHF (ischemic genesis): 7 cases – I, 8 – II, 9 – III, 8 – IV FC. Thickness of adipose tissue (2 cm below umbilical), weight of sealing gland and histological analysis of adipose tissue were performed.

Results: I phase: BMI: I FC – 31,8±2,1, II FC – 31,6±2,7, III FC – 28,4±1,9, IV FC – 25,8±2,3 kg/m^2. % of adipose tissue: I FC – 32,3±2.3, II FC – 34,1±2,5, III FC – 26,9±2,1, IV FC – 18,9±1,9. Leptin levels: I FC – 12,3±3, II FC – 24,1±2.1, III FC – 36,9±2,5, IV FC – 58,9±1,1 ng/ml.
II Phase: thickness of adipose tissue (2 cm below umbilical): I FC - 2,9±0,7, II FC – 3,6±0,6 , III FC – 3,05±0,5, IV FC – 1,35±0,4cm. Weight of sealing gland: I FC - 378±89, II FC - 511±104, III FC - 319±76, IV FC – 84,5±44 g. Histological analysis: in autopsy cases of CHF III-IV FC lymphoid infiltration, collagen accumulation in perivascular spaces were found.

Conclusions: In patients with III-IV FC of CHF not only amount of adipose tissue changes, but morphology, with development of fibrosis, inflammation and possibly apoptosis of adiposities.

Structural alterations of large intestinal wall – possible cause of systemic inflammation in patients with chronic heart failure of different classes

G.P. Arutyunov[1], L.I. Kafarskaya[1], N.A. Savelov[2], N.A. Bylova[1], Yu.A. Pokrovsky[3], M.I. Korsunskaya[3], N.V. Kokosadze[2], A.B. Risnenko[2], Z.A. Chernaya[1]

[1] Russin State Medical University, [2] Oncology Center, [3] City Clinical Hospital #4

Background: One of the causes of systemic inflammation may be a high blood level of endotoxin, but what a reason that leads to it? A cause for the increased endotoxin in blood is probably not only growth of enterobacteria but also changes in the intestinal wall. We purpose, that structural alterations of large intestinal wall may play an important role in development of high blood level of endotoxin.

Aim: Evaluating changes in histological and histochemical structure of large intestinal wall in patients with different functional classes of CHF.

Materials and methods: Eighty patients with CHF of ischemic genesis were enrolled in study and randomized to 2 groups: group 1 (n=40) included patients with I-II FC CHF; group 2 (n=40) consisted of patients with III-IV FC CHF. Also healthy volunteers (n=40) were included into the study. All patients underwent a 6-min test; evaluation of EF (echoCG); measurement of CRP in blood (EIA method); endotoxin (LAL); faeces plating on growth media; colonoscopy with cecal biopsy with subsequent histological and histochemical (OLA, Muc5, staining for CD8+ cells; Ki67) evaluation of tissue samples.

Results: No significant changes were revealed in patients of group 1. Patient of group 2 had the picture of pronounced chronic inflammation. Lymphocytes were represented by CD8+ cells. Also the number of mucin 5-producing glandular cells was increased. Analyses of blood revealed significant ($p<0.05$) differences between two groups: CRP 1.89 ± 0.03 U/l and endotoxin 0.39 ± 0.01 EU/ml in group 1 and CRP 9.6 ± 0.07 U/l and endotoxin 1.3 ± 0.08 EU/ml in group 2. Level of enterobacteria was 10^{10} CFU/g in patients with III-IV FC CHF and 10^{7} CFU/g in patients with I-II FC CHF.

Conclusions: Patients with high FC CHF have chronic inflammation of large intestine, which is probably one of causes for the increased blood endotoxin and development of systemic inflammation.

Probiotics in therapy of patients with chronic heart failure

G.P.Arutyunov[1], L.I.Kafarskaya[1], N.A Bylova[1], T.K., Z.A. Chernaya[1], M.I. Korsunskaya[2]

[1] Russian State Medical University, [2] City Clinical Hospital #4

Background: Recent data have suggested excessive bacterial growth of gram-negative flora in the large intestine in patients with CHF. Studies have demonstrated the inefficiency of selective decontamination alone in this group of patients.

Objectives: Evaluating the efficacy of probiotics in the multimodality treatment of patients with III-IV NYHA FC CHF.

Methods: Study enrolled 30 patients with decompensate III-IV NYHA FC CHF of ischemic genesis. Patients were randomized to 2 groups. Group 1 (n=15) received a standard therapy (ACEI, diuretics, cardiac glycosides, b-blockers) and group 2 (n=15) received probiotics (Primadofilus) and standard therapy. All patients underwent blood tests for CRP, IL-6, TNF-a (EIA method) and endotoxin (LAL test) and faeces plating on selective growth media.

Results: In the beginning of study all patients showed increased proinflomattory plasma cytokines and endotoxin. Faeces analysis showed excessive growth of gram-negative organisms (10^{10} CFU/g). After 14 days of therapy the probiotic treatment group showed statistically significant decreases in the level of faecal gram-negative bacteria and plasma endotoxin, but a significant increase in proinflomattory cytokines was observed (CRP, 10.1±0.3; IL-6, 11.5+0,02 units/ml; TNF-a, 6.6+0,05 units/ml) $p<0.05$.

Conclusions: So we show normalization of intestinal flora by the probiotic therapy, but systemic inflammation processes become even more active. The explanation: probiotics as microorganisms interact with Toll-like receptors on enterocytes, which in turn activate the immune system and trigger systemic inflammation. Correction of endotoxemia and changes in large intestinal microflora require more thorough development.

Role of natriuretic peptides in assessment for aortic valve replacement in aortic stenosis

Z. Davicevic-Elez[1], D. Tavciovski[1], R. Matunovic[1], S. Vukotic[2], Z. Mijailovic[1], Z. Jovic[1]

[1] Clinic of Cardiology, Military Medical Academy, Belgrade, Serbia, [2] Clinic for Urgent Medicine, Military Medical Academy, Belgrade, Serbia

Background: Symptom onset is critical point in natural history of aortic stenosis and the most important indication for aortic valve replacement.

Methods and Results: B-type natriuretic peptide (BNP) and N-terminal BNP (Nt -proBNP) were determined in 67 patients with severe AS (mean age, 69±6.5 years; mean gradient, 51±6 mm Hg; valve area, 0.77±0.2 cm^2) who were followed up for 198±15 days. Natriuretic peptides increased with NYHA class and with decreasing ejection fraction (EF). Even asymptomatic patients (n=35) frequently had elevated neurohormones. Asymptomatic patients during follow -up period had higher BNP and Nt-proBNP levels compared with those at study beginning (median for BNP, 90 pg/ml [interquartile range, 49-110 pg/ml] versus 79 pg/ml [range, 44 to 90 pg/ml], *p*<0,0001; median for Nt-proBNP, 109 pmol/L [interquartile range, 82 to 133 pmol/L] versus 99 pmol/L [interquartile range, 77 to112 pmol/L], *p*<0,0001). Symptomatic patients (n=32) decreased natriuretic peptides level after aortic valve replacement (BNP, 118 pg/ml [29 to 266 pg/ml] versus 71 pg/ml [32 to 119 pg/ml], *p*<0,0001; Nt-proBNP, 258 pmol/L [67 to 520 pmol/L] versus 31 pmol/L [18 to 53 pmol/L], *p*<0,0001). NT-proBNP level higher than 122 pmol/l was cutoff value for symptoms detection in patients with severe aortic stenosis (spec.71,78%, sens. 91,43%, *p*<0,0001, AUC 0,756).

Conclusions: Natriuretic peptides provide important information in severe aortic stenosis, beyond clinical and echocardiographic evaluation. Measurement of neurohormones may gain particular importance in optimal timing for aortic valve replacement in asymptomatic severe AS.

Chronic heart failure and level of kidney dysfunction in patients with arterial hypertension: Results of epidemiological research in Moscow

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Background: Results of several clinical trials show that such an early marker of kidney disfunction as microalbuminuria (MAU) is assosiated with increased risk of cardiovascular complications and maximal risk of chronic heart failure (CHF). Today there is no data on prevalence of glomerular hyperfiltration (GH) in patients with arterial hypertension (AH), its correlation with CHF. The **objective** of our study was to determine the prevalence of GH in patients with AH and its correlation with basic demographic, clinical (including CHF) data.

Methods: Retrospective analyses of 1160 case histories of outpatients and 1070 of in-patients Glomerular Filtration Rate (GFR) was calculated with Mayo formula. Depending on the GFR value patients were divided into 5 groups – GH and another 4 groups (according to National Kidney Foundation K/DOQI classification) - 90 ml/min/1,73 m2 and above, 60-90, 30-60, 29 ml/min/1,73 m2 and less.

Results: Mean GFR was 73,66± 23,22 ml/min/1,73 m2 at outpatients and 65,79 ±22,45 – at in-patients (p<0,008). GH was found in 232 outpatients (20,0%) and 121 in-patients (11.3%). In the group of GH there were more men (82,35%), patients were younger (mean age 51,93±9,85), continuance of AH was less (4,64±0,97 years). Prevalence of CHF was significantly lower in the group of GH. In 2 years GFR at such patients decreased by 12,12%, BP and MAU got higher (systolic BP – by 17,85 mm Hh, diastolic - by 7,4 mm Hg, MAU - 10 times as much), prevalence of CHF increased by 35,78%.

Conclusion: GH is typical for young outpatients, mostly male, with high normal blood pressure and AH of 1-2 degrees. In the group of GH just 1 FC of CHF was observed, so GH strategically changes patients prognosis and it is very important to start treatment at this stage to prevent the progression of kidney dysfunction and development of CHF.

Assessment of functional capacity and stratification patients with isolated left ventricular diastolic dysfunction

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Background: Cardiopulmonary Exercise Testing (CPET) is a gold standard of assessment of functional capacity, prognosis and for evaluation of putative mechanism underlying exercise intolerance in patients with HF.
The aim of our study was to assess value of CPET in stratificating patients with similar degree of LV diastolic dysfunction (LVDD), but with different origin.

Methods: By 2D echo and M mod LVEDD, LVESD, EDV, ESV, IVS, PW, LA, EF, FS were obtained, by PW Doppler: Vmax E, Vmax A, dec t E, VpE by Color M and E/e by TDI. By sub maximal testing using Bruce protocol and breath by breath method we obtained: *pVO2* - peak oxygen uptake, *VO2/HR* -oxygen pulse, percentage of pVO2 at *VAT*- ventilatory anaerobic threshold.
Study patients consisted from 60 patients aged 50.76 ± 8.9 with history of hypertension > 2years and 31 patients aged 49.84 ± 6.9 with history of previous myocardial infarction > 1year. All patients were classified as mild diastolic dysfunction i.e. impaired relaxation (E/A <1, DTE> 220ms) and preserved systolic function (EF>50%).

Results: Demographic, Doppler Echocardiographic and Cardiopulmonary characteristics at rest were similar among two groups. However, significantly higher values of reached *pVO2* (1715 vs 2083 ml, p<0.001),*VO2/HR* (12 vs 14.6, p<0.001), and *% pVO2 at VAT* (55 vs 64, p = 0.007) were found in patients after AMI compared with hypertensive group.
The were significant correlations in both groups between *pVO2* and LV dimensions (LVDD, LVSD) (r = 0.433, r= 0.513, respectively, p<0.001) , between *pVO2* and V max A and E/em (r = - 0.413, r =0.398, respectively, p<0.001)

Conclusions: In hypertensive and ischemic patients with similar echo-Doppler characteristics of LVDD at rest, the distinction in functional capacity appears during CPET. Functional capacity in both group are closely related to proper LV expansion and degree of impaired LV relaxation

Tissue Doppler Global Function Index in patients with Hypertensive Heart Diseasse, one year follow up

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Background: A global function index (GFI) derived from tissue Doppler imaging (TDI) has been proposed to improve the diagnosis of hypertrophic cardiomyopathy (HCM). We aimed to evaluate the usefulness of this index in hypertensive patients.
Study patients: 129 hypertensive patients, with preserved ejection fraction (EF), were divided in four groups according the remodeling proces NLV: normal left ventricular geometry (n=30), CR: concentrinc remodelling (n=31), CH: concentric hypertrophy (n=35), EH: excentric hypertrophy (n=33).

Methods: Relative wall thickness, left ventricular mass index (LVMI), left atrial volume index (LAVI) and EF were estimated by echocardiography. We measured coresponding velocities from tissue Doppler at the level of the septal mitral annulus (e,a,s), including GFI [(E/e)/s]. The same measurements were repeated after one year.

Results: There was significant correlation between the values of GFI and LVMI ($r = 0.302$, $p = 0.001$) and LAVI ($r = 0.552$, $p = 0.0004$) and significante difference of GFI between groups ($F = 17.879$, $p = 0.0001$), level of GFI progressively inceased from NLV, through the CR, CH and EH. GFI significantly increased during three years with significant time difference ($F=78.987$, $p=0.006$) and with significant difference between groups ($F=4.819$, $p=0.003$), more exspressed in EH group.

Conclusion: In hypertensive patients GFI was the clear reflection of remodeling process of left atrium and left ventricle. GFI, measured by TDI, might be simple and helpful indicator of the level of function and remodeling at the same time in hypertensive heart disease.

Complex therapy of CHF III-IV FC: aliskiren and changes in kidney function

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Background: Chronic heart failure (CHF) usually associated with decrease of blood pressure that leads to decreasing of perfusion pressure in kidney glomerule. In case of correction of renal hemodynamics we need to prescribe medication with positive inotropic effect, which associated with poor prognosis in patients with CHF. High doses of diuretics also leads to decrease of glomerule filtration.

Objectives: To evaluate glomerular filtration using aliskiren in complex therapy of CHF.

Methods: 63 patients were hospitalized due to decompensation of CHF of ischemic genesis. All of patients received standard therapy: diuretics (i/v), dopmin (i/v), cardiac glycosides (i/v). Prescription of ACE and betaadrenoblockers were limit due to low blood pressure. All patients were randomized into two groups: first group (n=32) received aliskiren (150 mg a day) in addition to standard therapy, second group (n=31) – placebo. At day 1, 8, 14, 28 pulse, blood pressure, urine volume, weight, glomerular filtration rate (GFR) (by MDRD) were evaluated. At day 1 and 28 ejection fraction, activity of plasma renin were evaluated. Study was approved by local ethical committee.

Results: at baseline urine volume was 1268±82 ml/24 h, in I group at day 2 urine volume increased to 2628±107 ml/24 h, in II group at day 4 - to 2185±99 ml/24 h. At baseline GFR was 55,5±4,5 ml/min. In I group statistically significant increased of GFR at day 3 - 119,8±12,4 ml/min/SA ($p<0.05$), in II group at day 8 GFR was 89,7±9,6 ml/min. Activity of plasma renin: at baseline 8,64±1,5 ng/ml/h, at day 28 in I group 6,98±1,43 ng/ml/h, in II group 8,76±1,9 ng/ml/h. Also use of aliskiren associated with decrease of duration of hospitalization.

Conclusions: in patients with III-IV FC CHF use of aliskiren in complex therapy leads to faster compensation and low duration of hospitalization.

Levosimendan improves renal function in patients with advanced chronic heart failure awaiting cardiac transplantation

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Background: Long-term impact of levosimendan on renal function remains undefined. Prospectively, we evaluated effects of levosimendan on renal function in patients with advanced chronic heart failure awaiting cardiac transplantation.

Methods: Of 40 patients, 20 were randomized to receive levosimendan (10-minute bolus 12 microg/kg, followed by 0.1 microg/kg/min for 24 hours; LS Group), and 20 received no levosimendan (Controls). The groups did not differ in age, heart failure etiology, left ventricular ejection fraction, and plasma brain natriuretic peptide. Patients were followed for 3 months.

Results: At baseline, the groups did not differ in serum creatinine (1.92 +/- 0.13 mg/dL in LS Group versus 1.91 +/- 0.12 mg/dL in Controls, P = .81) and creatinine clearance (43.7 +/- 2.9 mL/min versus 43.9 +/- 2.8 mL/min, P = .84). At 3 months, we found a decrease in serum creatinine and an increase in creatinine clearance in LS Group, but not in Controls, leading to a significant intergroup difference in serum creatinine (1.60 +/- 0.26 mg/dL in LS Group versus 1.90 +/- 0.14 mg/dL in Controls, P = .005) and creatinine clearance (53.6 +/- 8.6 mL/min versus 44.0 +/- 3.3 mL/min, P = .005). An improvement in creatinine > or = 0.5 mg/dL occurred in 50% patients from LS Group compared with 10% of Controls (P = .005).

Conclusion: Levosimendan improves long-term renal function in advanced chronic heart failure patients awaiting cardiac transplantation.

Patient education before and after heart transplantation (role of a transplant nurse)

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Background: Heart transplantation represents an established treatment modality for advanced heart failure patients. The successful outcome of heart transplantation largely depends of coordinated efforts of the entire team that is involved in patient managenem before and after heart transplantation.

Role of a transplant nurse in patient education: Since only a well educated patient can activelly and efficiently cooperate with a medical team in the treatment process, patient education is of paramount importance in every stage of disease. Patient education is focused on slowing of the progression of the disease, understanding of the underlying heart condition and on understanding of the proposed tretment srategies. To perform patient education efficiently transplant nurse has to be familiar with the basics of the underlying heart disease and with idiosyncrasies of living with a failing heart. She also has to be familiar with protocols for preparing a patient for hear transplant and with a structure and operation of the transplant network in Slovenia.
In the time prior to heart transplantation transplant nurse supports a patien with a failing heart and motivates him/her towards minimising risk factors that could worsen the underlying heart disease.
During preparations for heart transplantation transplant nurse coordinates patient work-up per transplant protocole.
At the time of heart transplant transplant nurse admitts a pacient to a clinic, performs pre-transplant laboratory work-up per protocole and prepares a patient for the upcomming surgical procedure. Prior to surgery transplant nurse is also responsible for administration of immunoinduction therapy.
After heart transplantation transplant nurse is responsible for preparing a patient for a life with a transplanted heart. The main focus is on complience
with medical management, especially immunosuppresive therapy.

Conclusion: In recent years the number of heart transplantatons has significantly increased in Slovenia. Thus transplant nurse is becomming an integral part of the heart transplant team. Through patient education transplant nurse significantly improves patients' understanding of the underlying heart disease and the proposed tretment srategies. A well educated patient can then cooperate with a medical team in the treatment process more efficiently which in turn may improve his/her long term treatment outcome.

Prognostic value of diastolic disfunction in patients after coronary artery bypass grafting

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Background: It is well known that left ventricle (LV) systolic function has important prognostic value in patients with coronary artery disease, who undergo coronary artery bypass grafting (CABG). However, and diastolic dysfunction, independently from systolic function of LV, may have prognostic value, that is a subject of newest research.

Aim: To determine relation between different forms disorders of diastolic filling of LV and appearance of postoperative complications in patients with heart failure who undergo CABG.

Methods: Disorders of diastolic filling of LV are determined by measuring of transmitral diastolic flow with Doppler echocardiography, in patients with symptoms and signs of heart failure. In preoperative period, patients treated with all necessary medications.

Results: In preoperative period, patients are treated with all necessary medications. We have involved 60 patients in our study: 41 men and 19 women, average age 63.04±5 years, with coronary artery disease and symptoms and signs of heart failure, according Framingham's criteria, and who indicated for CABG.

During two weeks of observation in postoperative period, in the group of patients with restrictive diastolic filling, we registered the highest level of mortality (4/26, 15%, $p<0.005$) and cardiovascular complications (11/26, 38.5% $p<0.005$). Only 7 patients had significantly reduced ejection fraction (EF) of LV. (EF<40%). Patients with restrictive filling, as a worst entity of diastolic heart failure, had a highest morbidity 38.5% and mortality 15,23%. In view of that in 3 deaths over 6, before operation, founded in a patients with EF>40%, it can be conclude that patients with coronary artery disease and signs of diastolic heart failure and relatively maintain (systolic function) EF, has also bad prognosis.

Conclusion: The study results sugests that restrictive type of diastolic filling patern of LV was sign of bad prognosis in patients with coronary artery disease and signs of heart failure in patients treated with CABG.

Role of natriuretic peptides in assessment for aortic valve replacement in aortic stenosis

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Methods and Results: B-type natriuretic peptide (BNP) and N-terminal BNP (Nt-proBNP) were determined in 67 patients with severe AS (mean age, 69±6.5 years; mean gradient, 51±6 mm Hg; valve area, 0.77±0.2 cm^2) who were followed up for 198±15 days. Natriuretic peptides increased with NYHA class and with decreasing ejection fraction (EF). Even asymptomatic patients (n=35) frequently had elevated neurohormones. Asymptomatic patients during follow-up period had higher BNP and Nt-proBNP levels compared with those at study beginning (median for BNP, 90 pg/ml [interquartile range, 49-110 pg/ml] versus 79 pg/ml [range, 44 to 90 pg/ml], p<0,0001; median for Nt-proBNP, 109 pmol/L [interquartile range, 82 to 133 pmol/L] versus 99 pmol/L [interquartile range, 77 to112 pmol/L], p<0,0001). Symptomatic patients (n=32) decreased natriuretic peptides level after aortic valve replacement (BNP, 118 pg/ml [29 to 266 pg/ml] versus 71 pg/ml [32 to 119 pg/ml], p<0,0001; Nt-proBNP, 258 pmol/L [67 to 520 pmol/L] versus 31 pmol/L [18 to 53 pmol/L], p<0,0001). NT-proBNP level higher than 122 pmol/l was cutoff value for symptoms detection in patients with severe aortic stenosis (spec.71,78%, sens. 91,43%, p<0,0001, AUC 0,756).

Conclusions: Natriuretic peptides provide important information in severe aortic stenosis, beyond clinical and echocardiographic evaluation. Measurement of neurohormones may gain particular importance in optimal timing for aortic valve replacement in asymptomatic severe AS.

Benefits of Aldosterone blockade in the treatment of post MI patients with heart failure

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Heart failure & left ventricular systolic impairment are relatively common in the setting of a myocardial infarction (MI). This syndrome is associated with a poor prognosis. Updated & revised guidelines for secondary prevention treatment following an MI have been published in the past 2 years. Aldosterone blockade with Eplerenone (Inspra) is an important additional therapy for these patients to prevent further mortality & morbidity. Clinical trial data from the EPHESUS trial (and subsequent analyses) and the concept of class effect will be discussed.

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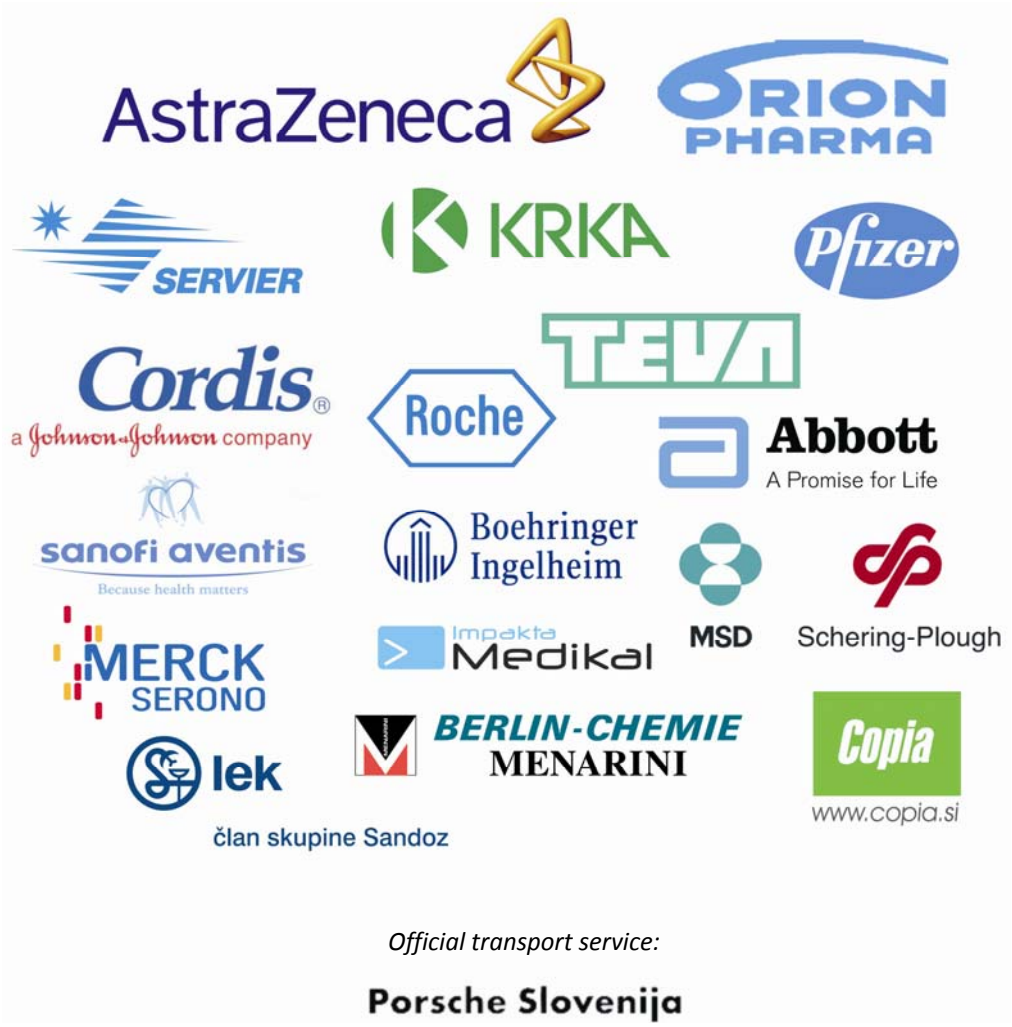

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