08:30 - 10:30 S8 - Clinical and Prognostic Parameters in CHF and Cardiomyopathy Hall E

Chairs: **A. Keren**
A. Shotan

08:30 **The Clinical and Radiological Signs during the Evolution of Acute Heart Failure Correlate with Lung Impedance**

M. Shochat [1], M. Kazatsker [1], V. Gurovich [1], E. Neyman [1], A. Asif [1], A. Frimerman [1], P. Rabinovich [2], D. Blondheim [1], A. Shotan [1], S. Meisel [1]
[1] Haifa, [2] Tel Aviv

08:45 **Blood Transfusion for Acute Decompensated Heart Failure – Friend or Foe? – Lessons from the First Israeli Heart Failure Survey**

A. Zhuchenko [1], M. Garty [1], E. Cohen [1], Z. Iakobishvili [2], A. Porter [2], M. Mittelman [3], A. Battler [2], S. Behar [4], V. Boyko [4], A. Shotan [5], S. Gottlieb [6], D. Hasdai [2]
[2] Petah Tikva, [3] Tel Aviv, [4] Tel Hashomer, [5] Hadera, [6] Jerusalem

09:00 **Echocardiogram Assessment of Pulmonary Hypertension; How Accurate Is It?**

O. Amir, Y. Adir, N. Salman, R. Wolff, D. Merhavi, H. Paz, N. Yaniv, R. Ammar, D. Weiler, B.S. Lewis
Haifa

09:15 **Renal Failure as a Prognostic Marker for Mortality in Heart Failure Patients – Are We Barking Up at the Wrong Tree?**

R. Wolff, O. Amir, H. Paz, D. Merhavi, N. Salman, A. Antebi, N. Yaniv, R. Ammar, B.S. Lewis
Haifa

09:30 **Peripartum Cardiomyopathy: Echocardiographic Predictors of Functional Recovery**

R. Kuperstein, M. Arad, M. Simchen, Y. Frenkel, H. Shani, M. Feinberg, D. Feimark
Tel-Aviv

09:45 **Brachial Artery Endothelial Function Predicts Mortality Risk in Patients with Advanced Ischemic Chronic Heart Failure**

M. Shechter, M. Arad, S. Matetzky, M. Feinberg, D. Freimark
Tel Hashomer, Tel Aviv

08:30 - 10:30 S8 - Clinical and Prognostic Parameters in CHF and Cardiomyopathy (Cont.) Hall E

10:00 **Clinical Predictors of Disease-carrier State in Familial DCM**

M. Arad [1], M. Feinberg [1], Y. Potashnik [1], G. Yoskovitz [1], M. Gramlich [2], E. Pras [1], L. Thierfelder [2], D. Freimark [1]
[1] Ramat Gan, [2] Berlin

10:15 **Clinical and Hemodynamic Outcome of Inferobasal Wall Injury Complicating Alcohol Septal Ablation in Hypertrophic Cardiomyopathy**

A. Keren, M. Potekhin, I. Gotsman, D. Zwas, M. Mosseri, R. Beeri, D. Leibowitz, G. Levin, H. Danenberg, A. Pollak, D. Admon, C. Lotan
Jerusalem

The Clinical and Radiological Signs during the Evolution of Acute Heart Failure Correlate with Lung Impedance

Michael Shochat [1], Mark Kazatsker [1], Vladimir Gurovich [1], Elena Neyman [1], Aya Asif [1], Aaron Frimerman [1], Paul Rabinovich [2], David Blondheim [1], Avraham Shotan [1], Simcha Meisel [1]

[1] Heart Institute, Hillel Yaffe Medical Center, Rappaport Faculty of Medicine, Technion, Haifa, [2] Department of Internal Medicine C, Sourasky Medical Center, The Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel

Background - there is no non-invasive method to predict acute heart failure (AHF) before the appearance of clinical signs. The implantable impedance device is the only method that predicts AHF with sensitivity of 48-76%. Our aim was to validate the ability of the non-invasively measured lung impedance (LI) to predict AHF by comparison with chest x-ray findings and with the physical examination in patients hospitalized for acute myocardial infarction (AMI).

Methods and Results- We used a non-invasive method for LI measurement. A radiological score (RS) of 0 to 10 was devised for lung fluid quantification. 37 healthy volunteers and 523 AMI patients with normal physical examination and chest x-ray on admission were monitored for 94±35 hours. LI decrease in healthy volunteers was< 6% (p=NS). LI decrease in 395 AMI patients that did not develop AHF was <12% from baseline (p=0.3) and RS increased from 0.8±0.5 to1.3±0.5 (p =0.06). In the other 128 AMI patients, LI decreased by >12%, which was predictive of AHF. At the interstitial stage of AHF, LI decreased by 13.2± 1.5% (p<0.0001) with an RS of 2.9±0.5 (p<0.05). When rales appeared, LI decreased by 21± 2.7% (p<0.0001) and RS further increased to 4.5±0.8 (p<0.001). At full-blown AHF, LI decreased by 36± 5.5% and RS increased to 9.5±1.1. LI correlated with RS (r= -0.92, p<0.001). The time elapsed from the point at which LI decreased >12% to the appearance of rales and radiological signs of AHF was 242±105 min.

Conclusions- Non-invasive LI monitoring has a 95% positive predictive value for prediction of AHF at the interstitial edema stage. Prediction was feasible early enough to allow initiation of effective therapy as a preventive measure.

Blood Transfusion for Acute Decompensated Heart Failure – Friend or Foe? – Lessons from the First Israeli Heart Failure Survey

Alexander Zhuchenko [1], Moshe Garty [1], Eytan Cohen [1], Zaza Iakobishvili [2], Avital Porter [2], Moshe Mittelman [3], Alexander Battler [2], Solomon Behar [4], Valentina Boyko [4], Avraham Shotan [5], Shmuel Gottlieb [6], David Hasdai [2]

[1] Rekanati, [2] Cardiology Department, Rabin Medical Center, Petah Tikva, [3] Internal A, Sourasky Medical Center, Tel Aviv, [4] Neufeld Cardiac Research Center, Sheba Medical Center, Tel Hashomer, [5] Cardiology Department, Hillel Yafe Medical Center, Hadera, [6] Cardiology Department, Bikur Holim Hospital, Jerusalem, Israel

Background: The impact of blood transfusion (BT) among pts with acute decompensated heart failure (ADHF) remains unknown. **Aim:** To examine the impact of BT among ADHF pts enrolled in the First Israeli Heart Failure Survey in 2003. **Methods:** Propensity score analysis of ADHF pts with and without BT. **Results:** Of the 4102 pts, 2335 had ADHF, of whom 166(7.1%) received BT. They were older (75.6 vs 73.6, p=0.04), and more likely to be females (54.8% vs 43.9%, p=0.007), diabetic (59.0% vs 51.1%, p=0.04), and with renal dysfunction (59.0% vs 40.2%, p<0.001). BT pts were more likely to receive inotropes (16.9% vs 8.0%, p<0.001), but had similar rates of concurrent ACS (41.0% vs 39.4%, p=0.68). Nadir hemoglobin levels were <10gr% in 92.7% BT pts vs 7.9% in non-BT pts; 15 BT pts had bleeding complication, 10 major. Major predictors for BT were ACS (OR=1.85, 95% CI 1.15-2.96), inotropes (OR=2.36, 95% CI 1.2-4.6), and nadir hemoglobin (OR=0.18, 95% CI 0.14-0.22). In-hospital, 30d, and 1y unadjusted mortality rates were higher for BT pts (10.8% vs 5.2%, p=0.002, 11.0% vs 8.5%, p=0.27, and 39.6% vs 28.5%, p=0.003, respectively). 103 matched pairs were identified with c-statistic of 0.97 and in-hospital, 30d, and 1y mortality rates tended to be lower for matched BT pts (8.7% vs 14.6%, p=0.20, 9.7% vs 18.4%, p=0.08, and 38.8% vs 42.7%, p=0.59, respectively). **Conclusions:** ADHF pts receiving BT had worse clinical features, and accordingly worse outcomes. However, BT *per se* in this setting seems to be safe and perhaps even beneficial.

Echocardiogram Assessment of Pulmonary Hypertension; How Accurate Is It?

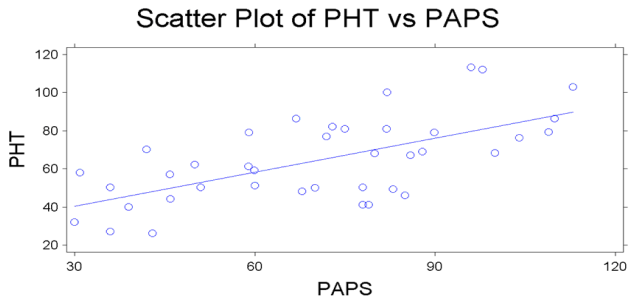
Offer Amir [1,3], Yochai Adir [2,3], Nabia Salman [1,3], Rafael Wolff [1,3], Dina Merhavi [1,3], Hagar Paz [3], Nissan Yaniv [1,3], Ronny Ammar [3], Daniel Weiler [2], Basil S Lewis [1]

[1] Cardiology, Lady Davis Carmel Medical Center, [2] Pulmonary Institute, Lady Davis Carmel Medical Center, [3] Pulmonary Hypertension Clinic, Cardiology, Lin Medical Center, Haifa, Israel

Background: Echocardiogram is the most common tool for assessment of pulmonary hypertension (PHT). The availability of this non-invasive test as well as its increasing reliability raised a valid question regarding the necessity of the "gold standard" invasive method for PHT measurements- the right heart catheterization. Accordingly, we assessed the PHT measurements correlation between the echocardiogram {"PHT"} and the right heart catheterization {"PAPS"}.

Patients and Methods: The echocardiogram and right heart catheterization data of 42 consecutive patients {mean aged 62 ± 11 year old, 21(52%) females}, were analyzed. Seventeen (40%) patients had systolic heart failure. An echocardiogram systolic pulmonary pressure (based on tricuspid regurgitation measurements and the Bernoulli equation) was compared to the direct measurement via the right heart catheterization.

Results: In a statistical analysis we found the correlation of the systolic pressure between the two tests to be modest ($r=0.63$, $p<0.0001$), figure-1.



Demographic parameters of age and sex had no significant impact on the echocardiogram prediction of measurements; however, both left ventricular ejection fraction and the magnitude of the tricuspid regurgitation decreased the echocardiogram reliability of the PHT measurements.

Conclusion: There is a modest correlation between the echocardiogram pulmonary measurements and the gold standard hemodynamic measurements. Accordingly, in patients where the accuracy of the measurements is important (such as in heart transplant evaluation, PHT treatment options etc), the echocardiogram may not serve as the final test and direct hemodynamic measurements are still irreplaceable.

Renal Failure as a Prognostic Marker for Mortality in Heart Failure Patients – Are We Barking Up at the Wrong Tree?

Rafael Wolff [1,2], Offer Amir [1,2], Hagar Paz [1], Dina Merhavi [1,2], Nabia Salman [1,2], Alon Antebi [1,2], Nissan Yaniv [1,2], Ronny Ammar [1], Basil.S Lewis [2]

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Background: Deteriorating renal function is an important risk factor for morbidity and mortality in chronic heart failure (CHF). There are several formulas for estimating renal function and glomerular filtration rate.
In this study we compared the predictive value of mortality of four renal function tests; serum urea, serum creatinine, Cockcroft-Gault (CCT) and estimated Modification of Diet in Renal Disease (eMDRD).
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Methods and Results: Our analysis included **366** CHF patients (267 Men, 99 women; age 66±13.5 years).Mean BMI was 27±5.4 kg/m², LVF was 30±0.14 and mean NYHA class was 2.8. Baseline creatinine, urea, CCT and eMDRD were calculated.
During mean follow-up time of 4 years, 85 patients died (23.2%). We compared the different renal function tests models between these two groups of patients, the patients who survived and the patients who died (Table-1).All four parameters were found to be significantly associated with mortality. However, in a stepwise logistic regression analysis adjusted for age, sex, LVF and ischemic etiology, eMDRD was the single and the most important parameter in predicting mortality.

Table-1

Renal function test model	Alive	Dead	P value
Creatinine	1.28±0.3	1.53±0.6	0.003
Urea	64	79.9	0.003
GFRcg	69	54.4	0.004
eMDRD	65.5	50.9	<0.0001

Conclusions: Our data suggests that among the different renal function tests models, the eMDRD is the strongest parameter in prediction of mortality in heart failure_patients. Accordingly, we suggest that eMDRD should be assessed routinely in heart failure patients.

Peripartum Cardiomyopathy: Echocardiographic Predictors of Functional Recovery

Rafael Kuperstein [1], Michael Arad [1], Michal Simchen [2], Yair Frenkel [2], Hagit Shani [2], Micha Feinberg [1], Dov Feimark [1]

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Background: Peripartum cardiomyopathy (PPCM) is defined as the onset of cardiac failure without identifiable cause 1 month pre, to 5 months post-partum. Subsequent pregnancy is hazardous in these patients but may be considered after recovery of left ventricular (LV) function.

Methods: We studied women with PPCM treated in the Leviev Heart Center between 1998-2008 to identify the predictors of LV function recovery (LVEF≥50% on follow-up). All women laboring at our hospital during 2007(n=10,370) were used as controls.

Results: 29 women were diagnosed with PPCM (11% of our dilated cardiomyopathy database). Mean age, primiparity and hypertensive complications were higher compared with controls (32±7 vs 30±5 years; 59% vs. 37.5% and 45% vs. 3.4% respectively, p<0.001 for all). . Follow-up time ranged 1-117 months (26±32mo).

Ten of 23 (43%) with adequate echocardiographic follow-up improved LVEF.

	Improvers (n=10)	**Non-improvers (n=13)**	**p**
Age(years)	36±6	28±6	0.005
Baseline LVEF%	32±9	26±8	0.08
LVEDD(mm)	57±7	52±5	0.08
LVESD(mm)	39±6	47±6	0.005
IVS(mm)	10.4±1.8	8.2±1.2	0.002
PW(mm)	9.1±1.0	8.1±1.5	0.1
LA(mm)	39±7.5	39±7	0.9
Follow up LVEF%	56±4	36±8	<0.0001

LVEF, LV ejection fraction; LVEDD, LV end-diastolic dimension; LVESD, LV end-systolic dimension; IVS, inter-ventricular septal width; PW, posterior wall width; LA, left atrial diameter.

There were no differences in height, weight, BSA or BMI.

Conclusions: Women who develop PPCM tend to be older primigravidas, and are often hypertensive. Nearly half recover LV function. Younger age, larger ventricle and thinner walls are associated with a worse prognosis and may be a clue for presence of a preexisting cardiomyopathy.

Brachial Artery Endothelial Function Predicts Mortality Risk in Patients with Advanced Ischemic Chronic Heart Failure

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Leviev Heart Center, Sheba Medical Center, Tel Hashomer, Israel, The Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

Background While endothelial function is impaired in chronic heart failure (CHF) patients, its association with mortality risk has not been reported.

Methods We prospectively assessed brachial flow-mediated dilation (FMD) in 82 consecutive advanced (New York Heart Association [NYHA] class IV) ischemic CHF patients with left ventricular ejection fraction (LVEF) 22±3%. Following overnight fasting and discontinuation of all medications for ≥ 12 hours, percent improvement in FMD (%FMD) and nitroglycerin-mediated vasodilation (%NTG) were assessed using high resolution (15 MHz) linear array ultrasound. All patients were followed for 14±2 months for pre-specified combined adverse cardiovascular events, including death, hospitalization for CHF exacerbation or myocardial infarction.

Results Subjects were divided into 2 groups: ≤ (n=41) and > (n=41) the median %FMD of 4.6%. Both groups were comparable regarding risk factors, LVEF, lipids, glucose, electrolytes, hemoglobin, creatinine clearance, and concomitant medications. During follow-up 22 (53.6%) patients with FMD ≤ had composite adverse cardiovascular events compared with only 8 (19.5%) with FMD > the median ($p<0.01$). Furthermore, 5 deaths (12.1%) occurred in patients with FMD ≤, compared with no deaths in FMD > the median ($p<0.03$) (Figure). Cox regression analyses revealed that FMD was an independent predictor for these events.

Conclusions FMD is associated with increased mortality risk in NYHA class IV ischemic CHF patients.

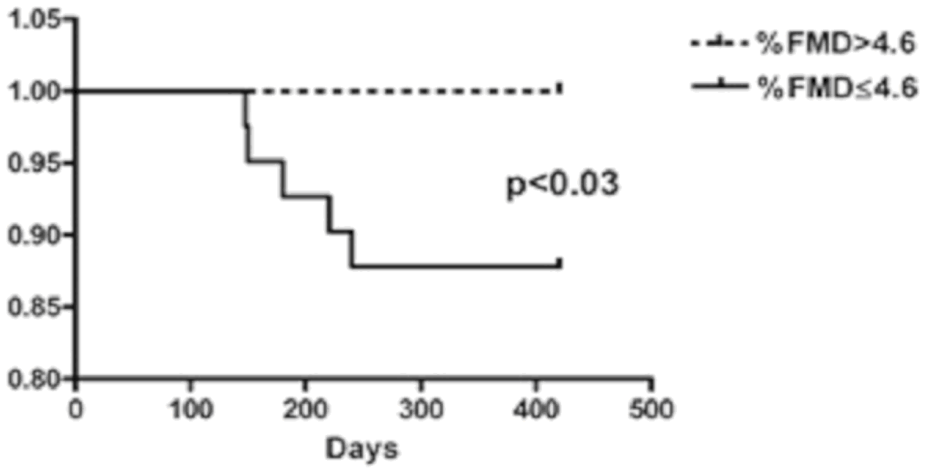

Figure: Kaplan-Meier plot showing proportion of survivors over time in advanced NYHA class IV CHF patients with FMD of the brachial artery > (dashed line) and ≤ (solid line) median value of 4.6%. Patients with FMD ≤ the median had higher mortality compared to those above the median (5 vs 0 events; $p<0.03$, by long-rank test).

Clinical Predictors of Disease-carrier State in Familial DCM

Michael Arad [1], Micha Feinberg [1], Yael Potashnik [1], Guy Yoskovitz [1,2], Michael Gramlich [3], Elon Pras [2], Ludvik Thierfelder [3], Dov Freimark [1]

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Idiopathic dilated cardiomyopathy (DCM) is a major cause of heart failure and heart transplantation in young adults. In about 30% of cases DCM is a familial disease, usually with autosomal dominant inheritance. In most families the disease-causing mutation is unknown and relatives undergo periodic screening to allow early diagnosis and therapy.

We previously identified a TTN Ins59014A mutation in a large Arab family with adult-onset DCM. Genotyping and clinical screening were then extended to include younger first degree relatives in order to identify the earliest predictors of the disease in healthy carriers.

The 11 affected individuals in the original cohort (M/F 6/5, 48±12 years), when compared with their mutation-negative first degree relatives (M/F 11/13, 43±7 years), showed differences in LVSF (21±7 vs. 39±7%, $p<0.0001$), LVEDD (58±9 vs 49±4mm, $p<0.001$), LVESVI (38±10 vs. 21±7, $p<0.0001$) and RVFAC (41±5 vs. 45±5%, $p<0.05$). There were no significant differences in the wall thickness, left atrial size or indexes of diastolic function including E/E' ratio. The DCM group had a higher prevalence of LBBB and left axis deviation ($p<0.01$) on ECG. The second screen identified 14 healthy mutation carriers (M/F 8/6, 29±12 years) who were compared with 16 mutation-negative relatives (M/F 10/6, 30±13years). No differences were recorded between the groups in ECG or echocardiographic parameters, including left ventricular size, and in systolic or diastolic function. Carriers did not differ from non-carriers in serum NT-proBNP, CK or troponin I.

We conclude that genetic diagnosis is instrumental for risk stratification and guided clinical follow-up in familial DCM.

Clinical and Hemodynamic Outcome of Inferobasal Wall Injury Complicating Alcohol Septal Ablation in Hypertrophic Cardiomyopathy

Andre Keren, Marina Potekhin, Israel Gotsman, Donna Zwas, Morris Mosseri, Ronen Beeri, David Leibowitz, Galina Levin, Haim Danenberg, Arthur Pollak, Dan Admon, Chaim Lotan

Heart Institute, Hadassah University Hospital, Jerusalem, Israel

Background: In selected patients with hypertrophic obstructive cardiomyopathy ***(HOCM)*** alcohol septal ablation ***(ASA)*** is used as an alternative to surgical myectomy. Prior to ASA assessment of the region perfused by the targeted septal branch ***(SB)*** is performed in order to avoid alcohol delivery or leakage to remote areas. We report the clinical signs and outcome of inferior wall injury ***(IWI),*** a rare and not fully understood complication of ASA.

Patients: ASA was performed in 26 pts, 17 (65%) males, aged 33-78 years (60±12). Follow up was 32±30 months. IWI was diagnosed in 3 of the 26 pts (11%) and in an additional case followed in our institution but not part of the consecutive series. ASA was performed as a palliative procedure in 1 of the 4 pts with IWI. The pt had multiple oncological diseases, severe HOCM and severe, only partially SAM related, mitral regurgitation.

Results: Following the injection of alcohol, pts with IWI typically developed long lasting chest pain, ST elevation in inferior leads, decreased flow in the PDA and thinning of the basal infero-posterior wall during follow up. Comparison of pts without and those with IWI revealed no differences in age, gender distribution, symptoms, NYHA functional class (3.3± 0.5 vs 3.4±0.3, respectively), echocardiographic maximal wall thickness (19.9±3.2 vs17.8±2.1 mm), maximal LV outflow gradient (72±33 vs 85±17 mmHg), degree of mitral regurgitation (3.1±1.5 vs 4.5±0.6, p=0.08), number of SB ablated and the amount of alcohol injected.

	Alcohol (ml)	CPK	ΔNYHA	ΔGrad	Full Grad Abolition	ΔMR
N0 IWI (n=23)	2.3±1.0	1016±436	-1.2±0.5	-52±29	6 (26%)	-1.8±1.4
IWI (n=4)	2.6±0.9	2609±1092	-2.0±0.7	-73±29	3 (75%)	-2.8±1.9
P value	0.4	**0.03**	**0.04**	0.13	**0.05**	0.30

Δ = change after ASA, Grad = outflow gradient, MR=mitral regurgitation

In 3 of the 4 cases with IWI, angiographic and myocardial contrast echo methods were used to predict the area of ablation. Persistent complete AV block occurred in 2 cases (50%) who had pacemaker or ICD implantation as compared with 16% of pts with no IWI (P=0.14). During the follow up there was an early and remarkable improvement in symptoms in all 4 pts with IWI. Three of the 4 remained asymptomatic and without baseline or provocable gradients 6-120 months after ASA. One patient with organic MR became symptomatic again after development of chronic atrial fibrillation. Only mild basal inferior hypokinesis was found on follow up echocardiograms in 3 of 4 pts. One pt without IWI died of pneumonia and sepsis 2.5 years after ASA.

Conclusions: IWI may occur during ASA despite the use of angiographic and echocardiographic contrast methods expected to predict this complication. IWI was associated with higher peak CK values, tendency for a higher rate of pacemaker implantation, and remarkably favorable hemodynamic and symptomatic outcome. Despite the positive clinical outcome in our small number of pts, all efforts should be made during ASA to minimize the extent of myocardial injury and avoid damage of remote non targeted myocardium, including the base of the inferior wall. Further research into optimal methods of TASH while minimizing myocardial damage should be pursued.