

= 0.001), and Si ($r = -0.58, p = 0.008$), but not with TBF. In pooled subjects, Si emerged as the strongest predictor of plasma leptin levels ($SC = -0.42, p = 0.017$), independently of IVGTT insulin ($SC = 0.34, p = 0.055$) and TBF ($SC = 0.11, NS, \text{joint } R^2 = 0.51, p < 0.001$).

Conclusions: Patients with CHF have elevated plasma leptin concentrations. In CHF, plasma leptin levels are positively related to plasma insulin and inversely related to insulin sensitivity. These relationships may be of pathogenetic importance in the increased energy expenditure observed in CHF.

1096-32 Increased Insulin and Glucose Levels in Heart Failure (HF)

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Background: Abnormalities of glucose (G) and insulin (I) metabolism may play a role in the etiology and progression of HF. This study examined the prevalence and clinical correlation of G and I abnormalities in a stable HF cohort.

Methods: Fasting G and I levels were obtained on 664 HF patients age (sd) 64 (10), EF 28% (7), NYHA-FC: I/II-64%, III/IV-36%.

Results: 177 patients (27%) were known to have diabetes mellitus (DM). Of the remaining 487 patients, 111 (23%) had elevated G (≥ 6.1 mM), with 53 (11%) in the DM range ($G > 7.0$ mM). In patients without known DM, the mean G, I (sd) levels and fasting insulin resistance index ($FIRI = G \times I/25$) were 5.8 (2) mM, 13.3 (19) $\mu\text{U/L}$ and 3.7 (8) (normal (sd), $I = 4$ (7) $\mu\text{U/L}$, $FIRI = 1.4$ (1)). G and I levels were higher in patients with NYHA \geq III than NYHA \leq II. In addition, a greater proportion of patients with NYHA \geq III had DM than NYHA \leq II. (Table)

	Patients without DM			DM n (%)
	G (sd) mM	I (sd) $\mu\text{U/L}$	FIRI (sd)	
NYHA \leq II	5.6 (1.0)	10.2 (10.1)	2.4 (2.8)	100 (24)
NYHA \geq III	6.3 (2.1)	19.6 (29.6)	6.2 (12.5)	77 (32)

* $p < 0.0001$ (log data). ** $p = 0.04$ NYHA \leq II vs NYHA \geq III

Conclusion: G and I abnormalities are common in HF patients and appear to worsen as HF symptoms increase. This suggests that abnormalities of G or I are either a consequence or a predictor of worsening symptoms of HF. Further study is needed to explore the role of G and I abnormalities in HF.

1096-33 Prognostic Value of Second Generation Cardiac Troponin T in Patients With Chronic Heart Failure

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Background: We previously reported that cardiac troponin T (TnT) detected by second generation assay elevated in patients with severe chronic heart failure (CHF) and suggested that latent micro myocardial damage generated in severe CHF patients. In the present study, we tested the hypothesis that TnT would be a prognostic marker of CHF.

Methods: We measured serum second generation TnT levels (Boehringer Mannheim) in 50 patients with CHF and investigated the relationships between cardiac event (death or admission because of worsening CHF) and TnT levels and other clinical parameters (NYHA class, CTR, LVEF and atrial natriuretic peptide level).

Results: Over a follow-up of 3 ~ 27 months, 7 patients were died and 9 patients were readmitted because of worsening CHF. All died patients showed TnT ≥ 0.04 ng/ml. Cardiac event-free rate in patients with TnT ≥ 0.05 ng/ml was significantly lower than that in patients with TnT < 0.05 ng/ml (1 year 27.3 vs 82.6%, $p = 0.0003$). In a multivariate Cox proportional hazard analysis, NYHA class ($p = 0.02$) and TnT ≥ 0.05 ng/ml ($p = 0.002$) were significantly and independently correlated to cardiac event-free rate.

Conclusion: The present study suggests that elevated TnT level identifies patients at increased risk of cardiac event.

1096-34 Growth Hormone Addition for Patients With Congestive Heart Failure: A Randomized, Placebo Controlled Study With Recombinant Human Growth Hormone in Patients With Congestive Heart Failure and Without Growth Hormone Deficiency

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Background: Experimental data in heart failure models, short term infusion

in human congestive heart failure (CHF) and an open trial in patients with idiopathic dilated cardiomyopathy have demonstrated beneficial effects of growth hormone (GH) on cardiac function.

Methods: Twenty patients with mild-moderate CHF (NYHA class II-III) of ischemic (n = 8) or non-ischemic (n = 14) etiology, and an ejection fraction $< 45\%$ were studied in a 3 months double-blind and placebo controlled trial with recombinant human GH (rhGH). They received either placebo (n = 10) or rhGH (n = 10) in a dose of 0.1 IE/kg/week for 1 week, and thereafter 0.25 IE/kg/week or a maximal daily dose of 2 IE subcutaneously. Cardiac function was determined by equilibrium radionuclide angiography at rest and exercise, and by Doppler-echocardiography at rest. A maximal sitting bicycle exercise test and Holter ECG were also used. Patients were evaluated at 0, 2 and 12 weeks after randomisation, respectively.

Results: There was a significant increase in insulin like growth factor I of 140% in the GH group at 3 months compared to baseline. Excellent patient compliance, no serious adverse events and no significant increase in ventricular arrhythmias in the GH group compared to placebo were found. However, no significant effect of rhGH on systolic or diastolic function, or maximal exercise capacity were found. Neither did NYHA functional classification nor dyspnea grade improve.

Conclusion: This is the first double blind and placebo controlled study of long time rhGH administration in patients with CHF of different etiologies. The treatment was safe and without serious adverse events. However no positive cardiac effects were found.

1096-35 Spontaneous Nocturnal Growth Hormone (GH) Secretion and Haemodynamic Profile in Chronic Heart Failure (CHF) Patients

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Background: CHF is characterised by activation of neuroendocrine system proportional to clinical severity. In untreated CHF pts GH level was found higher than in normal subjects. No data are available on GH secretion profile in treated pts with severe heart failure. **Aim** of our study was to assess: 1) GH secretion nocturnal profile in CHF pts in comparison to normal subjects 2) the relationship between GH, insulin growth factor 1 IGF-1, noradrenaline NA, adrenaline A, atrial natriuretic factor ANP and the haemodynamic profile in this pts.

Methods: In 7 normal subjects age-matched, and in 12 males, with post ischaemic CHF, mean age 63 ± 7 , mean EF (ECHO) $20.6 \pm 6.5\%$, in stable standard chronic therapy we evaluated overnight (every 20 min from 10 p.m. to 6 a.m.) spontaneous GH secretion (IRMA, Nichols), baseline IGF-1 (RIA, Nichols, after acid-ethanol extraction) and, only in pts, neuroendocrine profile and haemodynamic parameters via Swan-Ganz right heart catheterization.

Results: The mean nocturnal GH level is higher in pts compared with control group (1.4 ± 0.6 vs 2.5 ± 1.3). We did not found any differences for IGF-1 level between the two groups. According with normal subjects GH mean nocturnal value, patients were divided in two subgroups (5 pts vs 7 pts). Those with higher mean spontaneous GH level (3.3 ± 1.1 vs 1.4 ± 0.8 $\mu\text{U/l}$) have lower IGF-1 level (119 ± 63 vs 236.8 ± 35.9 $\mu\text{U/l}$) and a worse haemodynamic profile (mean PAP 44 ± 6 vs 30 ± 10 mmHg; PCWP 29 ± 6 vs 17 ± 7 mmHg; CO 3.1 ± 1 vs 4.8 ± 1 l/min; PVR 440 ± 138 vs 267 ± 123 $\text{dine/cm}^2/\text{sec}$) and higher sympathetic activation (NA 734 ± 303 vs 436 ± 132 , A 88 ± 60 vs 60 ± 34 , ANP 344 ± 184 vs 214 ± 174).

Conclusions: Our results shown that 1) CHF patients shown on average a higher nocturnal GH secretion respect to normal subjects 2) among this patients, those with normal GH nocturnal secretion have less severe haemodynamic picture and reduced sympathetic hyperactivation, secretion respect to normal subjects 2) among this patients, those with normal GH nocturnal secretion have less severe haemodynamic picture and reduced sympathetic hyperactivation.

1096-36 Cheyne-Stokes Respiration During Awake Day-Time in Chronic Heart Failure: Prognostic Implication

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It has been shown that in chronic heart failure (CHF), respiratory disorders such as Cheyne-Stokes (CS) and periodic breathing (PB), which are a frequent event during sleep, may occur even during day-time in patients (pts) with compensated heart failure. These alterations of breathing cause further ventricular impairment and sympathetic hyperactivation, but their impact on survival is still debated. To address the association between awake breathing disorders and short-term prognosis 155 moderate to severe CHF pts (mean

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