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Variation by Race in Factors Contributing to Heart Failure Hospitalizations

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Background: Hospitalizations for heart failure (HF) are more common in blacks. However, several studies have demonstrated better survival in black compared with white patients after a hospitalization for HF, even within an "equal access" setting of the Veterans Health Administration (VA). The reasons for this paradox of increased hospitalizations in the face of better survival are uncertain. Therefore, we examined variation by race in factors contributing to hospitalization and in severity of illness at the time of HF hospitalization, in an "equal access" setting. **Methods:** We reviewed medical records of 100 veterans (black = 52, white = 48) admitted with HF to a tertiary care VA hospital in 1999, drawn from a larger cohort of 352 veterans hospitalized for HF, that had demonstrated better survival in black patients. We evaluated differences by race in socio-demographic factors, reported behaviors (e.g., compliance with diet and medication), use of outpatient services in the previous year and clinical factors including comorbid illness and severity of illness at admission. Comorbidity was measured by a count of the number of co-morbid conditions. Severity of illness was measured with the APACHE II and EPICAL scores. The chi square test was used for comparison of categorical variables and the t-test for continuous variables. **Results:** There were no significant differences in age, marital status or proportion of diastolic heart failure between black and white patients. Severity of illness at admission (by APACHE and EPICAL) was also similar. However, black patients had fewer comorbid illnesses than white patients. Also, black patients had more frequent documentation of substance abuse, non-compliance with medications and social factors contributing to hospitalization and had fewer primary care visits in the year prior to hospitalization. **Conclusions**: Disease burden as measured by comorbid illness and severity classification was not associated with the higher likelihood of hospital admission among black compared with white patients. However, hospitalized black patients were more likely to be admitted with a history of medication noncompliance, fewer outpatient visits, uncontrolled hypertension, or other social factors. Thus black patients may experience more frequent acute illness that leads to hospitalization, but that also resolves quickly with treatment and is not associated with worse outcomes. Better survival after hospitalization for blacks with HF may be explained at least in part by hospitalization precipitated by non-clinical factors.

Racial Differences at time of HF hospitalization

	Whites	Blacks	p value
Age	66.8 ± 1.4	64.7 ± 1.6	0.33
Substance Abuse	4.1%	23%	0.006
Primary care visits in past year	4.5 ± 0.4	3.3 ± 0.3	0.02
Non-compliance with medications	16.7%	38.5%	0.01
Admission for social reasons	2.0%	19.2%	0.006
Systolic BP	132.9 ± 0.7	150 ± 0.6	0.006
APACHE II score	14.7 ± 0.4	14.5 ± 0.6	0.72
No. of comorbid illnesses	3.0 ± 0.2	2.4 ± 0.2	0.008
Diastolic HF	31.2%	26.9%	0.63

Values are mean ± s.e. unless otherwise stated.

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Results from a Pilot Study To Determine the Feasibility in Transitioning Outpatient CHF Patients from Intermittent Intravenous Inotrope Therapy to Nesiritide

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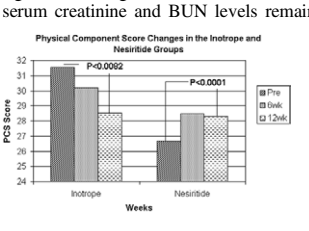
Background: The use of outpatient inotrope infusion for CHF has been heavily critiqued, in particular due to increased mortality associated with long-term inotrope use. Recently an alternative therapy for outpatient maintenance of advanced CHF patients has been identified for potential study. Nesiritide offers a unique ability to affect hemodynamic, natruretic and neurohormonal responses in the CHF patient. It is theorized that nesiritide will maintain/improve hemodynamics and symptoms in end-stage CHF patients. **Methods:** This is an open-label two-arm pilot study evaluating the hemodynamic, clinical and quality of life effects of nesiritide compared to inotropes. 30 stable patients were enrolled who were receiving standard outpatient inotrope therapy with milrinone and/or dobutamine. 16 patients were maintained on their current regimen. 14 patients were switched to nesiritide based on their willingness to try a different therapy. Nesiritide was administered at 2 μg/kg bolus followed by 0.01μg/kg/min infusion for four hours, twice per week. Hemodynamics (CI, SVRI, TFC, LCWI) were collected weekly using standard bioimpedance measurement. Short Form 36 questionnaires (SF36) were administered every six weeks for monitoring of quality of life issues. Endpoints included hemodynamics, SF36 scores, hospitalizations, mortality, renal function and diuretic use. **Results:** Among these NYHA class IIIb/IV patients; the nesiritide group was initially more ill than the inotrope group based on the physical component scores (PCS) of the SF36, 27 vs 32, respectively. Despite this initial difference, significant reduction in PCS was noted in the inotrope group ($p = 0.0082$), while PCS scores significantly improved in the nesiritide group ($p < 0.0001$). During the 3-month period of the study, no significant difference in hemodynamics was noted following the transition from inotropes to nesiritide. One patient in each group was hospitalized for CHF. No CHF related deaths occurred. No significant changes in diuretic use were noted between groups. In the nesiritide group serum creatinine and BUN levels remained stable. **Conclusions:** Nesiritide therapy showed the same hemodynamic benefit that has been observed with inotrope use in an outpatient setting. However nesiritide patients showed a clinically significant improvement over inotropes with respect to their PCS scores, indicating improvement in their quality of life associated with physical functioning. Further study with a larger sample and at multiple centers is warranted.



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Troponin T, but Not High-Sensitivity C-Reactive Protein, Predicts Outcomes in Patients Hospitalized with Acute Decompensated Heart Failure

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Background: Necrosis and inflammation are among the mechanisms proposed for exacerbation of chronic heart failure. However, the meaning of markers such as troponin T (TnT) and high-sensitivity C-reactive protein (hs-CRP) have not been yet thoroughly explored in this context. The purpose of this study was to assess the prognostic value of admission TnT and hs-CRP for the risk stratification in patients hospitalized with acute decompensated heart failure (ADHF). **Methods:** We conducted a prospective, multicenter, cohort study. Inclusion criteria: Patients admitted due to ADHF. Exclusion criteria: myocardial infarction within 3 months or suspected acute coronary syndrome. Serum samples were obtained at admission. Measurements were performed by a core-lab with a 3rd generation immunoassay for TnT and immunoturbidimetric assay for hs-CRP. The investigators were blinded of the test results until the end of the study. The primary outcome was the composite of death and/or cardiovascular re-hospitalization at 90 days. **Results:** 59 consecutive patients were included: median age 69 (58,79), male 68%, history of coronary disease 47%, hypertension 59%, diabetes 22%. The median CK value was 83U/L (54,152). The median TnT level was 0.03 ng/ml (0,0.06). Forty patients (68%) had TnT levels above 0.01 ng/ml (median 0.04 ng/ml [0.03,0.07]). Patients with events at 90 days had higher levels of TnT than those with no events: 0.04 ng/ml [0.01,0.09] vs 0.02 ng/ml [0,0.04] $p = 0.014$. Primary end-point and events according to TnT level are shown in the table below. Patients with TnT > 0.01 ng/ml were categorized according to the level of TnT below or above 0.07 ng/ml (percentile 75th of this population). The rate of events increased according to the marker level: TnT < 0.01: 21%, TnT 0.01-0.07 : 39%, and TnT > 0.07: 89%, $p = 0.0015$ (χ^2 test for trend). Fifty one patients (86%) had hs-CRP above 5 mg/L. The median value of hs-CRP at admission was 17.1 mg/L (8.1,50.2). No difference was observed in the event rate when patients were grouped by hs-CRP levels. **Conclusion:** This pilot study provides evidence that TnT concentration is a useful initial marker for risk stratification in patients hospitalized with ADHF including those without coronary disease. Although significantly elevated levels of hs-CRP were found, a relationship with the outcome could not be assessed at three months follow-up. Additional studies will be necessary to clarify the clinical value of our initial findings.

Primary end-point and events according to TnT level

End-points (EP)	All Patients (n = 59)	TnT < 0.01 (n = 19)	TnT ≥ 0.01 (n = 40)	P value
Primary EP (Death and/or re-hospitalization)	24 (41%)	4 (21%)	20 (50%)	0.034
Death	7 (12%)	1 (5%)	6 (15%)	0.41
Re-hospitalization	19 (32%)	3 (16%)	16 (40%)	0.063

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Impact of Carvedilol on QT Interval Dispersion in Patients with Chronic Heart Failure

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Background: The role of QT interval dispersion (QTD) in heart failure (HF) remains poorly defined and controversial. Our objective was to evaluate the impact of QTD in the clinical evolution of chronic HF patients under control due to the use of carvedilol (CVD). **Methods:** We selected 108 patients (22-82 years), 65.7% were male, 72.2% caucasians, with stable chronic HF and NYHA functional class (FC) II, III and IV on optimized treatment, with an left ventricle ejection fraction (LVEF) of ≤0.40 for therapy with CVD. All patients had complete history taken and physical exam performed, as well as, laboratorial evaluation, electrocardiogram, echocardiogram and followed-up at the HF clinic for an average period of 38.2 months. All used CVD at the maximum tolerated dose. The evaluated parameters were: general characteristics of the population, cardiopathy etiology, concomitant medication used, NYHA's FC, maximum CVD dose, LVEF and QTD before and 6 months after CVD and deaths. **Results:** A QTD reduction (109ms to 72ms) and an increase in the LVEF (0.27 to 0.35) was found after 6 months of therapy with CVD ($p < 0.001$). The general characteristics of the population ($p > 0.05$), concomitant medications ($p > 0.05$), CVD dose ($p = 0.80$), cardiopathy etiology ($p = 0.959$) did not influence in the QTD reduction. This reduction was related to the patients with worse NYHA FC pre-CVD ($p = 0.007$) and with NYHA FC improvement ($p = 0.028$). The ROC curve analysis post-CVD determined that QTD > 90ms was a mortality predictor ($p = 0.034$; OR = 3.912) (AUC = 0.061; sensibility = 29.2%; specificity = 90.5%; positive likelihood ratio = 3.06; negative likelihood ratio = 0.78). The multivariate analysis showed that the QTD reduction was an independent survival predictor ($p = 0.004$; OR = 5.48). **Conclusions:** CVD reduced the QTD and increased the LVEF in patients with chronic HF. We did not observe interaction among QTD reduction and population characteristics. The QTD reduction was not influenced by concomitant medications, cardiopathy etiology and CVD dose. The largest QTD reduction occurred specially in worst patients (NYHA FC III and IV). The presence of QTD reduction was an independent predictor of survival and the QTD > 90ms post-CVD was predictor of mortality.