

Figure: Hemodynamic data before(baseline), during (8, 24, and 72 hrs) and after (24 hrs) treatment with CRS. \* = p< 0.05 vs Baseline.

## 1088-124 Pletal in Lieu of Home Parenteral Inotropes

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Despite successful treatment with poly drug regimens for heart failure, some patients cannot be discharged from the hospital because of persistent symptoms or complications of heart failure. In order to facilitate home discharge we offered twelve patients with inotrope dependent failure, discharge on Pletal, an oral phosphosdiesterase inhibitor similar to Milrinone. Reasons for inotrope dependence included: limiting symptoms of low cardiac output, hyperkalemia, cognitive dysfunction, renal insufficiency, recurrent pleural effusions. All patients were informed of their prognosis as well as the previously noted increased risk of sudden death associated with continuous inotrope administration. Eleven of 12 patients had previously placed AICDs. All patients were on optimal dosages of carvedilol (mean 37.5 mgs/day). Pletal dosage was 100 mgs BID. Two elderly patients died at home, per their desire, within 3 weeks of discharge. Neither death appeared to be associated with tachyarrhythmia. In late survivors (mean 16months, range 3-22months) there have been two late deaths at 3 and 22 months associated with sudden death and malignancy respectively. No patient has required re-hospitalization. Appropriate AICD device discharges have occurred in 3 patients, 1 spontaneous, 1 hypokalemia related and 1 associated with increased Pletal dosage. All patients are able to perform activities of daily living, socialize and shop outside the home, and feel that the risk of sudden death is acceptably counterbalanced by improvements in quality of life. Oral Pletal therapy appears to facilitate hospital discharge, reduce readmission, and allows for an acceptable quality of life outside the hospital.

## 1088-125

## Levosimendan Is Safe and Effective in Patients With Severe Low Cardiac Output Heart Failure and Critical Hypotension

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Background: Management of decompensated advanced heart failure, severe hypotension, and progressive cardiogenic shock requires immediate inotropic therapy to restore cardiac output (CO) and blood pressure (BP). Levosimendan (levo) is effective in heart failure patients with low CO but relatively preserved BP. However, it is unknown if levo is effective when CO and BP pressure are both critically low.

**Methods**: Fifteen patients  $(64\pm18)$  years; LVEF  $21\pm3\%$ ) admitted to ICU due to decompensated chronic heart failure with critical hypotension and progressive cardiogenic shock, treated with medium-high doses of dobutamine (dob) and dopamine (dop) upon arrival, were given 0.1 mcg/kg/min levo in the first 24 hours after admission to ICU (Group 1). Clinical and hemodynamic improvement at 48 h after treatment, in-hospital mortality, and length of hospital and ICU stay were compared to 11 patients  $(59\pm18)$  years; LVEF  $20\pm3\%$ ), with the same baseline characteristics, treated with dob and dop (Group 2).

**Results:** Dob/dop were weaned at  $62 \pm 38$  hours in Group 1 and at  $120 \pm 48$  hours in Group 2 (P < 0.002). Hospital length of stay (LOS) was  $14 \pm 5$  days in Group 1 and  $22 \pm 8$  days in Group 2 (P < 0.01). ICU LOS was  $5 \pm 3$  days in Group 1 and  $9 \pm 5$  days in Group 2 (P < 0.05).

Conclusion: Our preliminary data suggest that introducing levo to conventional therapy at early stages of treating patients with severe heart failure and cardiogenic shock improves hemodynamic recovery and hospital outcomes compared to traditional intervention.

Clinical parameter*/ Outcome	Group 1 Levo, Dob/Dop (n=15)		Group 2 Dob/Dop (n=11)	
	Baseline	48 h	Baseline	48 h
Systolic BP (mmHg)	75 ± 9	91 ± 8 †	77 ± 6	93 ± 7.8
Heart Rate (beats/min)	106 <u>+</u> 16	100 <u>+</u> 12 †	103 <u>+</u> 20	107 <u>+</u> 16
Diuresis (mL/hr)	22 <u>+</u> 20	242 ± 159 †	30 <u>+</u> 27	114 <u>+</u> 112 †
Creatinine (mg/dL)	3.3 <u>+</u> 1.7	2.3 <u>+</u> 1.6†	1.5 <u>+</u> 0.7	2.8 <u>+</u> 2.4
In-hospital mortality (%)	27		64	

<sup>\*</sup> Mean <u>+</u> SD; †P < 0.05

all values: mean ± S.D

## 1088-126 Nesiritide Does Not Improve Renal Function in Patients With Chronic Heart Failure and Worsening Creatinine

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**Background:** Decompensated heart failure (CHF) is often associated with worsening renal function, a condition which limits therapeutic options. We characterized the effects of nesiritide on renal function in these high-risk patients with the greatest need for an intervention which improves kidney function.

**Methods**: In this randomized, double-blind, placebo controlled, cross-over study, 15 patients admitted for worsening heart failure, ejection fraction < 40%, & creatinine  $\geq 0.2$  mg/dl over baseline were given 24 hour infusions of nesiritide (2  $\mu$ g/kg bolus then .01  $\mu$ g/kg/min) or placebo during randomized consecutive 24 hour periods. Blood & urine samples were obtained and urine output recorded at hours 3, 6, 21, & 24 for each period. Glomerular filtration rate (GFR, by iothalamate), renal plasma flow (RPF, by PAH), urine output, and sodium excretion were measured for each time point. Diuretics & vasoactive drugs were held for 6 hours each day.

Results: There was no effect of nesiritide on urine output, sodium excretion, GFR, or RPF

	0-3 Hours	3-6 Hours	6-21 Hours	21-24 Hours	24 Hour Total.
GFR (ml/	min)				
placebo	43.4 ± 31.4	48.5 ± 30.3	41.4 ± 27.9	44.9 ± 27.9	43.1 ± 27.0
active	41.7 ± 31.1	42.5 ± 24.8	44.3 ± 30.0	38.2 ± 21.3	43.2 ± 27.2
RPF (ml/r	min)				·
placebo	130 ± 81.5	155 ± 74.9	156 ± 80.2	182 ± 109	160 ± 70.6
active	113 ± 55.5	121 ± 55.4	153 ± 88.3	127 ± 63.2	145 ± 69.9

Urine output (ml)					
placebo	$217 \pm 93$	226 ± 157	1988 ± 939	306 ± 305	2717 ± 1231
active	206 ± 97	268 ± 227	1877 ± 1077	263 ± 230	2634 ± 1349

Na Excretion (mmol/hr)					
placebo	5.19 ± 5.59	4.54 ± 5.27	9.94 ± 8.17	7.05 ± 12.9	8.51 ± 7.53
active	3.60 ± 2.55	5.70 ± 7.96	9.78 ± 8.59	7.27 ± 9.94	8.30 ± 6.67

Conclusion: In this population of patients with acute exacerbation of CHF and worsening renal function, nesiritide infusion exerted no significant impact on urine output, GFR, RPF, or sodium excretion as compared to placebo. While previous studies with higher doses of nesiritide have demonstrated increased sodium excretion and/or urine output, the present study suggests that these effects are not present with lower doses of nesiritide in patients with worsening renal function.