

0.78,  $p < 0.003$ ), TBW ( $r = 0.6$ ,  $p < 0.03$ ), and TBNa ( $r = 0.7$ ,  $p < 0.001$ ). In patients with high output CHF, aldosterone was not elevated but correlated with PRA ( $r = 0.8$ ,  $p < 0.02$ ) and ERPF ( $r = -0.75$ ,  $p < 0.003$ ). When all the groups were taken together, aldosterone continued to correlate strongly with PRA but now a weak correlation emerged with right atrial ( $r = 0.46$ ,  $p < 0.02$ ) and pulmonary wedge ( $r = 0.45$ ,  $p < 0.03$ ) pressures and cardiac output ( $r = -0.43$ ,  $p < 0.03$ ). We conclude that aldosterone is elevated in patients with untreated low but not high output CHF. It correlates well with PRA in all groups suggesting that PRA continues to determine aldosterone levels even in such diverse clinical states but poorly with indices of salt and water retention.

**994-99 Neurohumoral Activation and Hemodilution Under Parenteral Administration of Molsidomine and Isosorbide Mononitrate**

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Hemodilution and neurohumoral activation following vasodilation are thought to be major factors for the development of nitrate tolerance. To determine whether these mechanisms are part of the action profile of molsidomine (M), we investigated the hemodynamics, neurohumoral activation, and hemodilution during i.v. administration of M and isosorbide-5-mononitrate (I) in 15 patients with CHF (NYHA II-III). In a randomized, double-blind, cross-over design, each patient received a 24-hour infusion of M (6 mg/h), I (3.75 mg/h) and placebo (P). Measurements were performed at baseline, at 2 h, at 8 h, and at 24 h. Results (mean  $\pm$  SEM): Both vasodilators caused a continuous increase in plasma volumes with a maximum at 24 h: I  $+8.2 \pm 2.2\%$  ( $p < 0.0001$ ), M  $+18.5 \pm 2.7\%$  ( $p < 0.0001$ ). Plasma volumes did not change on placebo. The plasma renin activity (ng/ml/h) is shown below:

	Baseline	2 h	8 h	24 h
P	7.50 $\pm$ 2.37	6.95 $\pm$ 2.29	7.82 $\pm$ 2.17	7.53 $\pm$ 2.41
I	7.71 $\pm$ 2.32	9.61 $\pm$ 2.62*	10.15 $\pm$ 2.32**	8.20 $\pm$ 2.14*
M	6.53 $\pm$ 2.17	12.01 $\pm$ 2.49***	15.36 $\pm$ 2.65****	12.51 $\pm$ 2.74****

\* $p < 0.05$ , \*\* $p < 0.01$  (vs P), \* $p < 0.05$ , \*\* $p < 0.001$  (vs baseline) # $p < 0.05$  (I vs M)

Levels of Angiotensin II, aldosterone and vasopressin did not change significantly. Hemodynamically, a significant improvement of CO and diastolic PAP was observed at 2 h on both treatments. This effect was maintained on M at 8 h and 24 h ( $p < 0.05$ ), but not on I.

Conclusion: Continuous infusion of I led only to a moderate hemodilution and neurohumoral activation, but development of tolerance was observed starting at 8 h. In contrast, administration of M resulted in prolonged hemodynamic effects, although pronounced changes in plasma volume and neurohumoral activation occurred. We conclude that intravascular fluid shift and/or neurohumoral activation is not a major factor in the development of nitrate tolerance.

**994-100 Haemodynamical Effects of L-Carnitine on Patients With Congestive Heart Failure Due to Dilated Cardiomyopathy**

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The haemodynamical effects of L-Carnitine (LC) were studied on 34 patients (pts) in a double blind study. Pts with dilative cardiomyopathy (CM) and clinical stable heart failure (HF) (NYHA III-IV) were included in the study. All pts underwent left and right cardiac catheterization (CC) and cardiopulmonary exercise test (CPX) according to Weber protocol, in a baseline (BL) examination. After one (1M) and three months (3M) treatment with placebo (18 pts) and LC (2 g/die per os)(16 pts) right CC and CPX were repeated. All conventional drugs for HF remained unchanged during the study. The following mean values were determined prior to (b) and after (a) CPX: maximal time of CPX (CPX-T) (min), peak O<sub>2</sub> consumption (VO<sub>2</sub>) (ml/min/kg), arterial/pulmonary blood pressure (ABP/PBP) (mmHg), cardiac output (CO) (L/min).

**Results:**

	Placebo			L-carnitine		
	BL	1M	3M	BL	1M	3M
CPX-T	7.2	6.8	7.0	8.2	9.4*	9.5*
VO <sub>2</sub>	12.3	11.0	11.2	13.2	14.3*	14.9*
ABP(b)(S/D)	120/85	133/87	131/82	127/83	115/78*	133/83
ABP(a)(S/D)	142/96	151/94	166/96	150/92	154/95	165/95
PBP(b)(S/D)	37/13	38/17	38/18	34/12	26/8*	32/12
PBP(a)(S/D)	42/17	42/12	41/14	41/14	38/12	40/13
CO(b)	4.2	4.3	4.1	4.2	4.4	4.2
CO(a)	7.8	7.9	7.8	7.9	9.5*	9.8*

Systolic/diastolic (S/D), \* $p < 0.01$ , placebo vs L-carnitine

Conclusions: LC has improved CC and increased the duration of CPX. VO<sub>2</sub> was increased in one as well in 3 months after LC administration, thereby improving the functional status.

**995 Coronary Heart Disease in the Elderly: Evaluation, Management, and Outcomes**

Wednesday, March 27, 1996, 9:00 a.m.--11:00 a.m.  
Orange County Convention Center, Hall E  
Presentation Hour: 10:00 a.m.--11:00 a.m.

**995-61 Stents in the Elderly for Failed PTCA, Early and Intermediate Term Results**

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In order to determine relative acute and intermediate-term outcomes, 113 patients (pts) over age 65 undergoing intracoronary stent placement for acute or threatened artery closure during percutaneous transluminal coronary angioplasty (PTCA) were compared with 74 pts  $< 65$  y receiving stents and with 1,602 pts  $\geq 65$  y having non-emergent PTCA, 1988 through 1994. Compared with the younger pts and the elderly PTCA pts, the older stent pts had similar important baseline characteristics. The technical success rate was high in all three groups, 89.4% elderly stent group, 93.2% younger stent group, 90.9% elderly PTCA pts ( $p = NS$ ) and the rate of emergency bypass surgery was low; 2.7%, 5.4%, and 1.1% respectively ( $p = NS$ ). The rate of mortality for the elderly stent pts was 4.4% vs 0% for stent pts  $< 65$  y ( $p = 0.067$ ) and 2.2% for PTCA pts  $\geq 65$  y ( $p = NS$ ). The in-hospital MI rate was 3.5% for elderly stent pts vs 2.7% for stent pts  $< 65$  y ( $p = NS$ ) and 0.8% elderly PTCA group ( $p < 0.003$ ). The incidence of bradycardia was higher in the elderly stent pts (11.5%) than younger stent pts (1.4%) ( $p < 0.01$ ) or the elderly PTCA pts (4.1%) ( $p < 0.001$ ). The elderly stent group also had a more frequent need for blood transfusion (25.7%) and higher incidence of renal insufficiency (2.7%) vs the elderly PTCA group (6.1%,  $p < 0.0001$  and 0.5%,  $p < 0.05$ ), but not compared with the younger stent group (21.6% and 0%,  $p = NS$ ).

Survival free of MI was high for all three groups, 91.7% elderly stent pts, 98.3% younger stent pts, and 90.5% elderly PTCA pts at one year.

Thus, in pts with stents placed for PTCA failure, need for blood transfusion and other vascular complications were frequent but in-hospital and intermediate term event free survival were excellent in the elderly and is similar to that of elderly pts undergoing PTCA without need for stents.

**995-62 Outcome of Right Ventricular Infarction in Elderly Patients**

Héctor Bueno, Ramón López-Palop, Javier Bermejo, José L. López-Sendón, Juan L. Delcán. *Hospital General "Gregorio Marañón", Madrid, Spain*

It is known that, among patients (pts) with inferior myocardial infarction, those who develop right ventricular infarction (RVI) have a worse prognosis, and that LV diastolic dysfunction is a common feature in elderly pts, in whom cardiac output is more dependent on LV filling pressure. Therefore, it might be anticipated that RVI should have a particularly negative effect in the hemodynamics of inferior AMI in elderly pts. To confirm this hypothesis and to investigate its clinical significance, we studied the in-hospital outcome of 78 consecutive pts  $\geq 75$  years old admitted to the CCU with a first AMI of inferior location. We compared the pts with ECG or echocardiographic evidence of RVI ( $n = 30$ , 38%) with those without RVI. Baseline characteristics were similar in both groups except for age, which was higher in pts with RVI (81 vs 79 years old,  $p = 0.01$ ). LV ejection fraction did not significantly differ in both groups (0.47 in pts without RVI vs 0.43 in pts with RVI). The most significant results are shown in the table.

WEDNESDAY POSTER