

tal muscle is similar to that of respiratory muscle, but the former shows anaerobic metabolism at an earlier stage of exercise than the latter. This phenomenon is more prominent in patients with CHF.

981-51

### Reduced Ventilatory Efficacy in Patients with Chronic Heart Failure is Associated with Impairment of Pulmonary Gas Transfer

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Exertional hyperpnea in chronic congestive heart failure (CHF) is largely caused by ventilation of increased physiological dead space.

To investigate the pathogenesis of reduced ventilatory efficacy we investigated 71 pts with CHF due to dilated cardiomyopathy (n = 56), coronary artery disease (n = 11) and valvular heart disease (n = 4) with left ventricular ejection fraction of 28 ± 11%. Single breath measurement of pulmonary transfer factor for carbon monoxide (TLco), its subdivisions capillary blood volume (Qc) and membrane diffusing capacity (Dm), as well as cardiopulmonary exercise testing were performed. Ventilatory efficacy was assessed by calculation of the slope of the linear relation between minute ventilation (VE) and carbon dioxide output (VCO<sub>2</sub>) and compared to parameters of the pulmonary function. Patients were divided into two prospectively designed subgroups with normal or reduced ventilatory efficacy (VE/VCO<sub>2</sub> slope ≤35, n = 46 vs >35 l/l VCO<sub>2</sub>, n = 25). Pts with normal ventilatory efficacy showed normal pulmonary carbon monoxide transfer (9.5 ± 2.4 mmol/kPa/min), whereas pts with exertional hyperpnea revealed significantly impaired TLco (6.6 ± 1.5, p < 0.001). Qc and Dm were significantly reduced in pts with vs without hyperpnea (Qc: 61.3 ± 18.7 vs 71.3 ± 20.4 ml, p = 0.02, Dm: 12.2 ± 5.6 vs 17.6 ± 6.3 mmol/kPa/min, p < 0.001).

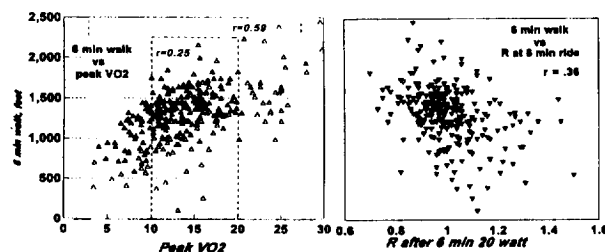
These data suggest that reduced ventilatory efficacy in patients with chronic heart failure is caused by hypoperfusion of alveolar capillaries and by impaired characteristics of the diffusive membrane.

981-52

### Six-minute Walk Compared to Peak and Low-level Aerobic Capacity in 302 Patients with Heart Failure

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Distances walked spontaneously during 6 min may reflect peak exercise capacity, the ability to sustain 6 min of low exercise without anaerobic metabolism, and non-metabolic factors such as stride. To determine how well 6-min walks in 302 heart failure patients reflected aerobic capacity at peak exercise (pkVO<sub>2</sub>) and/or R (VCO<sub>2</sub>/VO<sub>2</sub>) after 6 min of low-level exercise similar to walking, 6-min walks were measured within 48 hrs of bicycle exercise with gas exchange during 6 min 20-watt riding and then during incremental exercise.



Although 6 min walk correlated with extremes of pkVO<sub>2</sub>, it varied widely (r = 0.25) when pkVO<sub>2</sub> was 10–20 ml/kg/min (generally Class II-III). Although 6 min 20W ride required VO<sub>2</sub> 9 ± 2 ml/kg/min, similar to 3 METS estimated for walking, 6-min 20W R did not correlate well inversely with 6 min walk distance except at very short and long walks.

In moderate heart failure, 6 min walk reflects factors other than aerobic capacity at peak or during 6 min of sustained low-level exercise.

981-53

### Impaired Ventilatory Adaptations to Submaximal Exercise in Patients with Chronic Heart Failure

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The physiologic determinants limiting maximal exercise performance in patients with congestive heart failure (CHF) have not been completely characterized. Cardiac, peripheral, and ventilatory responses to exercise are attenuated in patients with CHF. While ventilatory factors are not believed to limit maximal exercise, several abnormalities of pulmonary function have been

documented. Maximal voluntary ventilation (MVV) is a brief hyperventilatory maneuver that is usually performed at rest. We hypothesized that this maneuver performed during exercise could further elucidate exercise-induced pulmonary abnormalities in patients with CHF. Accordingly, MVV (in L/min, determined by a 12-second maximal hyperventilatory effort) was measured at rest and during 10 minutes of submaximal bicycle exercise (at 55% of a previously determined maximal effort) in 9 normal subjects (NL) and 9 patients with CHF (mean EF 0.23). **Results:**

	VO <sub>2</sub> max	Peak V <sub>E</sub>	Rest MVV	Exercise MVV
NL	38.3 ± 7.1	132 ± 32	175 ± 42	193 ± 36
CHF	15.4 ± 1.9*	73 ± 13*	143 ± 28	143 ± 25*

Peak V<sub>E</sub> = peak ventilation during maximal exercise, \*P < 0.05, CHF vs normals

Although ventilatory factors did not appear to limit exercise (since MVV >> peak V<sub>E</sub> in both NL and CHF), MVV increased during submaximal exercise only in normal subjects but not in patients with CHF (+11% vs +0%, P < 0.05). The exercise-induced increase in MVV in normal subjects may represent an adaptive response to exercise related to exercise-induced bronchodilation or sympathetic stimulation of the diaphragm. These mechanisms may be attenuated in CHF secondary to bronchial edema from high left ventricular filling pressures, or due to intrinsic diaphragmatic muscle abnormalities. The absence of normal ventilatory adaptations to exercise may contribute to exercise intolerance in patients with CHF.

982

### Gender Differences — Diagnostic and Therapeutic

Tuesday, March 21, 1995, 3:00 p.m.–5:00 p.m.

Ernest N. Morial Convention Center, Hall E

Presentation Hour: 4:00 p.m.–5:00 p.m.

982-68

### Outcome Following Stress Testing: Are There Any Gender Related Differences? A Population Based Study

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Gender differences in management after stress tests have been documented, possibly reflecting gender bias. Whether these differences impact on long-term outcome is not known but is critical to assess appropriateness of care. We conducted a retrospective population based cohort study of 604 persons (413 men, 191 women) who had a first stress test in 1987 in Olmsted County MN with complete follow up until 1994 or death. At study entry, women were older (55 ± 17 years old vs 48 ± 14 years old for men) and more likely to have typical angina (38% vs 29% of men), hypertension (37% vs 30% of men), a family history of coronary artery disease (72% vs 59% of men), and a positive stress test (19% vs 12% of men) (p < 0.05). The frequency and relative risks (RR) of cardiac event (myocardial infarction, congestive heart failure cardiac death) with and without inclusion of revascularization (revasc) and overall death were:

	Women	Men	RR	95% CI
Cardiac event	14 (7%)	36 (9%)	0.8	0.4 to 1.5
Cardiac event incl revasc	18 (9%)	66 (16%)	0.5	0.3 to 0.9
Overall death	16 (8%)	19 (5%)	1.7	0.9 to 3.4

After adjusting for history of myocardial infarction, symptoms, age, stress test results and comorbidity, no gender difference in risk of death was seen. Women with a negative stress test were at a lower risk of cardiac event (RR 0.4, 95% CI 0.16 to 1.0); when revascularization was included as cardiac event, the relative risk was lower in women than in men (RR 0.4, 95% CI 0.2 to 0.6). **Conclusion:** These population based data suggest that there is no excess risk of overall death, cardiac death or myocardial infarction in women. The risk of cardiac event including revascularization, however, was lower in women: this may represent underutilization of revascularization in women or overutilization in men.

982-69

### Referral to Coronary Angiography After Stress Testing: Are There Any Gender Differences in a Population Based Setting?

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Previous studies reported conflicting results on gender bias in the referral to coronary angiography after exercise stress testing. Tertiary care center set-