with group participation via conference call, was developed with the goal of increasing accessibility of CR. Participants (N = 60) were post myocardial infarction, revascularization or cardiac transplantation, and had limited geo- graphical access to our program. Outcomes of peak aerobic capacity (Peak VO₂), exercise time and quality of life measures (Medical Outcomes Study – SF-36) were assessed before and after 36 sessions of 3 X per week exercise and teaching versus a control group (N = 26) of standard "institutional" CR patients. In the home exercise group, peak VO₂ increased from 18.3 ± 4.7 ml/kg • mln (-20%) by 21.5 ± 5.2 ml/kg • mln (-20%) and exercise time increased from 11.0 ± 4.0 to 14.1 ± 4.7 min (+29%), both p < 0.001, versus increases of 25% and 59% respectively in controls (p = ns between groups). Quality of life scores for physical functioning and physical role limitations improved in the home group by 14% and 44% (p = 0.001), similar to the 21% and 49% in controls. There were no major exercise related adverse effects in over 250 sessions of home monitored exercise. Thus home monitored CR provides improvements of physical function similar to institutional CR and extends participation to patients with limited geographic access to an established program.

741-2 Exertional Syncope in Cardiac Amyloidosis — A Common and Ominous Symptom of Restrictive Cardiomyopathy
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Primary amyloidosis is a systemic disorder characterized by multi-organ involvement including the heart, kidney and autonomic nervous system. Cardiac amyloid infiltration involves the myocardium, conduction system and valves and causes a restrictive cardiomyopathy with mortality due to sudden death or CHF. Syncope in amyloidosis may be non-cardiac or cardiac and, based on the pathology, both arrhythmia and heart block may be expected. To investigate causes of syncope, we retrospectively examined the records of 185 cases of biopsy-proven amyloid involving the heart and identified 26 cases (14.1%) of syncope. After investigation (including Holter, SAECG, and EP) the following well-recognized etiologies were determined: autonomic neuropathy in 7, ventricular arrhythmias in 3, heart block in 2, and miscellaneous/undetermined in 5. In 9 additional patients we identified a syndrome characterized by syncope with mild to moderate physical (6) or emotional (3) stress and occasionally associated with post syncope cyanosis. All 9 had severe cardiac amyloidosis (mean LV wall thickness 16 ± 12 mm), normal to small LV cavity and restrictive pathophysiology. NYHA CHF ranged from class 2 to 4. Exercise testing reproduced symptoms in 3 of 4 patients including cardiac arrest due to electromechanical dissociation in 1. Death occurred within 1 year of diagnosis in 6/9 (mean 4 months); and was sudden in 6 despite a pacemaker in 2 and AICD in 1. Conclusion: Cardiac syncope is common and multifactorial in cardiac amyloidosis. The commonest single cause is associated with exertion/stress and carries a very poor prognosis. The most likely mechanism is due to a restrictive cardiomyopathy with failure to augment the already reduced cardiac output, possibly in association with peripheral vasodilation, resulting in myocardial and cerebral hypoperfusion.

741-3 Effects of Training on Exercise Hemodynamics, Left Ventricular Volume, and Contractility in Chronic Heart Failure: Application of MRI
Background: There remains uncertainty as to central hemodynamic and left ventricular volume adaptations to exercise training in CHF. MRI offers an advancement in precision over previously available methods.
Methods: Twenty-five patients with first diagnosis of CHF due to CAD were randomized to a control (n = 13, age 55 ± 7 years, EF 33 ± 6%) and a training group (n = 12, age 56 ± 5 years, EF 32 ± 7%). Patients in the training group performed two hours of daily walking along with four sessions per week of high intensity monitored stationary cycling (40 minutes at 70 to 80% peak capacity) at a residential rehabilitation center for two months. Control patients received usual community care. Ventricular volume measurements with Cine-MRI, ventilatory gas exchange, and upright hemodynamic measurements (rest and peak exercise cardiac output, pulmonary artery, wedge, and mean arterial pressures, and systemic vascular resistance) were performed before and after the study period.
Results: Peak VO₂ increased in the trained group by 25% (p < 0.05) and did not change in controls. Parallel increases in maximal cardiac output and a widening of the a-V0₂ difference were observed in the trained group. None of the hemodynamic pressure measurements, ejection fraction, or volume measurements by MRI differed at rest or peak exercise within or between groups.
Conclusions: Intensive exercise training improves exercise capacity in CHF through the combination of an increase in maximal cardiac output and a widening of the a-V0₂ difference, but does not changes in ventricular function or central hemodynamics. Training does not lead to further myocardial deterioration as reflected by MRI measures.

741-4 Both Pulmonary Factors and Right Heart Function Limit Exercise Capacity in Patients With Congenital Heart Defects
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To clarify cardiopulmonary factors contributing to exercise capacity in pts with congenital heart disease (CHD), 31 pts and 21 age matched controls (NML) underwent maximal (Max) upright bicycle exercise with measurement of O₂ consumption (VO₂) and, in CHD pts, rest and exercise right ventricular ejection fraction (RVEF) using radionuclide angiography. Change in RVEF (ΔRVEF) was calculated as EF at peak exercise from RVEF. CHD pts were grouped according to RVEF responses to exercise. In the CHD group, FEV1 was reduced (73% predicted) as was Max VO₂ (19.5 ± 3.5 vs 30.5 ± 0.5 CHD vs NML). In the CHD subgroups, Max VO₂ was higher in the pts that increased RVEF compared to those who did not (22.8 ± 17.6 vs 16.5 ± 0.05; p < 0.05). Max VO₂ was then correlated with ΔRVEF and with FEV1 in both subgroups:

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<tr>
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<th>Max VO₂ vs ΔRVEF</th>
<th>Max VO₂ vs FEV1</th>
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<tbody>
<tr>
<td>1RVEFgroup</td>
<td>r = 0.6*</td>
<td>NS</td>
</tr>
<tr>
<td>1RVEFgroup</td>
<td>r = 0.7*</td>
<td>NS</td>
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<tr>
<td>2RVEFgroup</td>
<td>p = 0.05</td>
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Conclusion: Exercise tolerance in pts with congenital heart disease is decreased compared to normals. A more severe reduction in Max VO₂ is seen in pts unable to augment RVEF. At the higher levels of exercise achieved by pts with normal RVEF augmentation, pulmonary factors play an increasingly significant role in limiting Max VO₂. Monitoring of both pulmonary and right ventricular function should be included in the evaluation of pts with congenital heart disease.

741-5 Exercise Induced Angina Is Predictive of High-Risk Thallium Defects in Patients With Non-Q Wave Myocardial Infarction
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Although exercise treadmill test (ETT) is frequently performed for evaluation of patients with non-Q MI (NOMI), the diagnostic value and prognostic significance of ETT data in NOMI is not well established. We prospectively evaluated the information available from ETT parameters in the Veterans Administration Non-Q Wave Infarction Strategies In Hospital (VANWISH) study which has randomized 760 patients with confirmed NOMI to an invasive vs. noninvasive management strategy during the hospitalization phase. Patients randomized to noninvasive strategy underwent ETT using modified Naughton protocol and received thallium scintigraphy. The mean age of the 269 patients with available ETT data was 61 years, 38% had prior MI and 22% had prior CABG. The mean exercise duration was 8.1 ± 3.5 minutes and 63% had exercise duration < 6 minutes. Stepwise regression analyses revealed that older age, higher baseline heart rate and use of calcium blocker were powerful predictors of exercise duration < 6 minutes. A total of 75 (27%) patients had diagnostic ST segment depression (median ST 1 1.8 mm) during ETT and the best predictors for ST ↓ were lateral ECG changes of NOMI and prior CABG. A total of 61 (22%) patients had angina during ETT which was best predicted by prior history of angina, prior CABG, prior MI, and use of antanginal drugs. Exercise-induced angina was highly correlated (P < 0.001) with presence of high risk perfusion abnormalities and multiple areas of defect on thallium scintigraphy.
In summary, although several ETT parameters provide clinically meaningful information, the presence of exercise-induced angina appears to be most predictive of the high risk subset of patients with non-Q wave myocardial infarction.