Nine patients with chronic heart failure (New York Heart Association Function Class 3) were studied using standard methods. Oxygen saturation was measured in the hepatic vein (splanchnic circulation), femoral vein (working muscle) and in mixed venous blood. Measurements were made after 2 minutes at each exercise level and after exercise at 2, 5, 10 and 15 minutes. Results are given as mean values ± SEM.

Three hours after oral Pirbuterol (20mg) the test was repeated. The duration of exercise increased by 34% from 24.9 ± 3.6 min (P < 0.01). Cardiac output was increased at rest by 18% from 3.68 ± 0.2 l/min (P < 0.05) and at peak exercise by 22% from 6.22 ± 0.5 l/min (P < 0.005). Systemic vascular resistance fell at rest by 36% from 1640 ± 90 dynes.s.cm⁻⁵ (P < 0.05) and at peak exercise by 14% from 1050 ± 95 dynes.s.cm⁻⁵ (P < 0.05). Left ventricular filling pressure fell at rest by 11% from 28 ± 2.4 mmHg (P < 0.01) but was unchanged during peak exercise at 35mmHg.

Oxygen saturations in the pulmonary artery before and after Pirbuterol were 59 ± 3% and 61 ± 2% at rest and 21 ± 4% and 27 ± 4% at peak exercise. No significant changes occurred in femoral or hepatic vein saturations during exercise although in the femoral vein an increase was present post exercise (P < 0.05).

Pulmonary artery lactate concentrations were unchanged.

Exercise capacity in these patients was limited by fatigue rather than dyspnoea. Fatigue was associated with maximal oxygen extraction. Oral Pirbuterol increased cardiac output, muscle blood flow and exercise capacity.

**75 THE EFFECT OF BETA-BLOCKER THERAPY ON THE VALUE OF THE ST SEGMENT/HEART RATE RELATIONSHIP IN THE PREDICTION OF SEVERITY OF CORONARY ARTERY DISEASE**


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Exercise tests to predict the presence of severity of coronary heart disease, because of the changes in heart rate, may be affected by beta-blocker therapy. This investigation examined a group of patients with angina on beta-blocking therapy, using a new exercise test (Elamin et al., 1980, Cardiovasc. Res., 14: 681).

The maximal ST/HR slope (the progression of ST segment depression relative to increases in heart rate during exercise) was obtained in 41 patients on beta-blocker therapy who were admitted for coronary arteriography. The maximal ST/HR slopes in two patients with no significant disease were 9 and 10 mm-beats⁻¹.min⁻¹-10⁻³, averaged 18.6 mm-beats⁻¹.min⁻¹-10⁻³ (range 13 - 21) in 5 patients with single-vessel disease (75% luminal narrowing), 44.1 mm-beats⁻¹.min⁻¹-10⁻³ (range 32 - 52) in 22 patients with double-vessel disease and 86.5 mm-beats⁻¹.min⁻¹-10⁻³ (range 69 - 120) in 12 patients with triple-vessel disease. These results are not different from those previously reported in patients without beta-blocker therapy.

In addition in 16 patients, comparison of maximal ST/HR slopes in 16 patients obtained before (50.7 mm-beats⁻¹.min⁻¹-10⁻³; range 11 - 120) and after beta-blockade (51.0 mm-beats⁻¹.min⁻¹-10⁻³; range 9 - 120) showed no statistically significant difference (P > 0.5). In these patients beta-blockade was confirmed by a significant reduction in the elevation of the heart rate/work load relationship (P < 0.05).

Thus the maximal ST/HR slope is not significantly altered by beta-blockade; this exercise test may be used to predict the presence and severity of coronary artery disease in individual patients with angina in the presence of beta-blocker therapy.

**76 EFFECT OF LOWER BODY POSITIVE PRESSURE ON FOREARM AND TOTAL VASCULAR RESISTANCE**

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Roddie et al. showed that passive leg elevation markedly increased central venous pressure, and that this coincided with an increase in forearm blood flow and a fall in forearm vascular resistance. The decrease in forearm vascular resistance was inhibited by local nerve block, and they suggested that the response was mediated via intra-thoracic low pressure receptors [Roddie, Shepherd & Whelan (1957) J.Physiol. 130, 369].

The application of lower body positive pressure (LBPP), in contrast to passive leg elevation, allows a graded increase in central venous pressure. LBPP was applied systematically to 9 normal subjects (mean age 28.8 yrs, 4M, 5F). Pressures of +5, +10, +20, +30 mmHg were applied for 10 min periods, during which forearm blood flow (FBF) was measured using a mercury in rubber strain gauge. Cuff BP was obtained conventionally. A bi-directional continuous-wave Doppler blood velocity meter (Bach-Simpson BPM 202) was used to obtain aortic root blood velocity, and an Ekoline 21 M-mode echocardiograph was used to obtain aortic root diameter. Cardiac output (CO) was calculated from the systolic aortic cross-sectional area and the aortic mean velocity [Darase, Walter & Nupter (1980) Am. J. Cardiol. 46, 613].

FBF increased significantly at each level of inflation, from a control of 2.75 ± 0.37 ml/100g per min to a peak of 3.86 ± 0.74 ml/100g per min at +20 mmHg. BP increased significantly only at pressures of +10 mmHg and above. Forearm vascular resistance reflected the changes in FBF, failing significantly at each inflation pressure, and reaching a nadir at +20 mmHg.

CO increased systematically from a control of 4.9 ± 0.5 1/min to a peak of 6.9 ± 0.7 1/min at +30 mmHg, only reaching significance at +10 mmHg and above. Systemic vascular resistance showed a significant progressive fall at each level of inflation. These data confirm the findings of Roddie et al. They also imply that the low pressure receptors are able to respond to very small pressure changes and thus play an important role in the control of systemic vascular resistance.