ABSTRACTS

ADVANTAGES OF Rb-81 STRESS MYOCARDIAL PERFUSION IMAGING OVER STRESS ELECTROCARDIOGRAPHY.
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To determine the clinical utility of stress Rb-81 myocardial perfusion imaging (SRb), 46 patients (pts) underwent treadmill stress testing with SRb, and selective coronary angiography (SLA). Six studies were performed with a rectilinear scanner and 42 with a scintillation camera and specially constructed lead shield. Positive (+) for significant ischemic disease (SID) were: image defects, coronary stenoses > 75% on SLA, and flat or downsloping ST segment depression of >1mm over baseline on stress electrocardiogram (SLECG). Stress was carried to at least 85% of maximal predicted heart rate or to symptoms. Of 29 pts with +SLECG, only 23 had +SRb, all 23 with +SLECG and 1 had triple vessel disease (TVD). Of 19 pts with -SLECG, 7 had +SRb, 6 with +SLECG. One pt with a documented myocardial infarction also had -SLECG and +SRb but with -SLECG. -SLECG and -SRb in 12 pts were accompanied by -SLECG in 10. TVD was seen in 4 pts, 2 with SRb. Six pts with -SLECG had submaximal stress due to symptoms. +SRb was seen in 5 of the 6 all with +SLECG. The results reveal SRb to be more sensitive and specific than SLECG in detecting SID. +SRb may be seen with significant frequency at submaximal stress and in pts with TVD.

LEFT VENTRICULOGRAPHY DURING ANGINA INDUCED BY EXERCISE AND ATRIAL PACING.
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Stress is important in assessing left ventricular (LV) function in patients with ischemic heart disease (IHD), but it is far from clear which of the two stresses - exercise or atrial pacing provides most meaningful information regarding the functional status of the left ventricle. Twelve patients with IHD (Group A) and 13 patients with normal LV function (Group B) were studied. Left ventriculography was performed during exercise and pacing-induced angina in Group A. Group B underwent exercise and pacing ventriculography with similar exercise work loads and average pacing heart rates which produced angina in Group B respectively. In Group A, at rest the LV end-diastolic volume (LVEDV) was 90 ± 2 ml/m² which increased to 170 ± 4 ml/m² during exercise induced angina (p < 0.001), the LV end systolic volume (LVESV) at rest was 46 ± 4 ml/m² which also increased to 55 ± 3 ml/m² (p < 0.001). Consequently the ejection fraction (EF) did not increase significantly from 50 ± 3% to 54 ± 3% (p NS). Group B showed a decrease in LVEDV (p < 0.01), LVESV (p < 0.01) and an increase in EF (p < 0.001). Thus in IHD the effect of exercise induced angina on LV volumes was opposite to the response achieved in normal subjects, in Group A, during pacing induced angina there were similar directional changes as seen in Group B during pacing i.e. there was a decrease in LVEDV and LVESV (p < 0.05; p < 0.01) during exercise and pacing ventriculography consistently revealed new areas of asynergy while pacing ventriculography failed to produce such changes in four of 12 patients. This study clearly indicates that exercise ventriculography is of more help in assessing LV function and LV wall asynergy than pacing.

THE DURATION OF PROPRANOLOL INDUCED DEPRESSION OF MAXIMAL HEART RATE REMAINS TO INSIGHT--EVIDENCE THAT THE BIOLOGIC HALF-LIFE IS TRIPLE THE SERUM HALF-LIFE.
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Recently considerable attention has been given to the possibility that when propranolol (P) is discontinued, the biological effects of the drug may persist long after the drug is no longer detectable in the serum. In order to assess the effective duration of action of P, the heart rate response to maximal exercise (Max HR) was determined using a Bruce protocol in six healthy male volunteers before P, and following abrupt discontinuation of P (2, 14, 26 and 38 hr. post P) after receiving a steady state dosage of 200 mg. per day. Max HR at control was 188 ± 2.0 (SEM). Two hours after discontinuation of P, Max HR was 138 ± 2.0, at 14 hr. 162 ± 4.5, at 26 hr. 172 ± 2.8 and at 38 hr. 185 ± 2.8. The maximum work load at each level (speed and angle of treadmill) was unchanged for each individual, despite the lessor Max HR response post P. From these data a physiologic half-life of P was determined by subtracting the Max HR at each time interval from control and plotting the return to control Max HR on semi log paper. This value was calculated to be 13.3 ± 1.34 hr. Plasma P determined from blood samples taken at the same time as the exercise stress tests was 4.7 ± 0.6 hr. a value significantly less than the biological half-life (P<0.01). This data confirms our previous findings that the effect of P on resting hemodynamic parameters is significantly longer than can be accounted for by the presence of the compound in the plasma and suggests that when P is discontinued, at least 14 days must lapse until one is certain that the biologic effects of the drug have been effectively dissipated.

LATE RESULTS OF SURGICAL CLOSURE OF LARGE VENTRICULAR SEPTAL DEFECTS IN INFANTS AND CHILDREN.
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Surgical closure of large ventricular septal defects (VSD) has been accomplished successfully in 78 children under 2 years of age presenting with severe pulmonary hypertension, congestive heart failure and failure to thrive. Long term post-operative evaluation of these patients (pts.) has included assessment of growth changes, electrocardiographic (EKG) findings, presence of residual cardiac defects and changes in pulmonary artery pressures and pulmonary vascular resistance (Rp). Sixty-two pts. showed striking growth increases within 1 year of surgery. Serial EKGs on 31 pts. revealed the development of complete heart block over periods up to 16 years. Cardiac catheterizations have been performed in 41 pts. 1 to 8 years after surgery. Small residual VSDs were demonstrated in 16 pts., by oximetry; 8 evident only on selective cineangiocardiograms. With only 1 exception, pulmonary artery pressures and Rp had become normal or were significantly decreased. The marked clinical improvement and absence of complications in the pts. with pulmonary hypertension and elevated Rp preoperatively justifies early surgical closure of large VSDs.