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QUANTITATIVE ANALYSIS OF THE ADMISSION ELECTROCARDIOGRAM PREDICTS EXTENT OF CORONARY DISEASE AND OUTCOME IN REST ANGINA

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We studied 94 pts who were randomized to a trial of aspirin versus heparin in unstable angina and followed for 3 months, to determine the reliability of the emergency room ECG in predicting clinical outcome and extent of coronary disease. ST segment deviation ≥ 1mm was summed over 12 leads (IST), and the total number of leads (NOL) with ST or T wave changes were measured. 62 of the 94 pts had pain during the admission ECG, a comparison ECG before or after pain resolved, and no LVH. Clinical events consisted of recurrent ischemia, infarction, and PTCA or CABG for recurrent pain or compelling anatomy. Considering all 94 pts, an ECG with IST ≥2 mm and NOL ≥2 had a positive (+) predictive value (PV) for clinical events of 79%. An ECG with IST <2 or NOL <2 had a negative (-) PV of 64%. Considering only the 62 pts with pain during the ECG, the + PV improved to 87% and the - PV to 71%. 53 of these 62 pts underwent coronary arteriography. A jeopardy score (JS) ranging from 0-10, was assigned based on the number of vessels with a diameter stenosis ≥ 70% and the location of the stenoses. For example, proximal Cx JS=2, proximal RCA JS=3, proximal LAD JS=5, RCA + LAD JS= 8 etc. There was a significant positive linear correlation between NOL and JS, (r=0.64, SEE=2.4).

Conclusions: In pts with unstable angina, an admission ECG recorded during pain, showing ST segment changes, is by itself a reliable predictor of major clinical events. The total number of ECG leads with ST or T wave changes predicts the extent of myocardium in jeopardy.

NONINVASIVE THROMBUS IMAGING WITH TECHNETIUM 99^m MONOCLONAL ANTIBODY F(ab')$_2$ TO FIBRIN (T2G1s)

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Noninvasive detection of arterial thrombus has been difficult to achieve, but would be of great value in assessing efficacy of thrombolytic therapy and/or reocclusion post MI. We produced occlusive canine thrombi in one femoral artery (n=6) by crush injury and 2 hour temporary ligature occlusion. The opposite artery served as control. After release of the ligatures, 17.4±2 mCi antibody F(ab')$_2$ was injected intravenously and both femoral arteries had serial planar and tomographic imaging for 2 hours. At 2 hours, thrombi were removed, sectioned and well counted. By 2 hours, all thrombi were detected visually by imaging. In vitro, well counting showed thrombus/blood ratios of 5.2/1 and thrombus/left ventricle ratios of 29/1. Two hour blood clearance of F(ab')$_2$ was 54%.

We conclude that in an arterial model simulating clinical acute myocardial infarction (injection of F(ab')$_2$ after thrombus formation), uptake of the *intravenous* MAb is sufficiently rapid to be of value in detecting thrombus presence and the effects of therapy.

Wednesday, March 21, 1990
10:30AM-12:00NOON, Room 36
Isotope Imaging: New Insights and Approaches

REGIONAL ALTERATION IN CARDIAC ADRENERGIC FUNCTION IN INFARCTED AND NON-INFARCTED MYOCARDIUM: NONINVASIVE ASSESSMENT USING I-123 METAIODOBENZYLGUANIDINE.

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We tested the hypotheses that 1) differences in cardiac adrenergic function (CAF) exist between non-infarcted and infarcted myocardium and 2) abnormalities in CAF are more extensive than the associated abnormalities in myocardial perfusion (MP).

MP and CAF were assessed noninvasively following acute myocardial infarction (AMI) in 27 patients using rest Tl-201 and 4 hour I-123 metaiodobenzylguanidine (MIBG) myocardial tomograms. Imaging was performed using a high resolution 3-detector tomograph and ultrapure I-123. MIBG uptake was quantified (counts/pixel/mCi) in the infarct zone (IZ), border zone (BZ) and zone distant (DZ) from the infarct. MIBG uptake in IZ, BZ and DZ was 3.8 ±3.1, 6.0 ±3.0 and 9.2 ±3.5, respectively, p<0.001. Reduction in MIBG uptake was greater following anterior AMI compared with inferior AMI in all zones, 2.6 ±1.9 and 4.6 ±4.7 (IZ), p=0.019; 4.3 ±4.1 and 7.2 ±3.8 (BZ), p=0.018 and 7.3 ±3.2 and 10.5 ±3.1 (DZ), p=0.014. Defect scores reflective of the extent of the MIBG and Tl-201 abnormalities were computed. MIBG defect scores were greater than Tl-201 defect scores 51.9 ±22.0% and 23.3 ±17.7%, respectively, p<0.001.

Therefore, altered CAF occurs in both infarcted and non-infarcted myocardium and is more severe following anterior AMI. In addition, abnormalities of CAF are more extensive than the associated abnormalities of MP.

IN VIVO IMAGING OF ACUTE ARTERIAL THROMBI WITH TECHNETIUM-99M ANTI-PLATELET MONOCLONAL ANTIBODY.

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Membrane antigens which may be specifically targeted for the localization of thrombi are expressed when platelet granule contents are secreted. Technetium-99m (Tc-99m) labeled S12 Fab' monoclonal antibody is directed against the human alpha-granule GMP140 membrane antigen expressed during platelet activation. Tc-99m S12 Fab' was administered to rabbits with acute arterial thrombi formed by placement of embolization coils in the abdominal aorta. Fifteen minutes later, Tc-99m S12 Fab' (3.5 mCi/0.3 mg) was injected intravenously. Nonspecific uptake was evaluated by coinjecting an indium-111 labeled isotype-matched, nonspecific monoclonal antibody (OVTL-3) Fab-DTPA (1.5 mCi/0.3 mg). Serial scintigrams were acquired every hour for 5 hours (n=4), at which time thrombi were excised for ex vivo counting. Tc-99m S12 Fab' thrombus-to-blood (T:B) and thrombus-to-muscle (T:M) ratios for activity (cpm/g) were 75.6 ± 42.2 and 882.4 ± 559.3 respectively, which were 95 times (p<0.01) and 38 times (p<0.01) greater than the uptake for the nonspecific Fab. These results demonstrate that Tc-99m S12 Fab' specifically binds to activated platelets in vivo and allows rapid scintigraphic detection of arterial thrombi.