IMPACT OF SCREENING PATIENTS IN A HEART EMERGENCY ROOM ON THE CCU POPULATION

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In 1986 a fully equipped 24-hour a day out-patient facility "the Heart Emergency Room (ER)" was opened with the aim to give acute cardiac care and to screen patients presented with acute cardiac problems. Patients could be observed for a maximum of 24 hours and laboratory, ECG, X-ray and echocardiography facilities were available. The Heart-ER had important effect on the CCU population and CCU discharge diagnosis and workload.

CCU diagnosis(%)	pre Heart-ER		post Heart-ER		
years	'83	'84	'87	'88	'89
# CCU pts	639	716	958	883	824
# Heart-ER pts	-	-	2346	2381	2604
unstable angina (%)	20	19	21	26	26
heart infarction (%)	27	34	38	39	38
arrhythmia (%)	17	13	11	10	10
heart failure (%)	6	6	13	12	13
stable angina(%)	2	2	3	2	3
non cardiac (%)	28	26	13	11	10

Conclusion: Screening and treatment in the Heart-ER resulted in a 53% decrease of patients with non-acute cardiac disease and a 23% increase of more ill patients with acute cardiac disease in the CCU. The use of the sometimes scarce CCU beds was optimalized, but the total workload of nurses and CCU staff increased significantly.

REPERFUSION THERAPY IMPROVES INFARCT ZONE WALL MOTION IN INFERIOR AS WELL AS ANTERIOR WALL MI

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Previous studies have suggested that pts with acute inferior MI derive less benefit from reperfusion (by thrombolytic therapy or PTCA) than patients with anterior MI. To test this hypothesis, we evaluated LVEF and infarct zone wall motion (IZWM) in 108 consecutive patients enrolled in ongoing thrombolysis protocols in which lytic therapy was initiated within 6 hours of MI symptom onset. Mean time from MI symptom onset to lytic Rx was similar in the anterior (n = 38) and inferior (n =70) MI group, 187±89 vs 183±65 min, respectively, p=NS. Lytic Rx was successful (TIMI 2 or 3) in 71% (50/70) of inferior (IMI) and 79% (30/38) of anterior MI's (AMI), p=NS. Salvage PTCA following failed lytic Rx was successful in 8/8 of AMI and 15/16 of IMI pts in whom it was attempted. Left ventricular ejection fraction (LVEF) and IZWM by the centerline method were assessed from LV angiograms obtained 90 minutes (acute) after lytic therapy and at 7-10 days of follow-up (f/u). Acute and f/u LVEF were not significantly different for the entire group (.54±.11 vs .55±.12, p=NS), for AMI (.48 ± .12 vs .50± .12, p=NS), or for IMI (.56+/-.09 vs .57 +/- .11, p=NS) . In contrast, IZWM was improved for the entire group (-2.8 ± 1.3 vs -2.2 ± 1.5 SD/chord, p<.05), AMI (-3.4 ± 1.5 vs -2.6 ± 1.6, p<.05), and IMI (-2.5 ± 1.2 vs -2.0 ± 1.4, p<.05). Thus, although global function (LVEF) was unchanged, improvement in IZWM for both groups supports the use of reperfusion therapy in inferior as well as anterior MI.

POLYMORPHONUCLEAR LEUCOCYTES ARE ACTIVATED DURING STREPTOKINASE TREATED ACUTE MYOCARDIAL INFARCTION

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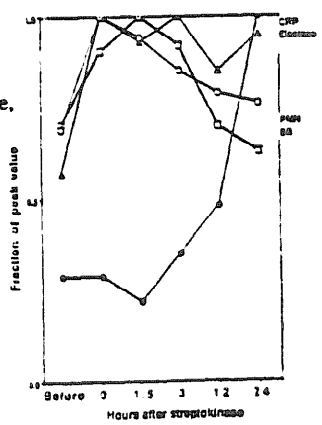
Background: Animal experiments have demonstrated that fibrinolysis is associated with activation of polymorphonuclear leucocytes (PMN) with release of elastase (E) and its catalytic product fibrin split product Bß 30-43 (Bß). These events can influence reperfusion damage.
Method: Thirty patients (63±10 yrs) with streptokinase treated AMI were investigated. PMN, E, Bß and C-reactive protein (CRP) were determined before administration of streptokinase and 0, 1.5, 3, 12 and 24 hrs after completed administration.
Results: PMN, E, Bß and CRP were increased before streptokinase, 3.9±0.6 hrs after onset of symptoms. No relation was seen to duration of symptoms. PMN, E and Bß reached peak levels 0-1.5 hrs after completed streptokinase treatment (Figure). CRP increased only after 12-24 hrs.
Conclusion: PMN are activated early during acute myocardial infarction. This activation is accentuated by streptokinase and occurs earlier than for CRP, that appears to react independently of streptokinase administration.



PROGNOSTIC IMPORTANCE OF DELAYED Q-WAVE EVOLUTION 3 TO 24 HOURS AFTER THROMBOLYTIC THERAPY FOR ACUTE MYOCARDIAL INFARCTION.

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The timing of Q-wave evolution and its prognostic significance was studied in 201 patients (pts) who received thrombolytic therapy for a first acute myocardial infarction (AMI). 141 pts (70%) had evidence of Q-wave AMI within 3 hours of the initiation of thrombolytic therapy, 31 (15%) developed Q-waves after 3 hours, but before discharge, and 29 (14%) were discharged with non-Q-wave AMI. Laboratory indicators of myocardial damage and in-hospital morbidity and mortality were greater among pts with Q-wave AMI than pts with non-Q-wave infarctions. When these indices were examined with respect to timing of Q-wave evolution, the prognosis of pts with delayed Q-wave development was similar to that of pts with non-Q-wave AMI. Thus, when compared to pts with early (< 3 hour) Q-wave evolution, pts with later or non-Q-wave AMI had smaller CK peak (mean 661-1081 vs 125[illegible]1541 IU, p=0.005), better preservation of left ventricular function as measured by radionuclide ventriculography before discharge (54%±11% vs 47±13%, p <0.01), and lower incidence of congestive heart failure at discharge (3% vs 15%, p=0.02). In-hospital mortality was lower among pts with late Q-wave evolution or non-Q-wave AMI (5/141 vs 0/60, p=N.S.). It is possible that delayed (> 3 hours) evolution of Q-wave AMI among pts receiving thrombolytic therapy may represent the higher prevalence of pts with incomplete AMI in this population.