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INVERSE CORRELATIONS BETWEEN PLASMA BETA-THROMBOGLOBULIN LEVEL AND: (1) TIME FROM ONSET OF ACUTE MYOCARDIAL INFARCTION; (2) MEAN TIME TO CLOT LYSIS FOLLOWING SYSTEMIC STREPTOKINASE.

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The platelet specific protein, beta-thromboglobulin (β-TG) is released into the circulation when blood platelets undergo the release reaction as in platelet aggregation and clotting. This paper describes a study of a cohort of patients (N = 40) in which β-TG was serially assayed by a radioimmunassay technique at various times from the onset of acute myocardial infarction.

It shows a negative correlation (R = -0.76, p = 0.01) between initial β-TG level and time elapsed since the onset of symptoms. Furthermore, patients fell into two groups - those with high initial or rising levels, and those in whom the levels were not elevated and did not rise. Both groups underwent thrombolytic therapy with intravenous streptokinase, the former manifesting significantly earlier ECG evidence of re-perfusion. Respective mean lysis times and standard errors of the mean (hours) were 0.798 (0.139) and 3.490 (0.498) with p = 0.001 on t-testing. Clearly when the clotting process is well established it is much more difficult to lyse the clot. β-TG is a marker for the age of the clot in that it reflects the platelet secretion in vivo that accompanies the early stages of clot formation.

We conclude that β-TG assay is not purely an esoteric research technique but has everyday clinical application in differentiating between early streptokinase-induces lysis, when some at-risk myocardium is still viable, and later spontaneous recanalization. Angiography cannot make this distinction.

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PLATELET FUNCTION AND PLASMA FIBRINOGEN IN YOUNG SURVIVORS OF MYOCARDIAL INFARCTION.

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An increased liability for thrombosis might be of pathogenetic importance in young survivors of myocardial infarction (MI). In 73 (58 men and 15 women) patients with MI below 45 years of age and 73 matched healthy controls plasma fibrinogen and platelet function tests were studied 3-6 months after the MI. At the time of the MI 77% of the patients were smokers but at the time of the investigation 27% of the patients smoked compared to 37% of the controls. Platelet aggregability was measured in vitro in platelet-rich plasma (PRP) as maximal aggregation to ADP and collagen. The platelet sensitivity to the inhibitory effect of prostacyclin (PGI2) was tested by preincubation of PRP with PGI2 before inducing aggregation with ADP 5 μM. Plasma levels of beta-thromboglobulin (BTG) and platelet factor 4 (PF4) were measured by RIA methods and plasma fibrinogen by heat precipitation. The table presents the results (means ± SE). * is p<0.04, ** is p<0.02 and ns is non significant.

	patients	controls	
ADP 1.0 uM	39.2 ± 2.3	35.6 ± 1.7	ns
Collagen 1.0 mg/l	64.7 ± 3.6	65.7 ± 3.4	ns
PGI2 0.5 ng/ml % inhib	21.6 ± 2.5	29.5 ± 2.0	**
PGI2 1.0 ng/ml % inhib	48.5 ± 2.9	55.9 ± 2.1	*
BTG ng/ml	26.4 ± 1.4	26.5 ± 1.6	ns
PF4 ng/ml	3.32 ± 0.39	3.05 ± 0.32	ns
Fibrinogen mmol/l	4.25 ± 0.13	3.95 ± 0.08	*

Severe emotional stress preceeding the MI occured in 7 patients - these cases had an increased platelet reactivity to ADP. The fibrinogen level was also elevated by smoking and obesity (multivariate analysis). Conclusion: young MI patients have elevated levels of fibrinogen and reduced platelet sensitivity to PGI2. This might cause an increased thrombotic tendency.

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NEUTROPHIL ELASTASE IS A MARKER OF NEUTROPHIL ACTIVATION IN ACUTE MYOCARDIAL INFARCTION.

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Elastase is released from human neutrophils as a result of active secretion, phagocytosis or cell lysis. It is a broad-spectrum protease which not only hydrolyses the major components of tissue matrix, but can also affect platelet aggregation, coagulation and fibrinolysis. Neutrophils localise at the site of myocardial infarction and have been implicated in subsequent cell damage. A radioimmunoassay was developed which detects elastase complexed to its inhibitors α_1- antitrypsin and α_2-macroglobulin as well as the free enzyme. Plasma concentrations of elastase in 31 healthy controls were 21.3 ± 13.8 ng/ml, and did not differ significantly from those in 22 patients with stable angina (23.6 ± 8.6 ng/nl). In 19 patients with myocardial infarction, however, plasma elastase levels rose to a peak within 48 hours of infarct which was significantly higher than normal levels (95.1 ± 83.7; P< 0.001), and then declined. Correction of the data for neutrophil count did not affect the significance of the observed differences. Measurement of whole blood elastase reflected the neutrophil count and was not otherwise informative. Thus, neutrophils are activated after myocardial infarction and release sufficient elastase for it to be detected systemically. This may extend tissue damage and affect coagulation and fibrinolysis at the site of infarction.

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BLOOD TAURINE AFTER MYOCARDIAL INFARCTION.

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Taurine, an amino acid that is present in high concentrations in the heart, is released from the heart after myocardial damage. There is evidence that the concentration of taurine in whole blood is raised after myocardial infarction (MI), and it has been suggested that blood taurine may be a measure of the degree of infarction. We have obtained serial measurements of blood taurine in patients admitted to a coronary care unit and have compared the results with those obtained for two cardiac enzymes (AST and HBD) and other blood parameters.

The patients were divided into two groups: those for whom there was a peak of AST activity (> 40 i.u./l) (Group 1, n = 24) and those for whom AST and HBD was not raised (Group 2, n = 15). For Group 1 patients, mean results were obtained for each of the parameters for the day on which AST peaked (designated Day 0) and for preceeding and subsequent days. For Group 2 a single mean was obtained. Results marked * in the table differ significantly (p < 0.05 or lower) from those for Group 2:

Group 1:	Day -2 n = 4	Day -1 n = 19	Day 0 n = 24	Day +1 n = 24	Day +2 n = 20	Days 3-6 n = 9	Group 2 n = 15
AST (i.u./l)	30	*116	*244	*143	* 81	38	21
HBD (i.u./l)	194	*329	*680	*691	*536	*393	143
Taur (umol/l)	*302	*299	*329	*279	238	219	236
Neut (10^9/l)	*8.4	*10.1	*11.0	*8.8	*7.3	5.9	5.3

It can be seen that blood taurine was significantly raised after MI and followed a pattern similar to the neutrophil count. Furthermore, several positive correlations (r = 0.63-0.79) were obtained between taurine and neutrophil count in both groups, but not between taurine and AST or HBD.

In another investigation we measured the amounts of taurine in neutrophils, platelets and plasma from patients with MI (n = 5) and controls (n = 9). We found no differences in the amounts present per neutrophil, per platelet or per ml of plasma.

Our data suggest that the increased level of taurine in blood after MI merely reflects the increased number of neutrophils present in blood following the event.