4:30

804-3 A Double Blind Randomized Trial of a 24 Hour Infusion of Magnesium Sulphate in Unstable Angina

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Magnesium (Mg^{2+}) has been shown to reduce coronary spasm, have favorable hemodymamic effects, inhibit platelet function, reduce arrhythmias, modulate autonomic function, reduce catecholamine secretion and to be protective during experimental acute myocardial ischemia.

Sixty-two consecutive patients with unstable angina (rest pain < 24 hours, ST/T changes and normal admission CK) were randomized in a double-blind fashion to Mg^{2+} (8 mmol bolus, then 3.0 mmol/h for 24 hours) or placebo within 12 hours of admission. All patients received aspirin, β blockade, iv nitrates and heparin, provided no contraindications existed. Four consecutive 12 hour urine collections were started at study entry for estimation of catecholamines (n = 31) or β thromboglobulin (n = 31).

84% underwent predischarge angiography. Baseline characteristics and extent of coronary disease were similar in both groups. Serum Mg^{2+} rose from 0.91 ± 0.02 to 1.71 ± 0.05 mmol/l (mean ± SEM) in the Mg^{2+} group. CKMB release was less in the Mg^{2+} group at 6 and 24 hours (28.3 ± 7.4 vs 6.6 ± 1.2 ng/ml at 6 hrs, $p < 0.05$). Regression of T wave changes on the 24 hour ECGs (compared with admission ECGs) occurred more frequently in the Mg^{2+} group (11 patients vs 0 patients, $p < 0.005$). On 48 hour Holter monitoring, a similar proportion had transient myocardial ischemia (TMI) in the two groups (16 (52%) in the Mg^{2+} group vs 15 (48%) in the placebo group), however there were fewer episodes of TMI in the Mg^{2+} group (57 vs 118, $p < 0.0001$) with a median total duration of 31.45 vs 102.73 mins respectively (p = ns). Frequency of ventricular ectopy and non-sustained ventricular tachycardia were similar in both groups. Heart rate variability was significantly lower in the placebo group in the 24 hours following, but not during, the infusion. Epinephrine excretion was lower in the Mg^{2+} group in the first 12 hour sample (1.05 ± 0.20 vs 1.61 ± 0.41 ng/mmol creat., $p < 0.05$), with no significant difference in norepinephrine excretion. Urinary β thromboglobulin was similar in both groups in all collections.

Thus Mg^{2+} appears to reduce CKMB release, ECG progression and catecholamines in the acute phase of unstable angina with no effect on β thromboglobulin excretion suggesting that its effect may be mediated via autonomic modulation but not platelet function. A larger scale trial investigating the effect of Mg^{2+} on mortality is warranted.

4:45

804-4 A Randomized, Double-blind Trial of Streptokinase Versus Placebo for the Management of Unstable Angina and Non-Q-wave Myocardial Infarction in Patients with Previous Coronary Artery Bypass Surgery

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This trial investigated the potential usefulness of thrombolysis in patients with previous coronary artery bypass surgery admired for unstable angina and non-Q-wave myocardial infarction, the rationale being more frequent non-Q-wave infarction in these patients and a high-fibrin content of older grafts. A total of 125 consecutive patients with symptoms evolving for less than 24 hr were randomized to intravenous streptokinase 1.5 million Units in 45 min, or to placebo. All patients received concomitant intravenous heparin, oral aspirin and standard anti-ischemic therapy. Mean age (62 ± 9 yrs), sex (78% male), time of previous surgery (8 ± 0.5 yrs), presence of a previous myocardial infarction (63%), prior medication including aspirin in 66%, an abnormal ECG at admission (79%) and time to treatment after the last ischemic episode (10 ± 9 hr) were similar in the 2 study groups.

Fatal and non-fatal myocardial infarctions in hospital occurred in 7 streptokinase (11.1%) and 3 placebo (4.8%) patients (relative risk 2.3, 95% confidence limits 0.62–8.48, p = 0.19) and at 1 month in an additional 2 patients in each group. Refractory angina in hospital was observed in 12 (19.1%) streptokinase patients and 13 (20.9%) placebo patients. A revascularization procedure because of recurrent symptoms was required at 1 month in respectively 22 (34.9%) and 16 (25.8%) patients.

Conclusion: The early cardiac event rate in unstable angina associated with previous bypass surgery is high. The nonsignificant excess in the risk of myocardial infarction with streptokinase in this population most likely to benefit, is similar to the excess reported in other populations of patients with unstable angina and non-Q-wave myocardial infarction and supports the lack of benefit of thrombolysis in unstable angina.

805 New Approaches in Emergency Resuscitation

Wednesday, March 22, 1995, 4:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Room 26

4:00

805-1 Theophylline Reverses Myocardial Infarction-Related Brady and Tachy Arrhythmias: Role of Adenosine

Barry D. Bertolet, James A. Hill, Luiz Belardinelli. *University of Florida, Gainesville, FL*

Brady and tachyarrhythmias often complicate the early management of acute myocardial infarction (MI). Endogenously released adenosine (ADO) from ischemic cardiac cells has been proposed as a potential mediator of these arrhythmias. In order to test this hypothesis, theophylline (THEO), a competitive ADO receptor antagonist, was administered to nine patients who developed sustained or hemodynamically significant brady or tachyarrhythmias immediately following an acute inferior MI.

Methods: Once such an arrhythmia was detected, continuous ECG monitoring was begun. THEO was then administered i.v. at a rate of 100 mg/min until the arrhythmia resolved or a maximum of 250 mg THEO was infused. Patients were then monitored for 24 hours for recurrent arrhythmias.

Results: Bradyarrhythmias were detected in 5 patients (3 with 3° AV block, 2 with 2° AV block). Tachyarrhythmias were detected in four patients (2 with atrial fibrillation, 2 with accelerated idioventricular rhythm). All patients converted to normal sinus rhythm within five minutes of the administration of THEO (178 ± 57 mg). No recurrent arrhythmia occurred in the follow-up period.

Conclusions: Many of the brady and tachyarrhythmias which occur early after inferior MI are ADO-mediated. ADO receptor antagonism appears effective in converting these arrhythmias to normal sinus rhythm and may be considered as primary therapy.

4:15

805-2 Faster but not more Complete Thrombolysis with Tissue Plasminogen Activator than with Streptokinase in Massive Pulmonary Embolism

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Mean pulmonary artery pressure (MPA), cardiac output (CO), and right ventricular ejection fraction (RVEF) were serially assessed with use of a 5-way, rapid response rate, Swan-Ganz catheter in 45 Pts submitted to thrombolytic therapy (TT) for massive pulmonary embolism (PE) defined as Miller score < 17/34, and mean pulmonary artery pressure >20 mmHg. Total pulmonary resistance (TPR) was determined from MPA and CO and expressed in dynes/sec/cm^{-5}. Pulmonary angiography was performed before and 24 h after TT. Streptokinase (STK) was used in 22 Pts, and tissue plasminogen activator (t-PA) in 23.

	T 0	T 1 h	T 2 h	T 12 h
TPR-STK	701 ± 261	591 ± 203	583 ± 211	380 ± 155
TPR-t-PA	735 ± 390	508 ± 330	405 ± 210*	338 ± 142
RVEF-STK	0.18 ± 0.02	0.20 ± 0.07	0.20 ± 0.09	0.26 ± 0.10
RVEF-t-PA	0.18 ± 0.08	0.24 ± 0.09	0.27 ± 0.09*	0.29 ± 0.09

*p < 0.05 between STK and t-PA

A significant decrease in TPR inversely correlated to a significant increase in RVEF occurred in both treatment groups, but significantly more rapidly in the t-PA group than in the STK group. The difference was significant 2 h and 4 h after onset of TT, but had vanished at 12 h. Miller score, similar in both groups before TT, was identical in both groups at 24 h.

Conclusion: t-PA induced a significantly faster hemodynamic improvement than STK in massive PE, but with a catch-up phenomenon apparent 12 h after onset of therapy.

4:30

805-3 Diagnostic Value of Transesophageal Echocardiography During Cardiopulmonary Resuscitation

Poll A. van der Wouw, Rudolph W. Koster, Ben J. Delemarre, Rien de Vos, Dick van Groeningen *Academic Medical Center, Amsterdam, the Netherlands*

Transesophageal echocardiography (TEE) permits the visualisation of the heart and great vessels without disturbing resuscitation attempts. Early information about the cause of the circulatory arrest can help in reestablishing