

Results: Complete data sets were available for 58 defibrillators; 3 had been replaced at follow-up. The mean (\pm SD) time difference between defibrillators and the atomic clock after 3 months was 1.50 ± 1.66 min (range 0 - 10 min). For each defibrillator model, the mean time difference after 3 months was 0.71 ± 0.63 min (Lifepack 9, N = 34), 1.60 ± 1.34 min (Lifepack 10, N = 5), 2.17 ± 1.33 min (Heartstream, N = 6), 3.33 ± 0.52 min (Lifepack 12, N = 6), 4.25 ± 4.03 min (HP Codemaster, N = 4), 2.00 ± 1.41 min (Zoll M, N=2) and 1.00 min (Lifepack 7, N=1). The Lifepack 9 defibrillator clocks had significantly less time change over 3 months than Heartstream, Lifepack 12 and HP Codemaster defibrillator clocks ($P < .0003$).

Conclusion: Defibrillator clocks are unable to maintain accurate time over 3 months. Without extra measures defibrillator clocks are too inaccurate to be used to assess collapse-to-first shock time interval.

POSTER SESSION

1028 Combined Procedure Outcomes in Coronary Artery Bypass Patients

Sunday, March 30, 2003, Noon-2:00 p.m.

McCormick Place, Hall A

Presentation Hour: Noon-1:00 p.m.

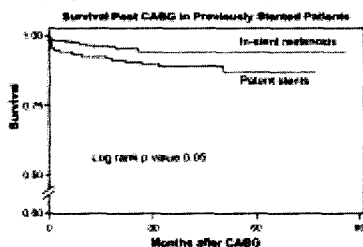
1028-96

Higher Mortality in Patients With Patent Stented Arteries Undergoing Coronary Artery Bypass Surgery

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Aim: Current surgical practice is to bypass a previously stented artery even if it is patent. Does bypassing a patent stented artery affect outcome? We sought to compare outcomes of patients with patent stents (<50% stenosis) with those with in-stent restenosis (>50% stenosis) undergoing coronary artery bypass grafting (CABG) to the stented arteries.

Methods: From 1995 through 2001, 359 patients had coronary stents and CABG at our institution. Of these, 162 had patent stents and 197 had stents with in-stent restenosis that were bypassed. Patients in the 2 groups were similar with respect to age, left ventricular function, extent of coronary disease, time from stent to CABG and prevalence of diabetes, smoking, hypertension and renal insufficiency. Survival data was determined using Social Security Death Index. **Results:** At a mean follow up of 42 ± 18 months, there were 18 deaths in patients with patent stents and 11 patients with in-stent restenosis undergoing CABG. Kaplan-Meier analysis is shown in the figure ($p=0.05$).



Conclusions:

Patients with patent stents undergoing CABG to the stented vessel had a higher mortality than did similar patients with in-stent restenosis.

1028-97

Minimally Invasive Coronary Bypass Grafting of the Left Anterior Descending Coronary Artery Improves Myocardial Perfusion in the Nonrevascularized Second Vessel Territory in Two-Vessel Coronary Artery Disease

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Background: Although coronary bypass grafting and coronary angioplasty (PTCA) are equally effective to achieve target vessel revascularization in multivessel coronary artery disease (CAD), little is known on the long-term impact of minimally invasive coronary bypass grafting (MIDCAB) on myocardial perfusion in two-vessel CAD.

Methods: Thirty-one patients (age 63 ± 10 yrs) with two-vessel CAD of the left anterior descending coronary artery (LAD) and the left circumflex ($n=18$) or right coronary artery ($n=13$) were studied by stress/rest perfusion SPECT. Perfusion was scored in a 18-segment model from 1 (normal) to 4 (absent uptake); percent extension and severity of perfusion defects were evaluated in the territory of the LAD and of the stenotic 2nd vessel. Quality of life was assessed by the SF-36 questionnaire. All patients underwent MIDCAB of the LAD, alone in 12 cases (Gr 1), or coupled to PTCA and stenting of the 2nd vessel in 19 (Gr 2). Clinical follow-up, SF-36 and SPECT were repeated 12 months after surgery.

Results: Baseline variables were not different in the 2 groups. During 12-month follow-up, no cardiac events occurred and significant improvement was observed versus baseline. SF-36 score increased from 102 ± 23 to 131 ± 12 in Gr 1 ($p=0.004$) and from 93 ± 21 to 131 ± 13 in Gr 2 ($p=0.0001$). Stress perfusion defects extension in the LAD territory

decreased from $77 \pm 20\%$ to $30 \pm 37\%$ in Gr 1 ($p=0.003$) and from $68 \pm 29\%$ to $13 \pm 19\%$ in Gr 2 ($p=0.0001$). Stress perfusion defects extension in the 2nd vessel territory decreased from $48 \pm 17\%$ to $20 \pm 23\%$ in Gr 1 ($p=0.005$) and from $55 \pm 18\%$ to $21 \pm 21\%$ in Gr 2 ($p=0.0004$). Severity score decreased from 2.02 ± 0.75 to 1.53 ± 0.70 in Gr 1 ($p=0.002$) and from 2.17 ± 0.55 to 1.17 ± 0.24 in Gr 2 ($p=0.0001$) for the LAD territory. It decreased from 1.96 ± 0.38 to 1.35 ± 0.49 in Gr 1 ($p=0.002$) and from 2.02 ± 0.50 to 1.31 ± 0.53 in Gr 2 ($p=0.0005$) for the 2nd vessel territory. No between-group difference at 12 months was significant.

Conclusion: In patients with two-vessel CAD, at 12 months after MIDCAB of the LAD, myocardial perfusion shows a similar sustained improvement both in the LAD and in the 2nd vessel territories, irrespective of revascularization by PTCA.

1028-98

Is Endoaneurysmorrhaphy Superior to Linear Suture for Left Ventricular Aneurysm Repair?

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Objectives: Endoaneurysmorrhaphy (EA) has been proposed as a more physiologic repair of postinfarction left ventricular aneurysm (LVA) than is linear repair. The aim of the present study was to evaluate early and late results of 2 different surgical techniques for the LVA repair (EA versus linear closure (LC)).

Methods: Between November 1993 and November 2001, 112 patients (pts), 90 males (80%), underwent surgical repair for LVA. EA (Group A) was employed in 69 pts (61.6%) and LC (Group B) in 44 patients (38.4%). Clinical outcomes and echocardiographic measurements of Group A were compared with those in Group B.

Results: Mean follow-up time is 44.5 ± 33.1 months. Preoperatively NYHA class, left ventricular (LV) ejection fraction (EF) and LV end-diastolic diameter were similar. Early overall mortality rate was 4.4%. Early postoperative increase in LVEF was greater after EA (4.6 ± 7.3 vs 0.6 ± 6.1 , $p=0.015$). Functional status improvement was significant for all the pts despite the different repair technique used ($p=0.0001$) as perhaps the LVEF increase ($p=0.0002$). There were no differences between the 2 groups for the NYHA class (change in NYHA class -1.3 ± 0.9 in Group A and -1.2 ± 0.8 in Group B, $p=0.479$) and for the LVEF (37.2 ± 8.7 vs 37.9 ± 9.3 , $p=0.721$). The actuarial survival rate at 5 years were $78 \pm 7\%$ for Group A and $85 \pm 7\%$ for Group B ($p=0.479$). Comparison with LogRank test revealed no significant difference in cardiac death between 2 groups ($p=0.148$). A preoperative NYHA class $>II$ ($p=0.018$) and a LVEF $<35\%$ ($p=0.005$) were associated with a higher incidence of early and late mortality.

Conclusions: LVA repair is an important therapeutic intervention. Both EA and LC appear extremely safe and efficacy showing a low operative mortality. Long term outcome resulted substantially similar for both repair methods. The surgical "combined" approach permits a better outcome for pts undergoing LVR.

POSTER SESSION

1048 Inflammation and Other Novel Biomarkers in Acute Coronary Syndromes

Sunday, March 30, 2003, 3:00 p.m.-5:00 p.m.

McCormick Place, Hall A

Presentation Hour: 3:00 p.m.-4:00 p.m.

1048-89

Multiple Complex Unstable Plaques Associated With Systemic Inflammation and Adverse Cardiovascular Outcomes

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Background: Recent observations demonstrate that patients with acute coronary syndrome (ACS) often harbor multiple unstable plaques, consistent with the concept that destabilizing processes such as inflammation may exert adverse influence throughout the coronary bed. This study was designed to determine whether there is a relationship between the presence and magnitude of systemic inflammation, the number of unstable complex lesions (CL) and adverse cardiovascular events. **Methods:** In 283 patients with ACS undergoing coronary angiography, we analyzed the association between the number of CL, systemic marker of inflammation (high sensitivity C-reactive protein, CRP) and major adverse cardiovascular events (MACE) at one year (cardiac death, non-fatal MI and revascularization). Univariate and multivariate analysis were performed. **Results:** A single CL was present in 32% of pts, multiple CL were identified in 23% and no CL were documented in 45% of cases. There was a direct association between increasing number of CL and increments in systemic marker of inflammation (in pts. with none, one and multiple CL, CRP = 0.22 , 0.53 and 1.85 mg/d, $p < 0.0001$). On multivariate analysis, CRP was independently associated with the presence of multiple complex coronary lesions ($p < 0.0001$). At one year follow up, major adverse cardiac events were reported in 24% of cases (death in 5.7%, MI in 3.9%, and revascularization in 17.7%). On multivariate analysis, only the presence of multiple CL (HR 2.88, $p=0.0007$) and history of congestive heart failure (HR 2.57, $p=0.037$) were independent predictors of MACE at 1 year.

Conclusions: These findings demonstrate a strong association between the presence of multiple complex unstable plaques, the magnitude of systemic inflammation and adverse clinical outcome.