3 groups based on receipt of cardiac catheterization (cath) < 12 hours of lytics (early cath, N=261), ≥ 12 hrs after lytics (late cath, N=851) or no cath (conservative treatment, N=1256). The likelihood of in-hospital mortality and reinfarction was determined using multivariate analysis.

Results: 41% pts underwent cath during index hospitalization. 69% of early cath pts had a PCI (percutaneous coronary intervention) as opposed to 61% of the late cath group (p < 0.0001). The outcomes of the early cath group did not differ significantly from the lytics only group. However, the incidence of in-hospital death was significantly lower in the late cath group as compared to the lytics only group (2% Versus 6.8%, p = 0.0002). This remained true even after controlling for differences in baseline and in-hospital characteristics between the 2 groups (late cath mortality OR 0.3, 95%CI 0.13-0.63). The likelihood of a reinfarction was, however, significantly high in the late cath group.

Conclusions: Although lysults substantially reduce mortality after an acute STE MI many pts may derive an additional benefit from the use of a cardiac cath and, if indicated, a PCI, especially if performed non-urgently.

### Table: Odds of death and reinfarction during index hospitalization

<table>
<thead>
<tr>
<th></th>
<th>DEATH</th>
<th>DEATH</th>
<th>REINFARCTION</th>
<th>REINFARCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95%CI</td>
<td>OR</td>
<td>95%CI</td>
</tr>
<tr>
<td>Late Cath</td>
<td>0.3</td>
<td>0.13-0.63</td>
<td>3.1</td>
<td>1.78-5.23</td>
</tr>
<tr>
<td>Cath</td>
<td>0.5</td>
<td>0.27-0.79</td>
<td>2.5</td>
<td>1.49-4.25</td>
</tr>
</tbody>
</table>

### Oral Contributions

#### 822 Cardiac and Systemic Markers as Predictors of Risk in Acute Coronary Syndromes

**Monday, March 18, 2002, 2:00 p.m. - 3:30 p.m.**

**Georgia World Congress Center, Room 264W**

**2:00 p.m.**

**822-1 Elevated White Blood Cell Count Is Associated With Higher Mortality in Patients With Acute Coronary Syndrome Independent of Other Cardiac Markers: A TACTICS-TIMI 18 Substudy**

Marc S. Sabatine, David A. Morrow, Sabina A. Murphy, Lauren A. Demopoulos, Peter M. DiBattiste, Carolyn H. McCabe, Christopher P. Cannon, Eugene Braunwald, Michael Gibson, Brigham and Women's Hospital, Boston, Massachusetts.

**Background & Methods:** Higher white blood cell counts (WBCC) have been associated with higher mortality in the setting of acute coronary syndromes (ACS). We sought to examine if this relationship was true independent of other cardiac markers of inflammation.

**Results:** In our study, patients with higher WBCC had a worse outcome, even when adjusting for other cardiac markers. The WBCC remained a significant predictor of mortality in this study.

**Conclusion:** WBCC is an independent predictor of mortality in patients with acute coronary syndromes. This finding highlights the importance of early intervention to reduce WBCC levels in this population.

**822-2 Elevated C Reactive Protein (CRP) Is Associated With a Higher Risk of Thrombotic Occlusion and Poorer Flow Following Percutaneous Coronary Intervention in Patients With Acute Coronary Syndromes: A TACTICS TIMI 18 Angiographic Study**

Michael Gibson, David Morrow, Ralph Vicari, Graham Wong, Laura Demopoulos, Peter DiBattiste, Sabina A. Murphy, Christopher P. Cannon, Eugene Braunwald, Harvard Clinical Research Institute, Boston, Massachusetts, Brigham and Women’s Hospital, Boston, Massachusetts.

**Background & Methods:** Higher CRP levels are associated with a higher risk of cardiac events. The relationship between baseline CRP and angiographic findings has not been explored. In the TACTICS TIMI 18 trial of invasive versus conservative strategies in the management of ACS, we hypothesized that elevations in CRP would be associated with unfavorable angiographic findings such as a higher risk of thrombotic occlusion.

**Results:** Patients with a closed artery (TIMI Flow Grade < 2) had higher CRPs than patients with an open artery (TIMI Flow 2/3). CRP > 3 mg/dL was associated with a higher risk of thrombotic occlusion (26.8%, 155/592 vs. 12.0%, 48/399, p = 0.003). Likewise, an elevated CRP > 2X threshold was associated with higher (i.e. slower) Corrected TIMI Frame Counts (CTFCs) on diagnostic catheterization (56.2 ± 30.6, median 45.3, n=41 vs. 43.1 ± 30.6, median 32, n=341, p=0.037 comparing medians).

**Conclusion:** Higher CRP levels are associated with a higher risk of thrombotic occlusion and a lower degree of TIMI perfusion.

**822-3 Effect of Atorvastatin on C-Reactive Protein in Patients With Acute Coronary Syndromes: A Substudy of the MIRACL Trial**

Scott Kinnier, Nader Rili, Peter Libby, Peter Ganz on behalf of the MIRACL Investigators, Brigham and Women’s Hospital, Boston, Massachusetts, Children’s Hospital Boston, Boston, Massachusetts.

**Background:** Coronary lesions of patients with acute coronary syndromes are characterized by intense inflammation. The MIRACL trial showed that atorvastatin 80 mg/day reduced recurrent ischemic events in the first 16 weeks. The purpose of this study was to determine whether intense lytic treatment can reduce serum C-reactive protein (CRP), a marker of inflammation, in patients with unstable angina or non-Q-wave MI. Methods: Blood samples were collected at baseline and at 16 weeks from 2,322 of 3,086 patients participating in the MIRACL trial. C-reactive protein (CRP) was measured by a highly sensitive immunoassay. The change in CRP was compared between patients randomly assigned to atorvastatin vs. placebo. The change in CRP was also evaluated separately in patients with unstable angina and non-Q-wave myocardial infarction.

**Results:** The baseline median CRP was similar in the patients assigned to atorvastatin (10.0 mg/L) and placebo (9.9 mg/L). The reduction in CRP over 16 weeks of treatment was significantly greater in patients on atorvastatin (-7.5 mg/L) compared to placebo (-4.0 mg/L, p < 0.001). Atorvastatin led to a greater reduction in CRP in patients with non-Q-wave MI (-15.9 vs. -10.0 mg/L, atorvastatin vs. placebo, p = 0.011), as well as in patients who were troponin negative (3.5 vs. 2.5 mg/L, atorvastatin vs. placebo, p < 0.001).

**Conclusion:** Atorvastatin (80mg/day) reduced CRP, a marker of inflammation, in patients with acute coronary syndromes. This was observed in patients with non-Q-wave MI as well as unstable angina. Thus, in patients with acute coronary syndromes, atorvastatin reduces inflammation and in combination with cardiac troponins are few. METHODS: Baseline levels of BNP (Biocoll Diagnostic) and cTnI (Bayer Diagnostic) were available for 1561 pts with non-ST elevation ACS randomized to early invasive(INV) vs. conservative(CONS) man-