Platelet Function Predicts Myocardial Damage in Patients With Acute Myocardial Infarction

Martin Frossard, MD*; Ingrid Fuchs, MD*; Judith M. Leitner, MD; Kety Hsieh, MD; Marianne Vlcek, MD; Heidrun Losert, MD; Hans Domanovits, MD; Wolfgang Schreiber, MD; Anton N. Laggner, MD; Bernd Jilma, MD

Background—Platelet activation is a hallmark of acute coronary syndromes. Numerous lines of evidence suggest a mechanistic link between von Willebrand factor or platelet hyperfunction and myocardial damage in patients with acute coronary syndromes. Thus, we assessed whether platelet function under high shear rates (collagen adenosine diphosphate closure times [CADP-CTs]) measured with the platelet function analyzer (PFA-100) may be enhanced in patients with myocardial infarction (MI) and whether it may predict the extent of myocardial damage as measured by creatine kinase (CK-MB) or troponin T (TnT) levels.

Methods and Results—Patients with acute chest pain or symptoms suggestive of acute coronary syndromes (n=216) were prospectively examined at an emergency department. CADP-CT was significantly shorter in patients with MI, particularly in those with an ST-segment-elevation MI (STEMI) compared with the other patient groups (unstable angina, stable coronary artery disease, or controls). Furthermore, CADP-CT and collagen epinephrine–CT at presentation were independent predictors of myocardial damage as measured by CK-MB or TnT. Patients with MI whose CADP-CT values fell in the first quartile had 3-fold higher CK-MB and TnT levels than those in the fourth quartile.

Conclusions—Patients with STEMI have significantly enhanced platelet function when measured under high shear rates. CADP-CT is an independent predictor of the severity of MI, as measured by markers of cardiac necrosis. Measurement of platelet function with the PFA-100 may help in the risk stratification of patients presenting with MI. (Circulation. 2004;110:1392-1397.)

Key words: platelets ■ myocardial infarction ■ creatine kinase ■ collagen

Platelet activation is a hallmark of acute coronary syndromes (ACSs).1–4 Drug trials have demonstrated that various antiplatelet drugs, including aspirin, clopidogrel,5 and glycoprotein (Gp) IIb/IIa inhibitors,6,7 provide substantial therapeutic benefit in patients with ACSs. Thus, numerous lines of evidence suggest that platelets play a dominant pathogenic role in the development and outcome of ACSs.

High shear rates prevail in stenotic arteries: under these conditions, platelet adhesion to the subendothelial matrix of injured vessel walls requires adhesion of plasma von Willebrand factor (vWF) to collagen or platelet adhesion to subendothelially located vWF.8 In addition, vWF enhances the adhesion of platelets to fibrin clots9 and stabilizes coagulation factor VIII. Interestingly, vWF is also a well-characterized marker of cardiovascular risk10; for example, plasma levels of vWF are increased in patients with coronary artery disease (CAD) and predicted subsequent ACSs in the large ECAT study, and vWF is an independent risk factor for recurrent myocardial infarction (MI) and death.11–13

Given the unequivocal role of platelets in CAD and the predictive value of vWF for cardiovascular diseases, 2 hypotheses were formulated: (1) patients with MI may have enhanced platelet function under high shear rates and (2) platelet function under high shear may predict the extent of myocardial necrosis in patients with MI, as assessed by established markers of myocardial damage. Thus, the aim of this study was to investigate whether patients with MI have increased platelet function, as measured with the platelet function analyzer (PFA-100), and whether this could predict the degree of myocardial damage.

Methods

Study Design

The study protocol and informed consent form were approved by the Ethics Committee of the Medical University of Vienna and followed the Helsinki Declaration. All patients with chest pain were seen at the Emergency Department, University Hospital, a tertiary care...
facility. Consecutive patients with chest pain suggestive of myocardial ischemia were eligible for the study.

We enrolled 212 consecutive white patients with chest pain and symptoms suggestive of ACSs. Patients with ECG abnormalities on admission that were strongly suggestive of ST-elevation MI (STEMI) were immediately transferred to the coronary care unit of the emergency department and received standard therapy, including thrombolysis or percutaneous coronary intervention (PCI), at the discretion of the physician.

Patient management was as described previously in a different study, and therapy followed current guidelines. Diagnosis of the various subtypes of ACSs was established by the World Health Organization criteria and the Consensus Document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction.

**Blood Collection and Laboratory Analysis**

All samples for analysis of platelet function testing were collected, after the initial 20 mL of blood was used for clinical chemistry analysis, into evacuated tubes (Vacuette tubes; Greiner Bio-One) containing 129 mmol/L (3.8%) citrate or 106 mmol/L (3.2%) citrate (Vacutainer tubes, Becton Dickinson) before any therapeutic intervention. Serum chemistry was determined with reagents from Roche on a Hitachi 917 analyzer. Troponin t (Troponin T STAT test) was measured on a Roche Instruments Elecsys 2010, and C-reactive protein (normal range 0.5 mg/dL) was measured with Olympus System Reagents (OSR 6147).

**vWF:RiCo Levels**

Plasma levels of vWF ristocetin cofactor activity (vWF:RiCo) were assayed by turbidimetry with a commercial kit from Behring, which consists of lyophilized platelets and ristocetin.

**Platelet Function Analyzer-100**

The platelet function analyzer (PFA-100), a US Food and Drug Administration–approved device (Dade Behring), was used for measuring platelet function because high shear rates prevail at sites of stenosis in the coronary circulation. The PFA-100 measures the time needed for a platelet plug to form after activation of platelets by pathophysiologically relevant stimuli (eg, collagen and adenosine diphosphate [CADP] or collagen and epinephrine [CEPI]), which is prognostic in peripheral arterial occlusive disease. The CADP cartridge was used as the primary outcome variable, because unlike the CEPI closure time (CT), its value is typically not confounded by aspirin intake. Because different laboratories use different citrate concentrations for sample collection, we determined CT values in blood anticoagulated with either 3.2% or 3.8% citrate. Because CADP-CT was shorter in blood anticoagulated with lepirudin compared with 3.8% citrated blood, we expected shorter CT values at lower citrate concentrations. This was indeed the case (Table 3), but CT values were well correlated between the 2 citrate concentrations (r = 0.85 and r = 0.86, P < 0.0001). All measurements were done 0.5 to 4 hours after blood sampling. Coefficients of variation for duplicate analysis averaged 10%, and day-to-day variability in healthy subjects was 9% to 12% for both cartridges.

<table>
<thead>
<tr>
<th>TABLE 1. Demographics of Categorical Variables</th>
<th>Controls (n=25)</th>
<th>CAD (n=20)</th>
<th>UAP (n=31)</th>
<th>Non-STEMI (n=42)</th>
<th>STEMI (n=94)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>76</td>
<td>67</td>
<td>71</td>
<td>86</td>
<td>89</td>
</tr>
<tr>
<td>Relevant medical history</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>10</td>
<td>20</td>
<td>13</td>
<td>16</td>
<td>18</td>
</tr>
<tr>
<td>Hypertension</td>
<td>49</td>
<td>60</td>
<td>68</td>
<td>52</td>
<td>46</td>
</tr>
<tr>
<td>Current smoker</td>
<td>46</td>
<td>30</td>
<td>55</td>
<td>51</td>
<td>56</td>
</tr>
<tr>
<td>Previous MI*</td>
<td>10</td>
<td>53</td>
<td>26</td>
<td>19</td>
<td>11</td>
</tr>
<tr>
<td>CAD*</td>
<td>15</td>
<td>65</td>
<td>29</td>
<td>21</td>
<td>20</td>
</tr>
<tr>
<td>Peripheral arterial occlusive disease</td>
<td>0</td>
<td>15</td>
<td>10</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Cerebral arterial vascular disease</td>
<td>7</td>
<td>20</td>
<td>6</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Family history of MI</td>
<td>50</td>
<td>40</td>
<td>58</td>
<td>54</td>
<td>60</td>
</tr>
<tr>
<td>Home medication</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>24</td>
<td>47</td>
<td>43</td>
<td>40</td>
<td>14</td>
</tr>
<tr>
<td>Aspirin, low dose†</td>
<td>10</td>
<td>60</td>
<td>39</td>
<td>19</td>
<td>22</td>
</tr>
<tr>
<td>β-Blockers‡</td>
<td>17</td>
<td>53</td>
<td>30</td>
<td>31</td>
<td>13</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>7</td>
<td>16</td>
<td>6</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Heparin, including low-molecular-weight type</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>NO donors*</td>
<td>7</td>
<td>37</td>
<td>17</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Statins*</td>
<td>7</td>
<td>47</td>
<td>32</td>
<td>19</td>
<td>14</td>
</tr>
<tr>
<td>Preclinical therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-Blockers</td>
<td>7</td>
<td>15</td>
<td>16</td>
<td>14</td>
<td>27</td>
</tr>
<tr>
<td>NO donors‡</td>
<td>44</td>
<td>50</td>
<td>48</td>
<td>40</td>
<td>51</td>
</tr>
<tr>
<td>Opiates‡</td>
<td>12</td>
<td>10</td>
<td>13</td>
<td>19</td>
<td>67</td>
</tr>
<tr>
<td>Nitro perfusor</td>
<td>2</td>
<td>5</td>
<td>13</td>
<td>14</td>
<td>10</td>
</tr>
<tr>
<td>In-hospital therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>0</td>
<td>5</td>
<td>32</td>
<td>55</td>
<td>84</td>
</tr>
<tr>
<td>Glycolytics inhibitor†</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>29</td>
<td>50</td>
</tr>
<tr>
<td>Fibrinolytic therapy‡</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>18</td>
</tr>
</tbody>
</table>

Values given are percentages. Controls included patients with chest pain of any other origin. UAP indicates unstable angina pectoris; ACE, angiotensin-converting enzyme. All other abbreviations are as defined in text.

*P<0.001, †P<0.0001, ‡P<0.01, by χ² test for descriptive purposes only.
Statistics
A sample size calculation, based on the interindividual variability of CADP-CT, indicated that 32 subjects per group would allow detection of a 12% difference between groups (α=0.05, β=0.2). However, we recruited MI patients in excess of that number to allow better comparison with previous studies on different markers. For descriptive purposes, all data are presented as mean and 95% confidence intervals (CIs). Data were visually examined by histograms, and the normal distribution was tested with the Kolmogorov-Smirnov test. Nonparametric tests were used; the Kruskal-Wallis ANOVA, Mann-Whitney U test, Wilcoxon test, Spearman’s correlation, and χ² test were used as appropriate. Multivariable regression analysis and all other statistics were calculated with Statistica 6.0 software (StatSoft Inc). The level of significance was set to a 2-sided probability value (P<0.05) and corrected for multiple comparisons according to the Bonferroni adjustment.

Results

Patient Characteristics
Demographic variables and selected laboratory values are presented in Tables 1 and 2. All patients were treated with 250 mg aspirin iv after the first blood sample had been taken after admission. MI was found in 64% of patients. Patients with STEMI presented at a median of 2.5 hours after the onset of chest pain. All patients undergoing PCI received a loading dose (300 mg) of clopidogrel. Additionally, eptifibatide, abciximab, and tirofiban were given to 27%, 16%, and 7% of patients with STEMI, respectively. Antiaggregation consisted of unfractionated heparin (peri-interventionally) and mainly enoxaparin and was given to all patients with STEMI.

Aspirin
As expected, chronic pretreatment with aspirin prolonged CEPI-CT to 236 seconds (CI, 212 to 259) compared with those patients who did not receive aspirin before admission (160 seconds; CI, 148 to 173, P<0.0001). This likely contributes at least in part to the differences in CEPI-CT between the different patient groups (Table 3). There was no significant difference in CADP-CT or vWF:RiCO levels between patients who were chronically pretreated with aspirin and those who were aspirin-naive (data not shown). This was also true for all subgroups of patients, although statistical power may be limited.

CASP-CT, CEPI-CT, and vWF:RiCO Levels in Patients and Controls
Patients with STEMI had shorter CADP-CTs than did all other patient groups, particularly with the 3.2% citrated blood (Table 3). Among 29 aspirin-pretreated MI patients, 14 had CADP-CTs of <193 seconds in 3.2% citrate. Those patients had CADP-CT values of 78 seconds (CI, 61 to 96). In aspirin-naive subjects only, STEMI patients had an ≈40% shorter CEPI-CT in 3.2% citrate than did controls or non-STEMI patients (P<0.05; data not shown). Furthermore, patients with STEMI or non-STEMI had higher vWF:RiCO levels than did those patients who did not receive aspirin before admission (160 seconds; CI, 148 to 173, P<0.0001). This likely contributes at least in part to the differences in CEPI-CT between the different patient groups (Table 3). There was no significant difference in CADP-CT or vWF:RiCO levels between patients who were chronically pretreated with aspirin and those who were aspirin-naive (data not shown). This was also true for all subgroups of patients, although statistical power may be limited.

Table 3. CADP and CEPI CTs and Plasma Levels of vWF:RiCO According to Diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Controls (n=25)</th>
<th>CAD (n=20)</th>
<th>UAP (n=31)</th>
<th>Non-STEMI (n=42)</th>
<th>STEMI (n=94)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CADP-CT, seconds, in 3.2% citrate</td>
<td>92* (78-102)</td>
<td>96* (74-122)</td>
<td>94† (86-102)</td>
<td>91‡ (80-102)</td>
<td>74 (70-79)</td>
</tr>
<tr>
<td>CADP-CT, seconds, in 3.8% citrate</td>
<td>102 (87-117)</td>
<td>111* (86-135)</td>
<td>111† (93-130)</td>
<td>116‡ (96-137)</td>
<td>87 (80-94)</td>
</tr>
<tr>
<td>CEPI-CT, seconds, in 3.2% citrate</td>
<td>161 (132-190)</td>
<td>203‡ (158-249)</td>
<td>189§ (155-223)</td>
<td>163† (138-189)</td>
<td>141 (121-161)</td>
</tr>
<tr>
<td>CEPI-CT, seconds, in 3.8% citrate</td>
<td>165 (142-188)</td>
<td>243‡ (202-284)</td>
<td>200† (170-230)</td>
<td>194 (168-219)</td>
<td>173 (154-192)</td>
</tr>
<tr>
<td>vWF:RiCO, %</td>
<td>155* (122-189)</td>
<td>165‡ (131-200)</td>
<td>142† (118-167)</td>
<td>214 (178-250)</td>
<td>214 (193-236)</td>
</tr>
</tbody>
</table>

Data are mean (95% CI). UAP indicates unstable angina pectoris. All other abbreviations are as defined in text.

*P<0.05, †P<0.001, ‡P<0.01, compared with STEMI only. Differences in CEPI-CT may be partly attributable to differences in aspirin home medication (cf Table 1).
levels than did control, CAD, or UAP patients (Table 3). As expected, \( r = -0.57 \) to \(-0.61, P<0.0001 \), and this trend was even stronger in patients with MI \( r = -0.65, P<0.0001 \); correlations between CEPI-CT and vWF:RiCO were less pronounced \( r = -0.31 \) to \(-0.45, P<0.00001 \).

**Baseline CADP-CT and vWF:RiCO Values Correlate With Peak Plasma Levels of Cardiac Markers of Myocardial Necrosis**

CADP-CT determined at the time of admission was correlated inversely with peak plasma levels of creatine kinase (CK), CK-MB, and TnT in patients with MI. The shorter the time needed for platelet plug formation under high shear, the greater the myocardial damage \( (P<0.001 \), Table 4). The correlation between CADP-CT and TnT or CK-MB was even stronger within the subgroup of patients with STEMI \( r = 0.50, P < 0.0001 \) for both markers and again stronger in the subgroup of STEMI patients with PCI. Correlations between CEPI-CT and TnT \( r = -0.47 \) or CK-MB \( r = -0.37 \) were similar even in the subgroup that was not chronically pretreated with aspirin \( P < 0.001 \). Likewise, markers of myocardial damage tended to be correlated with vWF:RiCO \( r = 0.04 \), Table 4; again this correlation increased in patients with STEMI and PCI \( r = 0.49, P < 0.001 \). Because peak levels of CK-MB and TnT were observed at a median of 9 and 22 hours after admission, respectively, CADP-CT thus predicted the degree of myocardial damage already at the time of admission to the emergency department.

For comparison, CK-MB values were \( \sim 3 \)-fold higher (293 U/dL; CI, 177 to 399 versus 97 U/dL; CI, 19 to 176), and TnT levels were 3.3-fold higher (5.3 \( \mu \)g/L; CI, 3.1 to 7.4 versus 1.6 \( \mu \)g/L; CI, 0.9 to 2.3) in the first versus the fourth quartile of CADP-CT in patients with MI (Figure 1).

**CADP-CT and CEPI-CT Are Independent Predictors of the Degree of Myocardial Necrosis in Patients With MI**

The following variables were not correlated with markers of myocardial damage: any drug intake, plasma levels of creatinine, uric acid, triglycerides, cholesterol, C-reactive protein, fibrinogen, hematocrit, platelet count, or body mass index. Furthermore, there was no influence of blood group, sex, or comorbidity, such as history of hypertension, diabetes, nicotine abuse, previous MI, or known CAD on peak values of markers of myocardial damage.

The only other predictor of CK-MB and TnT release was the leukocyte count at admission \( r = 0.35 \) to \(0.42, P<0.001 \) for all MI and \( r = 0.35 \) to \(0.32 \) in STEMI, \( P<0.01 \), which was higher in STEMI than in non-STEMI patients (Table 2). Leukocyte counts were also correlated with CADP-CT or CEPI-CT in MI patients \( r = -0.17 \) to \(-0.29, P < 0.06 \) to \( P = 0.002 \) in 3.2% and 3.8% citrate). Nonetheless, the association between CADP-CT or CEPI-CT and biomarkers of myocardial damage remained significant even after adjusting for leukocyte counts, vWF:RiCO levels, and aspirin use in multiple regression analysis.

**Discussion**

This study provides 2 major novel findings. First, MI patients, particularly those with STEMI, have enhanced platelet function as measured under high shear rates. Second, CADP-CT and CEPI-CT were predictive of the severity of myocardial infarction as measured by plasma levels of CK-MB and TnT.

**TABLE 4. Correlation Between CADP or CEPI CT or Plasma Levels of vWF:RiCO and Markers of Myocardial Necrosis in Patients With MI**

<table>
<thead>
<tr>
<th>Citrate concentration</th>
<th>CADP-CT</th>
<th>CADP-CT</th>
<th>CEPI-CT</th>
<th>CEPI-CT</th>
<th>vWF:RiCO</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK</td>
<td>( -0.41, P&lt;0.0001 )</td>
<td>( -0.35, P&lt;0.001 )</td>
<td>( -0.29, P=0.002 )</td>
<td>( -0.20, P=0.035 )</td>
<td>0.20, (P=0.040 )</td>
</tr>
<tr>
<td>CK-MB</td>
<td>( -0.45, P&lt;0.0001 )</td>
<td>( -0.36, P&lt;0.001 )</td>
<td>( -0.29, P=0.006 )</td>
<td>( -0.26, P=0.007 )</td>
<td>0.19, (P=0.047 )</td>
</tr>
<tr>
<td>TnT</td>
<td>( -0.46, P&lt;0.0001 )</td>
<td>( -0.41, P&lt;0.0001 )</td>
<td>( -0.47, P&lt;0.0001 )</td>
<td>( -0.38, P&lt;0.001 )</td>
<td>0.29, (P=0.004 )</td>
</tr>
</tbody>
</table>

Abbreviations are as defined in text.
Our data further confirm that patients with acute MI have increased vWF:RiCO levels\textsuperscript{29} and demonstrate, probably for the first time, similar levels in non-STEMI and STEMI patients. Plasma levels of vWF:RiCO were correlated well with CADP-CT in the MI patients ($r = -0.65$) of our study. Acute release of vWF markedly decreases CT values.\textsuperscript{18,28} Thus, the shortened CT values in MI patients could partly be due to increased vWF levels. However, STEMI patients had significantly shorter CT values than did non-STEMI patients, despite similar vWF:RiCO levels (Table 4). This indicates that STEMI patients could also have an intrinsic increase in platelet function.

Importantly, this enhanced platelet function was correlated with the degree of myocardial damage in MI patients, and this was observed well before peak levels of CK-MB and TnT were reached. It should be noted that CT values represent a measure of platelet function and, as such, are not specific markers for MI and thus will not directly impact the diagnosis of STEMI. Rather, the shortened CADP-CT fits well into the current concept of the pathogenic mechanisms of MI,\textsuperscript{30} which is caused by acute vascular obstruction due to occlusive thrombi. However, the current study design did not permit conclusions on the cause-effect relation between CADP-CT and the degree of myocardial damage, and the CEPI-CT values may additionally be confounded by aspirin intake.

Still, the negative correlation between CADP-CT and peak TnT levels may be therapeutically useful and not simply because the PFA-100 is similar to a rapid point-of-care test. For example, early-phase therapeutic trials with novel drugs may target MI patients with short CADP-CTs because the signal will conceivably increase if surrogate biochemical markers, such as TnT, are used to judge clinical efficacy. Furthermore, our study provides a rationale to investigate antiplatelet drugs that specifically inhibit CADP-CT in patients with MI to enhance epicardial vessel patency and perhaps to increase microvascular perfusion. Currently, GpIIb/IIIa antagonists and a vWF antibody are known to acutely prolong CADP-CT.\textsuperscript{31–33} So far, however, the clinical efficacy of GpIIb/IIIa inhibitors has been equivocal in terms of mortality in patients with STEMI, although early events may be reduced.\textsuperscript{34} Nonetheless, a subgroup analysis of the ADMIRAL trial suggests that early administration of abciximab in the ambulance or emergency department may provide further benefit.\textsuperscript{35} A vWF-antibody is of particular interest\textsuperscript{32} because in targeting vWF, CADP-CT is prolonged. Thus it acts primarily under high shear rates, which could yield a favorable benefit-risk ratio (efficacy versus bleeding). In addition, ADP receptor inhibitors other than clopidogrel with a faster onset of action will likely come to clinical trials and may be suitable alternative drugs. Finally, patients with shortened CADP-CTs may profit from more “aggressive” therapy, which could help physicians tailor treatment to the individual needs of a patient and thereby optimize the benefit-risk ratio of an intervention.

The observed correlations between CT values and cardiac enzymes were reasonably good, and the degree of correlation in STEMI ($r \geq -0.50$) was markedly better than that of myeloperoxidase ($r = 0.21$)\textsuperscript{25} or leukocyte counts with cardiac enzymes ($r = 0.13$ to $r = 0.28$).\textsuperscript{26,27} This degree of correlation between leukocyte counts and peak CK-MB\textsuperscript{26} was similar to that in the current study, provides external support for the validity of the current findings, and indicates that the results obtained for the CADP-CT measurements are likely robust.

Although the concept of screening for thrombophilia in patients with venous thromboembolism is well established, platelet function tests have been mainly used to detect platelet dysfunction rather than increased platelet function. Our study provides a rationale to look for platelet hyperfunction in patients with MI.

Limitations
Although this is a relatively large study involving numbers of patients with MI similar to those of other recent marker studies,\textsuperscript{23} ours is underpowered for subtle subgroup analysis, eg, women or diabetics. Furthermore, our control group was not a healthy control group but was recruited from patients attending the Emergency Department because of chest pain, who often had concomitant diseases. However, we did not intend to exclude such patients for whatever reason, because we assumed that our approach would bias against the current study and would provide a more conservative estimate of true differences. For reasons of feasibility, other platelet function tests were not assessed in these patients, which would allow direct comparison of sensitivity between methods or the determination of whether it is a high shear- and/or a PFA-100–dependent phenomenon. This should be addressed in future studies.

In summary, STEMI patients have significantly enhanced platelet function as measured under high shear rates, and CT values at admission are an independent predictor of the extent of myocardial necrosis, as measured by CK-MB and TnT levels. Measurement of platelet function with the PFA-100 may help in the risk stratification of patients presenting with MI.

Acknowledgments
This study was supported in part by Dade Behring, Inc, Deerfield, Ill.

References


Platelet Function Predicts Myocardial Damage in Patients With Acute Myocardial Infarction

Martin Frossard, Ingrid Fuchs, Judith M. Leitner, Kety Hsieh, Marianne Vlcek, Heidrun Losert, Hans Domanovits, Wolfgang Schreiber, Anton N. Laggner and Bernd Jilma

_Circulation_. 2004;110:1392-1397; originally published online August 16, 2004;
doi: 10.1161/01.CIR.0000141575.92958.9C

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/110/11/1392

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/