Advances in antithrombotic strategies as support to percutaneous coronary intervention (PCI) have improved periprocedural and long-term outcomes, but thrombotic and bleeding events are still frequent complications. Data from recent studies have shown that bleeding complications after PCI are associated with an increased risk of death at both short- and long-term follow-up.1-10 In particular, it has been shown that both access and nonaccess site bleeding events after PCI are independently associated with mortality, although this association was considerably stronger with bleeding complications not occurring at access site.9,10 To timely identify patients at higher risk of bleeding after PCI, various scores have been developed and validated.11-15 These scores allow estimating the individual patient the probability to develop bleeding complications simply based on the presence of clinical and procedural risk factors.

Platelet reactivity (PR) assessed by point of care assays seems to be effective in predicting ischemic and bleeding events after PCI;16-20 in particular, initial evidences are now available of a therapeutic window for PR after administration of a GPIIb/IIIa inhibitor.21-24 A total of 800 patients undergoing elective PCI via the femoral approach were included. PR was measured before PCI with the VerifyNow P2Y12 assay and low PR was defined as a P2Y12 reaction unit value ≤178. Calculation of the BRS included the following: age, sex, intra-aortic balloon pump, glycoprotein IIb/IIIa inhibitors, chronic kidney disease, anemia, and low-molecular-weight heparin within 48-hour pre-PCI. A new risk score including low PR (BRS-PR) was developed and validated in an independent cohort of patients (n=310). Bleeding events at 30 days after PCI were defined according to the thrombolysis in myocardial infarction, Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events (REPLACE)-2, and Bleeding Academic Research Consortium criteria. Both BRS and PR showed high discriminatory power for bleeding (area under the curve [AUC] >0.7 for all definitions). Discriminatory power of BRS-PR (AUC=0.809 for thrombolysis in myocardial infarction bleeding; AUC=0.814 for Bleeding Academic Research Consortium class ≥2 bleeding; AUC=0.708 for Bleeding Academic Research Consortium class ≥3 bleeding; and AUC=0.813 for REPLACE-2 bleeding) was significantly higher than that of BRS alone (P<0.001 for all bleeding definitions). In the validation set, BRS-PR showed higher discriminatory power for thrombolysis in myocardial infarction bleeding than BRS alone (AUC=0.788 versus 0.709; P=0.036).

Conclusions—PR has incremental predictive value on bleeding events after elective PCI via the femoral approach over a validated risk score of clinical and procedural variables. A risk score including PR yields significantly better prognostic performance compared with the original BRS. (Circ Cardiovasc Interv. 2015;8:e002106. DOI: 10.1161/CIRCINTERVENTIONS.114.002106.)
WHAT IS KNOWN

- Bleeding complications after percutaneous coronary intervention are associated with an increased rate of death at both short- and long-term follow-up.
- Various risk scores have been developed and validated to timely identify patients at higher risk of bleeding after percutaneous coronary intervention.
- Platelet reactivity assessed with the point of care assay VerifyNow seems to be effective in predicting ischemic and bleeding events after percutaneous coronary intervention.

WHAT THE STUDY ADDS

- The results of the current study suggest that platelet reactivity has incremental value over a validated risk score of clinical and procedural characteristics in predicting bleeding events after percutaneous coronary intervention via the femoral approach.
- We have developed and validated in an independent cohort of patients a new risk score including platelet reactivity, which yields significantly better prognostic performance compared with the original bleeding risk score.
- The new bleeding risk score including platelet reactivity may provide a more accurate risk stratification of patients and therefore lead to a better selection of treatment options.

Bleeding complications after PCI are common and may occur in up to 10% of cases. Various risk scores have been developed and validated in databases from the Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events (REPLACE)-1 and REPLACE-2 trials. The bleeding risk score was calculated as previously described by Nikolsky et al. Briefly, risk score models were created by identifying independent predictors of major bleeding in two databases from the Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events (REPLACE-1 and REPLACE-2). The new bleeding risk score including platelet reactivity was developed in an independent cohort of patients and showed better accuracy in predicting bleeding events than the original bleeding risk score.

Methods

Study Population and Design

This is a prospective study enrolling consecutive patients with stable angina or non-ST-elevation acute coronary syndrome (ACS) undergoing elective PCI via the femoral approach at the Department of Cardiovascular Sciences, Campus Bio-Medico University, Rome, Italy, and at the Cardiovascular Center Aalst, Aalst, Belgium, from June 2011 to May 2012.

Pre-PCI antiplatelet treatment consisted of clopidogrel 600 mg loading dose at least 6 hours before the procedure or 75 mg/d for at least 5 days. Administration of further antiplatelet drugs to patients already on chronic treatment was left to the operator’s discretion and based on clinical presentation. Procedural antiaggregation consisted of unfractionated heparin administered to achieve an activated clotting time of 250 to 300 s. Procedural success was defined as a reduction in percent diameter stenosis to <30% in the presence of thrombolysis in myocardial infarction (TIMI) flow grade 3 in the main vessel and all side branches >2 mm in diameter. After PCI, patients receiving bare-metal stents received clopidogrel 75 mg for at least 4 weeks, whereas those with non-ST elevation ACS or undergoing drug-eluting stent implantation received clopidogrel 75 mg for 12 months. Low-dose aspirin (80-100 mg) was administered to all patients before PCI and continued indefinitely. Access site hemostasis after sheath removal was achieved in all patients with manual compression.

Exclusion criteria were use of radial approach, upstream use of glycoprotein IIb/IIIa inhibitors, treatment with oral anticoagulant drugs, platelet count <70×10^9/L, high bleeding risk (active internal bleeding, history of hemorrhagic stroke, intracranial neoplasm, arteriovenous malformation or aneurysm, and ischemic stroke in the previous 3 months), and coronary artery bypass surgery in the previous 3 months.

Clinical follow-up at 30 days was obtained in all patients by office visit, telephone interview, or chart review. All events were classified and adjudicated by a physician not involved in the follow-up process. This study complied with the Declaration of Helsinki and was approved by the local ethics committees, with all patients giving written informed consent.

BRS Calculation

Bleeding risk score was calculated as previously described by Nikolsky et al. Briefly, risk score models were created by identifying independent predictors of major bleeding in databases from the Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events (REPLACE)-1 and REPLACE-2 trials. The new bleeding risk score including platelet reactivity was validated in an independent cohort of patients and showed better accuracy in predicting bleeding events than the original bleeding risk score.

Statistics

Continuous variables are reported as mean±SD or median with lower and upper quartiles, as appropriate. Categorical variables are reported as frequencies and percentages. Comparisons between continuous variables were performed using the Student t test or Mann–Whitney U test. Comparisons between categorical variables were evaluated using the Fisher exact test or the Pearson \( \chi^2 \) test, as appropriate. Receiver operating characteristic curve analysis was used to test the ability of BRS and PR values to discriminate between patients with and without bleeding events at 30-day follow-up. We assessed the incremental
value of combining BRS and PR together in predicting the primary end point. Area under the curve (AUC) was calculated for the logistic regression model including both BRS and PR; differences between AUCs for different models were assessed using the jackknife method, as described by DeLong et al.27 Furthermore, net reclassification improvement (based on 3 risk categories: <2%, 2%–5%, and >5%) and integrated discrimination improvement were used to compare the performance and predictive value of BRS alone or in combination with PR.28 A bleeding risk score including PR (BRS-PR) was then developed in the study population and validated in the cohort of patients enrolled in the Antiplatelet Therapy for Reduction of Myocardial Damage During Angioplasty-Bleeding Study (ARMYDA-BLEEDS) study.18 Besides clinical and procedural variables composing the BRS, low PR (LPR), defined as a PRU value ≤ 178,21 was also included in BRS-PR. Both BRS and LPR were entered in a multivariable logistic regression model to identify independent predictors of TIMI bleeding. Based on the z-score (model coefficient divided by SE), a weighted integer was assigned to LPR. BRS-PR was calculated as the sum of the integers of each variable included in the original BRS14 plus that of LPR. Based on BRS and BRS-PR values, patients were categorized in 4 risk groups: very low risk (0 points), low risk (2–6 points), intermediate risk (7–9 points), and high risk (≥10 points).

No a priori sample size calculation was performed. No adjustment for multiple comparisons was performed. Statistical analysis was performed using Stata/IC version 10.0 (STATA Corp, College Station, TX), and P values <0.05 (2-tailed) were considered significant.

**Results**

**Patient Population**

A total of 800 patients were recruited in this study. Main clinical and procedural characteristics are listed in Table 1. Clinical presentation was non–ST-elevation ACS in 226 patients (28%); however, only 87 patients (11%) received glycoprotein IIb/IIIa inhibitors, whereas no patients were treated with low

<table>
<thead>
<tr>
<th>Table 1. Clinical and Procedural Characteristics</th>
<th>Overall (n=800)</th>
<th>LPR (n=272)</th>
<th>No LPR (n=528)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>67±10</td>
<td>66±10</td>
<td>67±10</td>
<td>0.129</td>
</tr>
<tr>
<td>Male</td>
<td>590 (74)</td>
<td>195 (72)</td>
<td>395 (75)</td>
<td>0.342</td>
</tr>
<tr>
<td>Body mass index</td>
<td>26.1±3.3</td>
<td>25.9±3.0</td>
<td>26.2±3.4</td>
<td>0.219</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>236 (30)</td>
<td>77 (28)</td>
<td>206 (39)</td>
<td>0.003</td>
</tr>
<tr>
<td>Hypertension</td>
<td>630 (79)</td>
<td>213 (78)</td>
<td>417 (79)</td>
<td>0.827</td>
</tr>
<tr>
<td>Current smoking</td>
<td>162 (20)</td>
<td>63 (23)</td>
<td>99 (19)</td>
<td>0.141</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>199 (25)</td>
<td>89 (33)</td>
<td>149 (28)</td>
<td>0.187</td>
</tr>
<tr>
<td>Previous percutaneous coronary intervention</td>
<td>274 (34)</td>
<td>94 (35)</td>
<td>180 (34)</td>
<td>0.895</td>
</tr>
<tr>
<td>Previous coronary artery bypass graft</td>
<td>61 (8)</td>
<td>17 (6)</td>
<td>44 (8)</td>
<td>0.293</td>
</tr>
<tr>
<td>Previous cerebrovascular accident</td>
<td>25 (3)</td>
<td>7 (3)</td>
<td>18 (3)</td>
<td>0.520</td>
</tr>
<tr>
<td>Non–ST-segment–elevation acute coronary syndrome</td>
<td>226 (28)</td>
<td>82 (30)</td>
<td>144 (27)</td>
<td>0.392</td>
</tr>
<tr>
<td>Left ventricle ejection fraction, %</td>
<td>55±7</td>
<td>55±8</td>
<td>56±7</td>
<td>0.695</td>
</tr>
<tr>
<td>Left ventricle ejection fraction &lt;40%</td>
<td>75 (9)</td>
<td>31 (11)</td>
<td>44 (8)</td>
<td>0.159</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>42.0±4.5</td>
<td>41.7±4.6</td>
<td>42.1±4.6</td>
<td>0.244</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>13.9±1.8</td>
<td>13.7±1.9</td>
<td>14.0±1.9</td>
<td>0.145</td>
</tr>
<tr>
<td>Anemia</td>
<td>231 (29)</td>
<td>87 (34)</td>
<td>144 (26)</td>
<td>0.164</td>
</tr>
<tr>
<td>Platelet count, 10^9/L</td>
<td>231±69</td>
<td>229±71</td>
<td>232±68</td>
<td>0.561</td>
</tr>
<tr>
<td>Serum creatinine, mg/dL</td>
<td>1.04±0.29</td>
<td>1.01±0.31</td>
<td>1.05±0.28</td>
<td>0.066</td>
</tr>
<tr>
<td>Estimated glomerular filtration rate, mL/min per 1.73 m²</td>
<td>76.9±24.2</td>
<td>78.1±25.2</td>
<td>76.1±23.9</td>
<td>0.271</td>
</tr>
<tr>
<td>Estimated glomerular filtration rate &lt;60 mL/min per 1.73 m²</td>
<td>173 (22)</td>
<td>57 (21)</td>
<td>116 (22)</td>
<td>0.741</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>336 (42)</td>
<td>102 (38)</td>
<td>234 (44)</td>
<td>0.064</td>
</tr>
<tr>
<td>Target vessel</td>
<td></td>
<td></td>
<td></td>
<td>0.788</td>
</tr>
<tr>
<td>Left main stem</td>
<td>6 (1)</td>
<td>2 (1)</td>
<td>4 (1)</td>
<td></td>
</tr>
<tr>
<td>Left anterior descending</td>
<td>429 (54)</td>
<td>139 (51)</td>
<td>290 (55)</td>
<td></td>
</tr>
<tr>
<td>Left circumflex</td>
<td>139 (17)</td>
<td>49 (18)</td>
<td>90 (17)</td>
<td></td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>221 (27)</td>
<td>81 (30)</td>
<td>140 (27)</td>
<td></td>
</tr>
<tr>
<td>Saphenous vein graft</td>
<td>5 (1)</td>
<td>1 (1)</td>
<td>4 (1)</td>
<td></td>
</tr>
<tr>
<td>No. of stents implanted</td>
<td>1.4±0.9</td>
<td>1.3±0.8</td>
<td>1.4±1.0</td>
<td>0.153</td>
</tr>
<tr>
<td>Intracoronary balloon pump</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glycoprotein IIb/IIa inhibitors</td>
<td>87 (11)</td>
<td>36 (13)</td>
<td>51 (10)</td>
<td>0.124</td>
</tr>
<tr>
<td>Sheath size</td>
<td></td>
<td></td>
<td></td>
<td>0.734</td>
</tr>
<tr>
<td>6 French</td>
<td>713 (89)</td>
<td>241 (89)</td>
<td>472 (89)</td>
<td></td>
</tr>
<tr>
<td>7 French</td>
<td>87 (11)</td>
<td>31 (11)</td>
<td>56 (11)</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean±SD or n (%). LPR indicates low platelet reactivity.
molecular weight heparin or intra-aortic balloon pump. Sheath size was 6 French in 713 patients (89%) and 7 French in the remaining 87 (11%). BRS ranged from 0 to 19, with a mean (±SD) of 7.0±3.7. Pre-PCI PRU levels ranged from 34 to 425, with a mean (±SD) of 207±73 (median 204, lower quartile <155, and upper quartile >256). A total of 272 patients (34%) showed PRU values ≤178 and were classified as having LPR.

### Bleeding Events

At 30-day follow-up, a total of 28 (3.5%) TIMI (7 [0.9%] major and 21 [2.6%] minor), 44 (5.5%) BARC class ≥2, 32 (4.0%) BARC class ≥3, and 32 (4.0%) REPLACE-2 bleedings occurred. The source of bleeding was genitourinary in 5 patients, gastrointestinal in 4 patients, and cerebral in 1 patient, related to the entry site in 32 patients (including 2 retroperitoneal hemorrhages), and unknown in 1 patient with a 5 g/dL hemoglobin loss without an overt source of bleeding. The distribution of bleeding events according to BRS risk groups and to the presence of LPR is shown in Table 2.

LPR remained independently associated with a higher risk of bleeding events even after adjustment for diabetes mellitus, serum creatinine, and multivessel disease.

### Discrimination Analysis

At receiver operating characteristic curve analysis, BRS could significantly discriminate between patients with and without bleeding according to all definitions (AUC, 0.717; 95% confidence interval [CI], 0.639–0.795 for TIMI bleeding; AUC, 0.733, 95% CI, 0.666–0.800 for BARC class ≥2 bleeding;
The integrated discrimination improvement was estimated at 0.047 (P<0.001) for TIMI bleeding, at 0.061 (P<0.001) for BARC class ≥2 bleeding, at 0.024 (P<0.001) for BARC class ≥3 bleeding, and at 0.051 (P<0.001) for REPLACE-2 bleeding.

**New Bleeding Risk Score**

Using this study population as a development cohort, we built a new risk score including LPR in addition to the variables used for the determination of BRS. Henceforth, we used a multivariable model of predictors of TIMI bleeding including LPR and BRS, and, on the basis of the z score, a weighted integer score of 4 was assigned to LPR. BRS-PR ranged from 0 to 23 with a median value of 8 (lower quartile <6, upper quartile >10). The discrimination ability of this new bleeding risk score including PR (BRS-PR) was tested in the development cohort for all bleeding definitions, and in a validation cohort represented by the population of patients enrolled in the ARMYDA-BLEEDS study18 for TIMI bleeding only.

In the development set, BRS-PR could significantly discriminate between patients with and without bleeding according to all definitions (AUC, 0.809; 95% CI [0.74–0.87] for TIMI bleeding; AUC, 0.814; 95% CI [0.76–0.87] for BARC class ≥2 bleeding; AUC, 0.813; 95% CI [0.75–0.87] for REPLACE-2 bleeding). Discriminatory power was significantly better than that of BRS alone (P<0.001 for all bleeding definitions). The distributions of patients with and without bleeding events in the different risk groups of BRS and BRS-PR are shown in Figure 2.

In the validation set (n=310), BRS-PR could significantly discriminate between patients with and without TIMI bleeding with an AUC of 0.788 (Hosmer–Lemeshow P=0.399). The discriminatory power of BRS-PR was significantly better than that of BRS alone (AUC, 0.709; P=0.036). The rates of TIMI bleedings in the 4 risk score groups are shown in Figure 3 for both the development cohort and the validation cohort.

**Discussion**

Major findings of the present study are that (1) PR has incremental value over a validated risk score of clinical and procedural characteristics in predicting bleeding events after PCI.
Increasing concern has been raised in the past few years over the bleeding complications associated with aggressive antiplatelet therapy required in patients treated with coronary stenting. There is compelling evidence that hemorrhagic events after PCI are associated with worse clinical outcomes at long-term follow-up.\(^1\)\(^-\)\(^10\) Ndreppea et al\(^8\) have shown in a study of 5384 patients undergoing PCI that the occurrence of bleeding events within 30 days from stenting was associated with a \(\approx\)3-fold increase in 1-year mortality. Similar results have been recently shown in a large registry (>3.3 million PCI procedures), where bleeding after PCI was associated with a significant increase in inhospital mortality, and 12.1% of deaths were related to periprocedural bleeding.\(^10\) Moreover, both access site and non–access site bleeding were associated with increased inhospital mortality, although this association was stronger for nonaccess bleeding.\(^9\) The fact that even access-site complications carry important prognostic consequences has been confirmed in a recent analysis of 14180 patients treated with coronary stenting where both access and non–access site bleeding events occurring within 30 days after PCI were independently associated with an increased risk of 1-year mortality.\(^9\)

With these premises, an effort to timely identify patients at high bleeding risk seems mandatory to apply appropriate treatment strategies to reduce hemorrhagic complications. In recent years, several risk scores for post-PCI bleeding have been proposed.\(^11\)\(^-\)\(^15\) Although most of these were based on ACS patients undergoing urgent revascularization,\(^11\)\(^,\)\(^12\)\(^,\)\(^14\) Nikolsky et al\(^13\) have developed a risk score model based on the REPLACE-2 trial\(^23\) and validated it in the REPLACE-1 trial\(^23\) population. Similar to the present, the latter studies were mainly composed by stable coronary artery disease patients. This bleeding risk score was based on both clinical and procedural characteristics and demonstrated good prognostic accuracy for major bleeding, significantly discriminating between patients at different levels of risk for major bleeding.

Platelets play a key role in pathogenesis of both thrombotic and bleeding complications. Interindividual variability in response to antiplatelet agents, and clopidogrel in particular, exposes a large proportion of patients to either too high or too low residual PR. Hyporesponders to antiplatelet therapy have high PR and therefore increased risk of thrombotic complications\(^17\)\(^,\)\(^20\)\(^,\)\(^29\), on the other extreme of the spectrum, hyperresponders present low residual PR and therefore increased risk of hemorrhagic complications.\(^15\)\(^,\)\(^19\)\(^,\)\(^30\) Using the VerifyNow P2Y\(_12\) platelet function test, we have recently proposed a threshold of 178 PRU for the definition of LPR,\(^21\) which was associated with a significant increase in 30-day bleeding in patients undergoing elective PCI. Moving from the evidence that both clinical risk scores and PR could predict post-PCI bleeding, we have investigated whether the combination of these parameters could improve their prognostic performance.

In the present study, both PR and BRS alone were able to discriminate between patients with and without bleeding; however, the combination of the two factors together led to a significant increase in discriminatory power. Noteworthy, the superiority of the combination of BRS and PR together over the 2 factors alone was confirmed for all 3 definitions of bleeding used in the present study (TIMI, BARC, and REPLACE-2 definitions). Moreover, we have developed a new risk score including PR in addition to the clinical and procedural variables of BRS. Interestingly, BRS-PR has shown significantly better discriminatory performance compared with the original BRS, showing an AUC >0.8 for all bleeding definition in the development cohort and an AUC of 0.788 for TIMI bleeding in the validation set.

Timely identification of patients at risk of bleeding is key for the adoption of preventive measures. Access-site selection (ie, femoral versus radial approach) is one essential variable, especially when one considers the prognostic implications of often under-rated entry-site complications.\(^9\)\(^,\)\(^10\) Identifying a patient at high risk for bleeding after PCI via the femoral approach could lead to the decision to choose the radial approach for coronary angiography and intervention. Growing evidence suggests that the use of radial approach improves patients’ outcomes, especially in the setting of ACS, by significantly reducing bleeding complications.\(^31\)\(^-\)\(^33\) A pharmacological management tailored on the basis of patients’ risk profile is another potential modality of bleeding prevention. This is particularly true in an era in which we have at our disposal a great variety of antithrombotic drugs, with different efficacy and safety profile. Preferring bivalirudin to unfractioned heparin,\(^34\) or clopidogrel instead of prasugrel\(^35\) and ticagrelor,\(^36\) and limiting the use of glycoprotein IIb/IIIa inhibitors are all possible pharmacological strategies to reduce the incidence of bleeding complications. However, medical management of patients with CAD should always be a tradeoff between thrombotic and bleeding events prevention, and a thorough evaluation of risk profile on an individual basis is mandatory.

In this view, an additional tool such as BRS-PR could be of use in the selection of the appropriate pharmacological and procedural strategies for patients undergoing PCI.

This study has some limitations that are worth mentioning. The assessment of bleeding according to BARC criteria was analyzed retrospectively. By study protocol, only patients undergoing elective PCI via the femoral approach were included; thus, these results might not be applicable to patients undergoing urgent revascularization and radial procedures. However, this allowed to remove a potential confounder related to the vascular access. Although this could be partially responsible for the higher discriminatory power yielded by PR in this compared with other studies,\(^19\)\(^,\)\(^21\) no specific factors have been identified for these discrepancies. In the light of the composition of our study population, we elected the REPLACE bleeding risk score, which was developed in a cohort of predominantly stable patients, and therefore, the observations of this study cannot be extrapolated for patients with ACS undergoing urgent PCI; however, we think that the specificity of BRS-PR is crucial in determining its high prognostic value. No patient was treated with bivalirudin or P2Y12 receptor inhibitor different from clopidogrel, and therefore the observations of the present study cannot be extended to patients receiving those medications. Although the 178 PRU cutoff was derived from our previous work,\(^21\) this might not be ideal for the definition...
of LPR. No adjustment for multiple comparisons was carried out when analyzing the primary end points; however, given the strong correlation between bleeding events resulting from the wide overlap between different definitions, we think that this would not affect the interpretation of the results. Finally, long-term follow-up data were only available for a minority of patients and no specific analysis was attempted.

In conclusion our study suggests that a new risk score (BRS-PR), taking in account also PR, could help to better stratify patients undergoing elective PCI according to bleeding risk profile. This is of particular importance as bleeding events heavily affect prognosis of patients undergoing PCI via the femoral approach, contributing to increase mortality rate on the long term. A more accurate stratification of patients could lead to a better selection of treatment options to improve their prognosis, avoiding both bleeding complications and thrombotic events. Further investigations are needed to confirm the efficacy of BRS-PR and to evaluate the potential role of PR as an. The authors declare no conflicts of interest.

Disclosures

None.

References


Incremental Value of Platelet Reactivity Over a Risk Score of Clinical and Procedural Variables in Predicting Bleeding After Percutaneous Coronary Intervention via the Femoral Approach: Development and Validation of a New Bleeding Risk Score

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