BACKGROUND
Monotherapy with a P2Y₁₂ inhibitor after a minimum period of dual antiplatelet therapy is an emerging approach to reduce the risk of bleeding after percutaneous coronary intervention (PCI).

METHODS
In a double-blind trial, we examined the effect of ticagrelor alone as compared with ticagrelor plus aspirin with regard to clinically relevant bleeding among patients who were at high risk for bleeding or an ischemic event and had undergone PCI. After 3 months of treatment with ticagrelor plus aspirin, patients who had not had a major bleeding event or ischemic event continued to take ticagrelor and were randomly assigned to receive aspirin or placebo for 1 year. The primary end point was Bleeding Academic Research Consortium (BARC) type 2, 3, or 5 bleeding. We also evaluated the composite end point of death from any cause, nonfatal myocardial infarction, or nonfatal stroke, using a noninferiority hypothesis with an absolute margin of 1.6 percentage points.

RESULTS
We enrolled 9006 patients, and 7119 underwent randomization after 3 months. Between randomization and 1 year, the incidence of the primary end point was 4.0% among patients randomly assigned to receive ticagrelor plus placebo and 7.1% among patients assigned to receive ticagrelor plus aspirin (hazard ratio, 0.56; 95% confidence interval [CI], 0.45 to 0.68; P<0.001). The difference in risk between the groups was similar for BARC type 3 or 5 bleeding (incidence, 1.0% among patients receiving ticagrelor plus placebo and 2.0% among patients receiving ticagrelor plus aspirin; hazard ratio, 0.49; 95% CI, 0.33 to 0.74). The incidence of death from any cause, nonfatal myocardial infarction, or nonfatal stroke was 3.9% in both groups (difference, −0.06 percentage points; 95% CI, −0.97 to 0.84; hazard ratio, 0.99; 95% CI, 0.78 to 1.25; P<0.001 for noninferiority).

CONCLUSIONS
Among high-risk patients who underwent PCI and completed 3 months of dual antiplatelet therapy, ticagrelor monotherapy was associated with a lower incidence of clinically relevant bleeding than ticagrelor plus aspirin, with no higher risk of death, myocardial infarction, or stroke. (Funded by AstraZeneca; TWILIGHT ClinicalTrials.gov number, NCT02270242.)
A mong patients who have an acute coronary syndrome or who have undergone percutaneous coronary intervention (PCI), the risk of thrombotic events is lower with dual antiplatelet therapy with aspirin and a P2Y<sub>12</sub> receptor inhibitor than with aspirin alone.\textsuperscript{1} Even with dual antiplatelet therapy, the risk of adverse events remains unacceptably high among patients with enhanced thrombotic risk due to clinical factors (e.g., diabetes mellitus) or angiographic factors (e.g., complex coronary artery disease).\textsuperscript{2,4} The use of more potent P2Y<sub>12</sub> inhibitors or extension of the duration of dual antiplatelet therapy lowers residual ischemic risk among such patients but increases bleeding.\textsuperscript{5-9} Although previously considered relatively benign, post-PCI bleeding has been shown to be associated with a substantial and durable risk of death, approximating or even exceeding that associated with myocardial infarction.\textsuperscript{2,10,11}

Addressing the clinical imperatives of lowering the risk of bleeding while preserving ischemic benefit requires therapeutic strategies that decouple thrombotic risk from hemorrhagic risk. One approach involves shortening the duration of dual antiplatelet therapy through early withdrawal of P2Y<sub>12</sub> inhibition.\textsuperscript{12} Although several studies have shown the feasibility of this approach, they generally have enrolled predominantly low-risk patients and were underpowered for ischemic events.\textsuperscript{13-15} Reducing the duration of aspirin therapy may allow for more prolonged use of potent P2Y<sub>12</sub> inhibitors while avoiding aspirin-related bleeding risk, particularly with respect to gastrointestinal toxicity.\textsuperscript{16} We designed the Ticagrelor with Aspirin or Alone in High-Risk Patients after Coronary Intervention (TWILIGHT) trial to test the hypothesis that in patients undergoing PCI who are at high risk for ischemic or hemorrhagic complications and who have completed a 3-month course of dual antiplatelet therapy with ticagrelor plus aspirin, continued treatment with ticagrelor monotherapy would be superior to ticagrelor plus aspirin with respect to clinically relevant bleeding and would not lead to ischemic harm.

**METHODS**

**TRIAL DESIGN AND OVERSIGHT**

We conducted this randomized, placebo-controlled trial in 187 sites across 11 countries. The trial rationale and design have been described previously.\textsuperscript{17} The Icahn School of Medicine at Mount Sinai designed and sponsored the trial, which was supported by an investigator-initiated grant from AstraZeneca. The executive and steering committees were responsible for trial conduct, the integrity of the data analysis, and the reporting of results. National regulatory agencies and institutional review boards or ethics committees of participating centers approved the trial protocol, which is available with the full text of this article at NEJM.org. An independent data and safety monitoring board provided external oversight to ensure the safety of the trial participants. All the authors vouch for the adherence of the trial to the protocol, and the first, second, and last authors vouch for the accuracy and completeness of the data. The committee members and participating investigators are listed in Table S1 in the Supplementary Appendix, available at NEJM.org. AstraZeneca provided financial support and supplied ticagrelor for the trial but had no role in the design, collection, analysis, or interpretation of the data, in the preparation of the manuscript, or in the decision to submit the manuscript for publication.

**TRIAL POPULATION**

Patients who underwent successful PCI with at least one locally approved drug-eluting stent and whom the treating clinician intended to discharge with a regimen of ticagrelor plus aspirin were eligible to participate. Patients also had to have at least one additional clinical feature and one angiographic feature associated with a high risk of ischemic or bleeding events.\textsuperscript{5,6,17} The clinical criteria for high risk were an age of at least 65 years, female sex, troponin-positive acute coronary syndrome, established vascular disease, diabetes mellitus that was being treated with medication, and chronic kidney disease. Angiographic criteria included multivessel coronary artery disease, a total stent length of more than 30 mm, a thrombotic target lesion, a bifurcation lesion treated with two stents, an obstructive left main or proximal left anterior descending lesion, and a calcified target lesion treated with atherectomy. Key exclusion criteria included presentation with ST-segment elevation myocardial infarction, cardiogenic shock, ongoing long-term treatment with oral anticoagulants, or contraindication to aspirin or ticagrelor. (Tables S2 and S3 show all the criteria and their relation to bleeding and ischemic risks.)
TRIAL REGIMEN
All enrolled patients received open-label ticagrelor (90 mg twice daily) and enteric-coated aspirin (81 to 100 mg daily) after the index PCI. At 3 months after hospital discharge, patients who had not had a major bleeding event (see below) or an ischemic event (stroke, myocardial infarction, or coronary revascularization) were eligible to be randomly assigned in a 1:1 ratio in a double-blind fashion to receive aspirin or matching placebo for an additional 12 months along with continuation of open-label ticagrelor treatment. For the determination of eligibility for randomization, we defined a major bleeding event as Bleeding Academic Research Consortium (BARC) type 3b or higher. BARC bleeding types range from 0 (no bleeding) to 5 (fatal bleeding); type 3b indicates overt bleeding leading to a decrease in hemoglobin level of at least 5 mg per deciliter, cardiac tamponade, surgical intervention, or intravenous treatment with vasoactive drugs. Nonadherence to treatment with ticagrelor or aspirin rendered patients ineligible for randomization. A 3-month course of dual antiplatelet therapy before randomization was considered sufficient on the basis of trials that have suggested equipoise for such a duration.13,18

Randomization was performed with a secure Web-based system; an independent statistician who was not involved with the trial generated the randomization sequence, which was stratified according to site with randomly varying block sizes of 4, 6, and 8. Follow-up was performed by telephone at 1 month after randomization and in person at 6 and 12 months after randomization. Adherence was assessed with manual pill counts, and nonadherence was classified according to the underlying reason, as described previously.19 After 12 months of protocol-mandated therapy, patients were switched to a standard-of-care antiplatelet regimen at the discretion of their treating physician, followed by final telephone follow-up 3 months later.

END POINTS
The primary end point was the first occurrence of BARC type 2, 3, or 5 bleeding between randomization and 1 year in a time-to-event analysis. The key secondary end point was the first occurrence of death from any cause, nonfatal myocardial infarction, or nonfatal stroke in a time-to-event analysis. Secondary bleeding end points included BARC type 3 or 5 bleeding; Thrombolysis in Myocardial Infarction (TIMI) major or minor bleeding; Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) moderate, severe, or life-threatening bleeding; or major bleeding as defined by the International Society on Thrombosis or Haemostasis (ISTH).23 Other secondary end points included death from cardiovascular causes, myocardial infarction, ischemic stroke, and definite or probable stent thrombosis. Myocardial infarction was defined according to the third universal definition,24 and revascularization and stent thrombosis were classified according to the Academic Research Consortium.25 Table S4 lists the primary and all secondary end points and their associated definitions. All clinical events were adjudicated by an external independent committee, the members of which were unaware of the treatment-group assignments.

STATISTICAL ANALYSIS
The sample-size and power calculation was based on a superiority assumption for the primary end point of BARC type 2, 3, or 5 bleeding. Assuming a bleeding incidence of 4.5% at 1 year with ticagrelor plus aspirin, we chose a sample size of 8200, which provided 80% power to detect a 28% lower incidence in the ticagrelor-plus-placebo group with a type I error rate of 0.05. The key secondary end point (composite of death from any cause, nonfatal myocardial infarction, or nonfatal stroke) was evaluated with the use of a prespecified noninferiority hypothesis. Under the assumption of an incidence of 8.0% at 1 year in the ticagrelor-plus-aspirin group, a sample size of 8200 provided 80% power to rule out an absolute difference in risk of 1.6 percentage points, with a one-sided type I error rate of 0.025. This margin is consistent with those in other trials that have evaluated pharmacologic and device-based interventions within a noninferiority framework.13,26

The cumulative incidence of the primary and secondary end points was estimated by the Kaplan–Meier method. Data from patients who had not had a primary end-point event between randomization and 1 year were censored at the time of death, the time of last known contact, or 365 days, whichever came first. Hazard ratios and 95% confidence intervals were generated with Cox proportional-hazards models. Absolute differences and 95% confidence intervals for
primary and key secondary end points at 1 year were calculated with Kaplan–Meier estimates and Greenwood standard errors.27 Primary analyses of bleeding and ischemic end points were performed in the intention-to-treat and per-protocol populations, respectively. Patients who underwent randomization and did not fulfill enrollment criteria, were not eligible for randomization, or never received protocol-mandated therapy were excluded from the per-protocol analysis.

RESULTS

PATIENT CHARACTERISTICS

From July 2015 through December 2017, a total of 9006 patients were enrolled after PCI, and 7119 were randomly assigned 3 months later to receive ticagrelor plus placebo or ticagrelor plus aspirin (intention-to-treat population). The last patient underwent randomization in April 2018, and the database was locked in July 2019. Details regarding the reasons for 1887 enrolled patients not undergoing randomization, the baseline clinical and procedural characteristics among patients who did and those who did not undergo randomization, and adverse events among patients who did not undergo randomization are provided in Tables S5 through S9.

Ascertainment of the primary end point was complete in 98.4% of the patients who underwent randomization, and data on vital status were obtained in 99.7% (Fig. 1). Demographic, clinical, and procedural characteristics were well balanced between the treatment groups; the mean age was 65 years, 23.8% of the patients were female, 36.8% had diabetes mellitus, and 64.8% underwent PCI for an acute coronary syndrome indication (29.8% with non–ST-segment elevation myocardial infarction) (Table 1 and Table S10). Adherence to ticagrelor treatment 1 year after randomization was similar in the ticagrelor-plus-placebo group and the ticagrelor-plus-aspirin group (87.1% and 85.9%, respectively) (Fig. S1).

BLEEDING

Table 2 shows the incidences of primary (BARC) and secondary (TIMI, GUSTO, and ISTH) bleeding end points, and Figure 2 shows the Kaplan–Meier curves for the primary end point. The primary end point occurred in 141 patients (4.0%) who received ticagrelor plus placebo, as compared with 250 patients (7.1%) who received ticagrelor plus aspirin (hazard ratio, 0.56; 95% confidence interval [CI], 0.45 to 0.68; P<0.001), for a difference in risk of −3.08 percentage points (95% CI, −4.15 to −2.01). The incidence of BARC type 3 or 5 bleeding was 1.0% in the group that received ticagrelor plus placebo and 2.0% in the group that received ticagrelor plus aspirin (hazard ratio, 0.49; 95% CI, 0.33 to 0.74). The treatment effect for the primary end point was consistent across predefined subgroups (Fig. S2).

ISCHEMIC EVENTS

Ischemic events were analyzed in the per-protocol population, which included the 7039 patients who underwent randomization and had no major deviations from the protocol (3524 who received ticagrelor plus placebo and 3515 who received ticagrelor plus aspirin). The key secondary composite end point of death from any cause, nonfatal myocardial infarction, or nonfatal stroke occurred in 135 patients (3.9%) who received ticagrelor plus placebo and in 137 patients (3.9%) who received ticagrelor plus aspirin (hazard ratio, 0.99; 95% CI, 0.78 to 1.25), for a difference in risk of −0.06 percentage points (95% CI, −0.97 to 0.84) (Fig. 3). The incidence of death from any cause was similar in group that received ticagrelor plus placebo and the group that received ticagrelor plus aspirin (1.0% and 1.3%, respectively), as were the incidences of myocardial infarction (2.7% in both groups) and definite or probable stent thrombosis (0.4% and 0.6%) (Table 2). There were 16 instances of ischemic stroke in the group that received ticagrelor plus placebo and 8 instances of ischemic stroke in the group that received ticagrelor plus aspirin (0.5% and 0.2% of patients, respectively). The effect of ticagrelor monotherapy on the key secondary end point was consistent across predefined subgroups (Fig. S3).

ADDITIONAL ANALYSES

Landmark analyses of the period from 15 to 18 months after PCI showed similar incidences of BARC type 2, 3, or 5 bleeding in the group that received ticagrelor plus placebo and the group that received ticagrelor plus aspirin (0.7% and 0.5%, respectively; hazard ratio, 1.24; 95% CI, 0.64 to 2.40); the incidence of death, myocardial infarction, or stroke was also similar in the two groups (0.9% and 1.1%; hazard ratio, 0.84; 95%
CI, 0.51 to 1.40). Sensitivity analyses conducted with an imputation-based approach to account for the 112 patients who were lost to clinical follow-up yielded similar effects for the primary and key secondary end points.\textsuperscript{28} (Details of these analyses are provided in Tables S11 and S12 and Fig. S4.)

**Discussion**

Our trial was designed to examine the effect of withdrawing treatment with aspirin while continuing treatment with ticagrelor alone after 3 months of dual antiplatelet therapy in patients who received drug-eluting stents and were at high risk for bleeding or ischemic events. Ticagrelor monotherapy was associated with a 44% lower risk of BARC type 2, 3, or 5 bleeding over 1 year than ticagrelor plus aspirin (absolute difference in risk, 3.1 percentage points). The bleeding-related benefits of ticagrelor monotherapy extended to more severe BARC type 3 or 5 bleeds and persisted when alternative bleeding scales were considered. In this trial, there was no evidence of a higher risk of death, myocardial infarction, or stroke among patients who received ticagrelor monotherapy than among those who received ticagrelor plus aspirin. The treatment effect with respect to both bleeding and ischemic end points was consistent across subgroups. In aggregate, these results show that a transition to an antiplatelet strategy of treatment with ticagrelor alone after a 3-month course of dual antiplatelet therapy in high-risk patients who had undergone PCI provided a clinical benefit of less bleeding without ischemic harm.
Two previous studies showed that among patients who had undergone PCI and were at relatively low risk for ischemic events, clopidogrel monotherapy after 1 to 3 months of dual antiplatelet therapy was associated with a significantly lower incidence of bleeding than clopidogrel plus aspirin, without an apparent difference in ischemic risk.\textsuperscript{29,30} The modest size of those studies, as well as the low-risk nature of the patient population, precluded conclusive inference regarding the effect of clopidogrel monotherapy on ischemic end points. Distinct from these trials, we enrolled a larger population of patients who more commonly had both clinical and angiographic high-risk criteria and were treated with a more potent P2Y\textsubscript{12} inhibitor, ticagrelor.

In contrast to our findings, the findings of the GLOBAL LEADERS trial showed that 1 month of dual antiplatelet therapy followed by ticagrelor monotherapy for an additional 23 months was not associated with a lower incidence of bleeding.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ticagrelor plus Placebo (N = 3555)</th>
<th>Ticagrelor plus Aspirin (N = 3564)</th>
</tr>
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<tr>
<td>Age — yr</td>
<td>65.2±10.3</td>
<td>65.1±10.4</td>
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<tr>
<td>Female sex — no. (%)</td>
<td>846 (23.8)</td>
<td>852 (23.9)</td>
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<tr>
<td>Nonwhite race — no. (%) †</td>
<td>1110 (31.2)</td>
<td>1086 (30.5)</td>
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<td>Body-mass index‡</td>
<td>28.6±5.5</td>
<td>28.5±5.6</td>
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<td>Enrolling region — no. (%)</td>
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</tr>
<tr>
<td>North America</td>
<td>1484 (41.7)</td>
<td>1488 (41.8)</td>
</tr>
<tr>
<td>Europe</td>
<td>1251 (35.2)</td>
<td>1258 (35.3)</td>
</tr>
<tr>
<td>Asia</td>
<td>820 (23.1)</td>
<td>818 (23.0)</td>
</tr>
<tr>
<td>Diabetes mellitus — no. (%)</td>
<td>1319 (37.1)</td>
<td>1301 (36.5)</td>
</tr>
<tr>
<td>Diabetes treated with insulin — no. (%)</td>
<td>335 (9.4)</td>
<td>374 (10.5)</td>
</tr>
<tr>
<td>Chronic kidney disease — no./total no. (%) †</td>
<td>572/3410 (16.8)</td>
<td>573/3425 (16.7)</td>
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<td>Anemia — no./total no. (%)</td>
<td>675/3405 (19.8)</td>
<td>654/3423 (19.1)</td>
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<td>Current smoker — no./total no. (%)</td>
<td>726/3553 (20.4)</td>
<td>822/3562 (23.1)</td>
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<td>Hypercholesterolemia — no. (%)</td>
<td>2157 (60.7)</td>
<td>2146 (60.2)</td>
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<td>Hypertension — no./total no. (%)</td>
<td>2580/3555 (72.6)</td>
<td>2574/3563 (72.2)</td>
</tr>
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<td>Peripheral arterial disease — no. (%)</td>
<td>245 (6.9)</td>
<td>244 (6.8)</td>
</tr>
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<td>Previous myocardial infarction — no. (%)</td>
<td>1020 (28.7)</td>
<td>1020 (28.6)</td>
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<tr>
<td>Previous PCI — no. (%)</td>
<td>1502 (42.3)</td>
<td>1496 (42.0)</td>
</tr>
<tr>
<td>Previous CABG — no./total no. (%)</td>
<td>362/3554 (10.2)</td>
<td>348/3564 (9.8)</td>
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<td>Multivessel coronary artery disease — no. (%)</td>
<td>2272 (63.9)</td>
<td>2194 (61.6)</td>
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<td>Previous major bleeding event — no. (%)</td>
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<td>32 (0.9)</td>
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<tr>
<td>Indication for PCI — no./total no. (%)</td>
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<td>Asymptomatic</td>
<td>234/3554 (6.6)</td>
<td>223/3563 (6.3)</td>
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<tr>
<td>NSTEMI</td>
<td>1024/3554 (28.8)</td>
<td>1096/3563 (30.8)</td>
</tr>
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</table>

\textsuperscript{*} Plus–minus values are means ±SD. Percentages may not total 100 because of rounding. CABG denotes coronary artery bypass graft, NSTEMI non–ST-segment elevation myocardial infarction, and PCI percutaneous coronary intervention. † Race was reported by the patient. ‡ The body-mass index is the weight in kilograms divided by the square of the height in meters. § Chronic kidney disease was defined as an estimated glomerular filtration rate of less than 60 ml per minute per 1.73 m\textsuperscript{2} of body-surface area.
than a conventional antiplatelet strategy. These results may be attributable to differences in trial design (double-blind vs. open-label), patient case mix (high-risk patients vs. all comers), duration of therapy after randomization (12 months vs. 23 months), comparator regimens (ticagrelor plus aspirin vs. dual antiplatelet therapy followed by aspirin), bleeding ascertainment (judicatied vs. site-reported), or protocol adherence. Consequently, any putative bleeding-related advantage associated with the withdrawal of aspirin therapy may have been attenuated in GLOBAL LEADERS.

For instance, ticagrelor monotherapy was associated with a nonsignificant 14% lower incidence of BARC type 3 or 5 bleeding at 1 year in GLOBAL LEADERS, whereas a 51% lower incidence was observed in our trial.

To be included in our trial, patients had to have clinical and angiographic factors associated with a high risk of either bleeding or ischemic events after PCI, design features that reflect the primary and key secondary end points of the trial. With respect to bleeding, patients at high risk are most likely to have a benefit from reduced exposure to antiplatelet therapy. With regard to ischemic events, we enrolled patients with a high risk of such events to identify signals of harm after withdrawal of aspirin therapy. Although
most of the prespecified criteria are associated with excess thrombosis (e.g., diabetes mellitus), others are linked to both types of events (e.g., renal impairment). Moreover, only patients whom a clinician had already decided to treat with aspirin and ticagrelor were eligible for enrollment, which resulted in a trial population with a level of ischemic and bleeding risk that reflects the overall trial design and clinical preferences for ticagrelor use.

Major adverse events occurring early after PCI and nonadherence to dual antiplatelet therapy precluded randomization at 90 days, criteria that led to the population of patients who underwent randomization having a clinical and angiographic profile distinct from that of the initially enrolled participants. Nonetheless, several high-risk characteristics (e.g., diabetes mellitus and long stent length) remained prevalent among the patients who underwent randomization. Moreover, the incidences of bleeding and ischemic events at 1 year in these patients were similar to or higher than those reported in trials in which all events from PCI onward were considered, thereby substantiating the high-risk nature of our trial population.29-31

Although guidelines recommend ticagrelor in the context of acute coronary syndrome alone, 33% of the trial participants were in stable condition at the time of enrollment.3 Potential reasons for the inclusion of such patients by trial investigators may have included a perceived lack of benefit with clopidogrel or clinical equipoise with regard to P2Y13 inhibitor choice in high-risk patients who are stable after PCI. Corroborating such tendencies, findings from usual-care registries show that in current practice, more than 10% of patients who have undergone PCI and are treated with ticagrelor initially present with a non–acute coronary syndrome indication.32,33 From a clinical standpoint, our results suggest that ticagrelor monotherapy may be a suitable antiplatelet strategy to lower the risk of bleeding while simultaneously preserving ischemic benefit in patients who have undergone PCI and are characterized by a minimum threshold of risk. These effects appear consistent in patients whose condition is either stable or acute. Whether the findings would be similar in a lower-risk population or if a different antithrombotic regimen were used remains unknown.

The limitations of our trial include the lack of power to detect differences in the risk of important yet rare clinical events, such as stent thrombosis and stroke. Although ischemic strokes were more common among patients who received placebo than among those who continued to receive aspirin, only 24 such events occurred
during the trial, thereby precluding conclusive inference for this end point. Other studies have shown that P2Y₁₂ inhibitor monotherapy is not associated with a higher risk of cerebrovascular events than dual antiplatelet therapy. Results from this trial may not be generalizable to all patients who have undergone PCI, given the requirement in our trial for both high-risk (clinical and angiographic) features and a willingness to be treated with ticagrelor. In addition, the observed treatment effects do not apply to all enrolled participants but rather to those patients who were able to take 3 months of dual antiplatelet therapy without any major adverse events. Our primary end point included bleeding events of varying severity, which may have altered the risk–benefit calculation for considering ticagrelor or monotherapy. A lower-than-expected incidence of the composite end point of death, myocardial infarction, or stroke may have biased our results for this key secondary end point toward the null.

Our trial showed that in high-risk patients who had undergone PCI and were treated with ticagrelor and aspirin for 3 months, an antiplatelet strategy of continuing ticagrelor alone resulted in substantially less bleeding than ticagrelor plus aspirin, without leading to ischemic harm over a period of 1 year.

A data sharing statement provided by the authors is available with the full text of this article at NEJM.org.

Supported by AstraZeneca.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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APPENDIX

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REFERENCES


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