Periprocedural Myocardial Infarction in a Randomized Trial of Everolimus-Eluting and Paclitaxel-Eluting Coronary Stents
Frequency and Impact on Mortality According to Historic Versus Universal Definitions

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Background—A consensus definition for periprocedural myocardial infarction (MI) in coronary stent trials has not been established. Differences between a historic definition, based on modified World Health Organization (WHO) criteria, and a proposed universal definition have not been compared in a prospective clinical trial.

Methods and Results—We randomly assigned 3687 patients with stable coronary artery disease to undergo stenting with either everolimus-eluting stents (2458 patients) or paclitaxel-eluting stents (1229 patients). Serial creatine kinase (CK) and CKMB or troponin measurements were obtained before and after stenting. MI was classified by protocol according to the WHO definition (total CK >2× normal with elevated CKMB) and post hoc according to the Universal/Academic Research Consortium (ARC) definition (CKMB or troponin >3× normal). Protocol MI was determined in 58 (1.6%) and universal/ARC MI in 287 (7.8%) patients within 48 hours post index procedure. There were substantial differences in frequency of universal/ARC MI if only CKMB (5.4%) or troponin (18.7%) data were included for evaluation. Total stent length was a strong predictor of both protocol and universal/ARC MI. Mortality at 2 years was low (2.3%) and was not different for either MI definition. The mortality rates did not increase with elevations of CKMB or troponin to >10× normal.

Conclusions—There was a marked difference in periprocedural MI rates according to protocol or universal/ARC MI definitions. No association was present between periprocedural MI and mortality up to 2 years by either definition.

Clinical Trial Registration—URL: http://www.clinicaltrials.gov. Unique identifier: NCT00307047.

Key Words: periprocedural myocardial infarction ■ stent ■ universal definitions ■ PCI

Myocardial infarction (MI) after a percutaneous coronary intervention (PCI) is an important safety event and is included as a component of the primary end point in coronary device and pharmacological clinical trials. Although any elevation of creatine kinase (CK) or its MB isoform (CKMB) after a PCI has been shown to represent myocardial necrosis,1 the clinical significance of small infarctions manifest by low-level biomarker elevation remains controversial.2,3 Whereas some studies have suggested that any level of elevation is an independent risk factor for mortality,4,5 others have only noted Q-wave or large non-Q-wave MI or events associated with unsuccessful coronary procedures to be predictive of late mortality.6,7 Thus, the available data are inadequate to establish a single consensus definition for periprocedural MI that is appropriately sensitive for detection of myocardial necrosis while maintaining specificity for potentially clinically meaningful events.

Prior stent clinical trials have relied mostly on a modified World Health Organization (WHO) definition for establishing a biomarker threshold for periprocedural MI. Under this definition, MI is defined by elevation of total CK to >2× normal along with elevated or “positive” CKMB.8 In 2007, a
global task force for universal definition of MI proposed a threshold of 3× the 99th percentile upper reference limit of normal for CKMB or troponin (I or T) to define periprocedural MI and recommended troponin as the preferred biomarker.9 Although controversy and debate continue, this universal definition has been adopted by an academic research consortium (ARC) of clinical researchers and stent manufacturers and supported by the United States Food and Drug Administration for use in coronary stent clinical trials.10

The SPIRIT IV trial was a prospective, randomized, multicenter trial of PCI comparing 2 drug-eluting stents, the everolimus-eluting XIENCE V versus the paclitaxel-eluting TAXUS Express stent, in patients with stable coronary artery disease in which serial CK and CKMB or troponin measurements were obtained routinely before and after PCI.11 We sought to compare the frequency of periprocedural MI according to the historic WHO criteria versus the newer global task force universal definition and to assess the impact of periprocedural MI by each definition on subsequent mortality.

### WHAT IS KNOWN

- The clinical significance of small infarctions manifested by low-level biomarker elevation remains controversial.
- There are inadequate data to establish a single consensus definition for periprocedural MI that is appropriately sensitive for detection of myocardial necrosis while maintaining specificity for potentially clinically meaningful events.

### WHAT THE STUDY ADDS

- The study confirms a marked difference in periprocedural MI rates according to whether historic or universal/ARC MI definitions are used.
- Use of a more sensitive definition of periprocedural MI based on troponin >3× the diagnostic level for MI increases the sensitivity for diagnosis, but with an event rate of nearly 20% may provide less discrimination for clinically important events.
- The lack of an association of periprocedural MI by either definition with 2-year mortality supports other studies in low risk patients with otherwise successful procedures.

### Methods

#### Study Design and Population

The design and patient population of the Spirit IV trial have been described previously.11,12 In brief, patients who were 18 years or older and had a maximum of 3 previously untreated lesions 28 mm or less in length (maximum of 2 lesions per native epicardial artery) with visual reference vessel diameter of 2.5–3.75 mm were included in the study. Patients with acute MI preceding the index procedure were excluded. A total of 3687 patients were enrolled at 66 US sites and were randomly assigned in 2:1 fashion to receive either everolimus-eluting stents (XIENCE V, Abbott Vascular, Santa Clara, CA) (2458 patients) or paclitaxel-eluting stents (TAXUS EXPRESS, Boston Scientific, Natick, MA) (1229 patients). This study was approved by the institutional review board at each participation center, and consecutive eligible patients signed written informed consent. Independent study monitors verified 100% of case-report form data onsite. The follow-up rate at 2 years was 96.0%.

All patients received at least 300 mg of aspirin before the procedure and then at least 80 mg per day, continued indefinitely. All patients also received at least 300 mg of clopidogrel either before or within 1 hour after the procedure and 75 mg per day for at least 1 year according to American College of Cardiology/American Heart Association guidelines.

CK and CKMB measurements were performed at baseline within 48 hours before the index procedure except when there was evidence of acute or recent (<7 days) MI or unstable angina before the procedure, in which case preprocedure biomarkers were required to be obtained within 24 hours and documented to have returned to normal. If a successful percutaneous intervention in a nontarget vessel was performed within 24–48 hours before the index procedure, CK and CKMB were required to be <2× the upper limit of normal (ULN) at the time of the index procedure. Postprocedure measurement was required to be performed between 12 hours after the procedure and hospital discharge. Preprocedure and postprocedure CKMB measurements were also required if CK measurement was greater than ULN. If postprocedure CKMB was ≥3× ULN, serial measurements of CK and CKMB were to be done until a decline was noted. Preprocedure and postprocedure troponin I or T measurements were not required by protocol unless an acute coronary syndrome was suspected. All biomarkers were normalized according to each individual site laboratory’s normal range.

#### Study End Points

The study end points for this retrospective analysis included periprocedural MI and all-cause mortality. Periprocedural MI for this analysis referred to the MI occurring within 48 hours post index procedure by either protocol or universal/ARC definition. Protocol MI was defined according to the modified World Health Organization criteria as total CK elevation >2× ULN with elevated CKMB. The universal/ARC MI was defined as a CKMB or troponin elevation ≥3× ULN. For either definition of MI, elevation of biomarkers at baseline required 2 values at least 6 hours apart that were stable or declining before PCI and subsequent reelevation >20% of nadir and above threshold for diagnosis. The ULN was determined at each clinical center as the value diagnostic of MI. Cardiac death was defined as any death due to acute MI; cardiac perforation/pericardial tamponade; arrhythmia or conduction abnormality; cerebrovascular accident within 30 days of the procedure or cerebrovascular accident suspected of being related to the procedure; and death due to complications of the procedure, including bleeding, vascular repair, transfusion reaction, or bypass surgery; or any death in which a cardiac cause could not be excluded. All protocol end points were adjudicated by an independent committee that was blinded to the treatment assignments. In the case of universal/ARC MI, 2 of the authors (Drs Pervaiz and Cutlip) performed review of all CKMB and troponin data to verify the periprocedural universal/ARC MI criteria. In addition, complications of PCI of each subgroup were evaluated using a composite of side branch occlusion (%DS >95), residual dissection (defined as National Heart, Lung, and Blood Institute grade B or greater), decreased Thrombolysis In Myocardial Infarction flow (<3 in main vessel), or distal embolization post index procedure.

#### Statistical Analysis

Dichotomous variables, including event rates, are presented as counts and percentages and compared using the Fisher exact test. Continuous variables are presented as means with standard deviation or as medians with interquartile range. The Student t test and generalized estimating equations analysis were used for comparison of continuous variables for patient-level and lesion-level analyses, respectively. Univariable and multivariable stepwise logistic regression analyses were used to determine the predictors of periprocedural MI according to the protocol and universal/ARC MI definitions. All analyses were performed with a probability value <0.05 considered...
The multivariable model was created using stepwise regression, where variables were entered into the model at the 0.20 significance level and removed at the 0.05 level (from the Wald $\chi^2$ statistic). Variables were eligible for inclusion in the multivariable model-building process if the variable was present for 90% of the subjects in the analysis, had a univariate probability value $P < 0.20$, and, if highly correlated with another variable ($r > 0.5$ and $P < 0.05$), had the higher level of significance.

**Results**

**Patient and Procedural Characteristics by MI Status**

The postprocedural biomarker data and MI status are summarized in Figure 1. Of 3687 randomly assigned patients, evaluable biomarker data were available post index procedure for 3642 (99%). Total CK was available for 3615 (98%), CKMB was available for 3079 (84%), and troponin was available for 1113 (30%) patients. Both CKMB and troponin data were available after the procedure in 1061 (29%) patients. The availability of paired biomarker data at baseline was 98.9% for CK (3575/3615), 89.2% for CKMB (2746/3079), and 79.8% for troponin (888/1113). Elevation (greater than ULN) was observed in 5.8% of CK (209/3575), 4.0% of CKMB (109/2746), and 8.8% of troponin (78/888) at baseline. Protocol MI was adjudicated in 58 (1.6%) and universal/ARC MI was confirmed in 287 (7.8%) patients. Missing baseline biomarker data were treated as not elevated at baseline. Of the 58 protocol MIs, 2 (3.4%) had missing baseline total CK data. Of the 287 universal/ARC MIs, 39 (13.6%) had neither CKMB nor troponin baseline data.

Table 1 shows baseline clinical and lesion characteristics for protocol MI, universal/ARC MI, and no MI groups. Compared with the no MI group, both protocol MI and universal/ARC MI groups had significantly smaller preprocedure MLD and longer total stent length per lesion. In addition, the universal/ARC MI group had significantly more multivessel disease and more circumflex target lesions compared with the no MI group. An increased frequency of PCI complications was observed in both protocol MI and universal/ARC MI groups compared with the no MI group.

**Analysis of Cardiac Biomarker Measurements by MI Status**

The postprocedural peak cardiac biomarker levels of CKMB and troponin are summarized for both the periprocedural protocol MI and the universal/ARC MI groups in Table 2. In general, troponin measurements were demonstrated to be more variable than CKMB measurements. Compared with the universal/ARC MI group, wider distributions (median, 25th and 75th percentiles) of both CKMB and troponin measurements were observed for the 58 adjudicated periprocedural protocol MIs. In addition, for the group of universal/ARC MI with troponin $> 3 \times$ ULN ($n = 208$), the median of CKMB was actually $< 3 \times$ ULN.

**Correlates of Periprocedural MI**

The baseline clinical, angiographic, and procedural variables associated with either definition of periprocedural MI are shown in Table 3. Total stent length was a strong predictor ($P < 0.01$) of periprocedural MI by either protocol or universal/ARC definition. Number of treated lesions was also a strong predictor of universal/ARC MI.

**Frequency of Periprocedural Universal/ARC MI by CKMB Versus Troponin**

Figure 2 shows distributions of the peak CKMB and troponin values. There were differences in MI rates based on availability of CKMB or troponin data and substantial differences in levels of biomarker relative to the ULN. For the 1113 patients with troponin data available (with or without CKMB), there were 208 (18.7%) evaluated as MI by universal/ARC definition using only troponin data. Of the 3079 patients with CKMB data available (with or without troponin), a lower rate of universal/ARC MI (5.4%, 167/3079) was determined, based on CKMB data only. Of the 1061
patients with both CKMB and troponin levels available, 149 (14.0%) had troponin $>3\times$ normal but CKMB $<3\times$ normal, whereas only 7 (0.7%) patients had CKMB $>3\times$ ULN with troponin $<3\times$ normal. Thus, at a threshold of $3\times$ ULN, there were 95 (9.0%) patients who were concordant for periprocedural universal/ARC MI by CKMB and troponin both above the threshold and 810 patients (76.3%) with CKMB and troponin both below the threshold.

**Periprocedural MI and Impact on Mortality**

Low rates of all-cause mortality were observed at 30 days, 1 year, or 2 years, regardless of whether or not periprocedural MI occurred by either protocol or universal/ARC definition (Table 4). Figure 3 shows mortality within 2 years according to CKMB and troponin threshold values within 48 hours post index procedure. Similarly low all-cause mortality rates were observed among various groups by CKMB or troponin levels.

**Discussion**

To our knowledge, this is the first randomized clinical trial to analyze differences in frequency and outcomes of periprocedural MI according to historic WHO and updated universal definitions. There was a marked difference in MI rates by historic (protocol) and universal/ARC definitions. There was no association between postprocedural MI and mortality using either definition, even at high levels of CKMB or troponin elevation ($>10\times$ ULN). Furthermore, there was a difference in potential MI rates based on whether CKMB or troponin data were used for universal/ARC MI definition, with rates $>3\times$ as high for troponin compared with CKMB (18.7% versus 5.4%). At a threshold of $3\times$ ULN, 85.3% of patients were concordant for assessment of periprocedural MI by both CKMB and troponin.

Most prior stent trials have used the historic or modified WHO definition of periprocedural MI. The frequency of periprocedural MI in SPIRIT IV by this historic definition is consistent with other contemporary trials.13,14 There are limited data from other stent trials to compare periprocedural MI rates using the universal/ARC definition. In a pooled sample from older bare metal stent clinical trials, the frequency of MI was 8.3%, using a definition of CKMB $>3\times$ ULN,7 but these studies were conducted before routine loading of thienopyridines and common use of high-dose statin therapy, both of which have been associated with lower rates of periprocedural MI.15–17 In the more contemporary EVENT registry, using a definition of CKMB $>3\times$ normal, MI occurred in 6.6% of patients, with a rate of 3.7% among patients undergoing stenting for “on-label” indications and 9.0% among patients with stenting for “off-label” indication.18 Similarly, in the REPLACE 2 trial comparing heparin
Table 2. Analysis of Postprocedure Cardiac Biomarker Measurements*

<table>
<thead>
<tr>
<th>Variable</th>
<th>CKMB</th>
<th>Troponin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol MI (n=58)†</td>
<td>10.97</td>
<td>52.24</td>
</tr>
<tr>
<td>(Q1, Q3)</td>
<td>(6.76, 19.53)</td>
<td>(16.47, 203.88)</td>
</tr>
<tr>
<td>Universal/ARC MI (n=287)‡</td>
<td>3.66</td>
<td>9.42</td>
</tr>
<tr>
<td>(Q1, Q3)</td>
<td>(1.79, 7.25)</td>
<td>(5.30, 25.00)</td>
</tr>
<tr>
<td>CKMB &gt;3× ULN (n=167)</td>
<td>5.67</td>
<td>18.79</td>
</tr>
<tr>
<td>(Q1, Q3)</td>
<td>(3.97, 9.83)</td>
<td>(7.82, 69.00)</td>
</tr>
<tr>
<td>Troponin &gt;3× ULN (n=208)</td>
<td>2.56</td>
<td>9.96</td>
</tr>
<tr>
<td>(Q1, Q3)</td>
<td>(1.10, 7.25)</td>
<td>(5.57, 26.13)</td>
</tr>
</tbody>
</table>

CK indicates creatine kinase; MI, myocardial infarction; ARC, Academic Research Consortium; ULN, upper limit of normal.

‡Results of univariable regression.
†Protocol MI was adjudicated prospectively by an independent clinical events committee.
‡Universal/ARC MI was evaluated by 2 of the study authors (Drs Pervaiz and Cutlip). The No MI group consists of subjects without MI by either protocol or universal definition.

Figure 2. Histogram showing numbers and percentages of patients within each group defined by a range of biomarker ratios for creatine kinase (CKMB) and troponin. Ratios are based on maximum value normalized to individual clinical center upper limit of normal (ULN).

CMKB >3× ULN in 3.9% and troponin >3× ULN in 22.5%. One-year mortality was higher for patients with CMKB >5× normal but troponin at levels ≥10× normal was a not a predictor. Cavallini et al reported 2-year mortality among 2363 patients undergoing elective PCI with documented normal baseline CMKB and troponin and absent CMKB elevation after PCI. Troponin elevation >3× ULN as assessed by a central laboratory was present in 19.7% of patients and was associated with increased mortality (hazard ratio, 1.68; 95% confidence interval, 1.01–2.40) in crude analyses but was no longer significant after adjustment for baseline risk factors. A recent small study of 32 patients undergoing late gadolinium-enhanced cardiac MRI after PCI found that only 3 of 26 patients meeting the universal definition of MI based on troponin levels had imaging evidence of myocardial necrosis.22 In our study, less than one-third of patients had postprocedure troponin data available, whereas the majority (84%) had CKMB data available. Similar to these other studies, the adjudicated rate of universal/ARC MI based on troponin data was ≥3× higher compared with the rate based on CKMB data.

Because troponin was required only in setting of acute coronary syndrome in SPIRIT IV, these differences must be interpreted with some caution. In this regard, our results confirm prior reports of the importance of obtaining baseline troponin data if troponin is to be used to define periprocedural MI. There were 61 additional patients meeting criteria for

Table 3. Predictors of Periprocedural Protocol MI and Universal/ARC MI*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Protocol MI†</th>
<th>Universal/ARC MI‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR [95% CI]</td>
<td>OR [95% CI]</td>
</tr>
<tr>
<td>Total stent length</td>
<td>1.03 [1.01, 1.06]</td>
<td>1.03 [1.02, 1.04]</td>
</tr>
<tr>
<td></td>
<td>0.005</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No. of treated lesions, ≥2 vs single</td>
<td>1.15 [0.64, 2.06]</td>
<td>1.83 [1.41, 2.37]</td>
</tr>
<tr>
<td></td>
<td>0.64</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Ilb/IIa inhibitor use, yes vs no</td>
<td>1.88 [1.05, 3.36]</td>
<td>1.24 [0.92, 1.66]</td>
</tr>
<tr>
<td></td>
<td>0.03</td>
<td>0.16</td>
</tr>
<tr>
<td>Treatment, XIENCE V vs TAXUS</td>
<td>0.80 [0.47, 1.37]</td>
<td>0.75 [0.58, 0.97]</td>
</tr>
<tr>
<td></td>
<td>0.41</td>
<td>0.03</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; ARC, Academic Research Consortium; OR, odds ratio; CI, confidence interval; XIENCE V, everolimus-eluting stents; TAXUS, paclitaxel-eluting stents.

*The analysis was performed on single lesion–treated subgroup.
†Protocol MI was adjudicated prospectively by an independent clinical events committee.
‡Universal/ARC MI was evaluated by 2 of the study authors (Drs Pervaiz and Cutlip).

Table 4. Mortality at 30 Days, 1 Year, and 2 Years by Periprocedural MI Definition*

<table>
<thead>
<tr>
<th>Time</th>
<th>Protocol MI (n=58)</th>
<th>Universal/ARC MI (n=287)</th>
<th>No MI (n=2813)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-Cause Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 Days, n (%)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>3 (0.1)</td>
</tr>
<tr>
<td>1 Year, n (%)</td>
<td>0 (0)</td>
<td>2 (0.7)</td>
<td>29 (1.0)</td>
</tr>
<tr>
<td>2 Years, n (%)</td>
<td>0 (0)</td>
<td>4 (1.4)</td>
<td>65 (2.3)</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; ARC, Academic Research Consortium.

*Protocol MI was adjudicated prospectively by an independent clinical events committee. Universal/ARC MI was evaluated by 2 of the study authors (Drs Pervaiz and Cutlip). The No MI group consists of subjects without MI by either definition.
periprocedural MI by universal/ARC MI definition but who were determined to have baseline elevation (Figure 1). Among these 61 patients, 50 had elevated baseline troponin, whereas only 18 had baseline CKMB elevation. Of all the available baseline biomarker data, elevation was observed in 8.8% of troponin but in only 4.0% of CKMB. A report from the EVENT registry noted baseline troponin elevation in 6% of patients undergoing stenting for stable coronary artery disease.23 Using the more sensitive 99th percentile of upper reference limit, Prasad et al24 demonstrated baseline troponin elevation in 37% of patients undergoing nonemergent PCI.

Our study found no impact of periprocedural MI on mortality at 30 days, 1 year, or 2 years for either the protocol or universal/ARC MI definitions. This is in contrast to several earlier studies and adds to the controversy regarding the clinical significance of these events. Part of the explanation may be the low frequency of MI, especially large MI and those caused by procedure complications, as well as the low overall mortality rate, which may reflect improvements in safety of coronary stent procedures related to technique, second-generation drug-eluting stents, adjunctive pharmacological management, and exclusion of high-risk patients from enrollment. In addition, the exclusion of baseline elevation of CKMB or troponin in the evaluation of periprocedural MI may have had an effect on outcomes. The studies by Jeremias et al23 and Prasad et al24 cited above found that baseline elevation of troponin was an independent predictor of 1-year mortality, whereas isolated postprocedure elevation of either biomarker was not an independent predictor.

Implications
These results have several implications for future clinical trials of coronary stenting. Trials using the universal/ARC MI definition can be expected to have increased rates of MI compared with the historic definition using total CK. To the extent that troponin is used, these rates will be proportionately even higher. It should be reconsidered if troponin should be assessed at the same threshold as CKMB to define events of similar significance. These data suggest that a very sensitive definition of periprocedural MI (eg, troponin elevation >3× ULN in patients with baseline normal troponins) is not warranted because the present as well as prior studies suggest this is not of prognostic importance. Indeed, a diagnosis of MI in approximately 20% of patients offers little ability to discriminate differences in patient safety and may needlessly worry large numbers of patients and physicians. Periprocedural MI should be defined on the basis of a threshold that signifies a degree of myocardial necrosis that may be associated with adverse subsequent outcomes and allows discrimination of relative safety differences between comparative devices or pharmacological therapies. Large elevations of CKMB (approximately >8–10× normal), development of new Q waves, or smaller elevations associated with major procedural complications have demonstrated such prognostic significance. The appropriate threshold for troponin is not known and requires further study in a larger group of patients.

Limitations
This study is based on a clinical trial population that may not be representative of the general population undergoing stent procedures. MI rates will vary with different sampling intervals and among populations including more complex lesions and patients. The assessment of universal/ARC MI was performed retrospectively by 2 of the study authors (Drs Pervaiz and Cutlip). In the absence of baseline elevation of biomarker, we cannot exclude that subsequent elevations were not due to an acute event at time of presentation rather than PCI procedure. The number of MIs in the study is limited for assessment of risk factors for periprocedural MI by either definition. Limited numbers of large or complicated MIs and low mortality also limit inference regarding association of periprocedural MI and late mortality. There were also too few Q-wave MIs by the protocol definition (n=5) to determine their prognostic significance. Finally, the modest proportion of subjects with both CKMB and troponin data limits conclusions regarding the relative differences between these biomarkers in determination of MI and impact on late mortality.

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DISCLOSURES
Dr Hermiller has served as a consultant for Abbott Vascular and Boston Scientific Corp. Dr Wang has served on the medical advisory board for Boston Scientific Corp and as a speaker for Abbott Vascular and Boston Scientific Corp. Dr Applegate has served on advisory boards for and received research grants and honoraria from Abbott Vascular and St Jude Medical. Dr Kereiakes has served on scientific advisory boards for Abbott Vascular and Boston Scientific. Dr Sood, Dr Sudhir, Dr Hou, Kyoko Hattori, Xiaolu Su, Sherry Cao, and Dr Yaqub are employees of Abbott Vascular. Dr Stone has served as a consultant for Abbott Vascular, Boston Scientific, and Medtronic. Dr Cutlip has received institutional research support from Medtronic.

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