Coronary Stents and Noncardiac Surgery

John W. Riddell, MD; Laurence Chiche, MD; Benoît Plaud, MD; Martial Hamon, MD

**Case Presentation:** A 55-year-old man was admitted for investigation of a persistent cough. Chest x-ray revealed an opacity near the right upper-lobe bronchus. He was an ex-smoker, and his only previous medical history was of an acute coronary syndrome leading to the implantation of a drug-eluting stent (DES) in his proximal left anterior descending artery 3 months before this admission. A transbronchial biopsy was required, so a discussion ensued between the cardiology and the respiratory teams about whether to stop his oral antiplatelet agents (OAA), in this case, a combination of clopidogrel and aspirin.

**Noncardiac Surgery and Stent Thrombosis**

Interruption of OAA therapy, as often happens for noncardiac surgery, has been shown to be an important factor in stent thrombosis. The consequences of stent thrombosis are severe, with a 64% rate of death or myocardial infarction and a mortality rate of between 9% and 45%. In recent years, there has been an explosion in the use of DES, for which the risk of stent thrombosis, although likely to be similar to the standard bare-metal stent in the early phase, is less well defined owing to the longer, potentially indefinite period of time over which it may occur. Concern about the possible increased incidence of stent thrombosis with DES led to the publication of a consensus statement that highlighted the importance of not prematurely discontinuing dual OAA therapy and increasing its recommended duration in the case of DES to 1 year.

The timing and definition of stent thrombosis vary between studies, which has led to a call for standardizing definitions. In addition, although most of the trials have included low-risk patients and coronary lesions, the use of DES in the real world is much less controlled, potentially increasing the risk of stent thrombosis.

It has been well established that patients who undergo noncardiac surgery soon after stent implantation are at increased risk for stent thrombosis. The reasons for this increased risk may include nonendothelialization of the stent, interruption of dual OAA therapy, and the potentially prothrombotic state associated with surgery itself.

The risks of surgery after coronary stenting have been described in 8 observational, mainly retrospective, studies (summarized in Table 1). The mortality rate in such patients ranged from 2.5% to 21.4%.

Noncardiac surgery performed in patients who have had recent coronary stenting exposes them to an increased risk of major cardiac events in the perioperative period, especially if the OAA therapy is interrupted. Good theoretical reasons exist for delaying elective noncardiac surgery after coronary stenting, thereby allowing the stents to endothelialize.

**Likely Clinical Settings**

Two possible clinical scenarios exist for patients with stents who have noncardiac surgery. The first is the situation in which a patient requires noncardiac surgery and, as a result of a preoperative coronary risk assessment, undergoes an angiogram with subsequent stent implantation before the surgery is scheduled. The American College of Cardiology/American Heart Association guidelines clearly define patients who should undergo revascularization and, as a result of a preoperative coronary risk assessment, undergoes an angiogram with subsequent stent implantation before the surgery is scheduled. The American College of Cardiology/American Heart Association guidelines clearly define patients who should undergo invasive coronary assessment. However, the advice about who should receive revascularization predates the use of DES and is based on normal, nonpre-
operative practice. Therefore, some additional consideration may be warranted before preoperative revascularization is undertaken. In particular, consideration should be given to the urgency of the surgery and to the mode of revascularization, including, when applicable, the type of stent to be used. Since the publication of these guidelines, the Coronary Artery Revascularization Prophylaxis (CARP) trial, a randomized trial of coronary revascularization or not before vascular surgery, showed no difference in outcomes between groups. The study excluded patients with left main stem disease. The actual method of revascularization strategy was not randomized and was 60% percutaneous coronary intervention versus 40% CABG. A reanalysis of this trial comparing percutaneous coronary intervention with CABG suggested that percutaneous coronary intervention offered no benefit but that coronary artery bypass graft surgery (CABG) might. More recently, the Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echo V (DECREASE-V) pilot study, a randomized trial of coronary revascularization or not in high-risk patients with demonstrable ischemia, including 3-vessel disease and left main stem disease, before major vascular surgery, showed no benefit for the patients who underwent preoperative revascularization. Again, the method of revascularization strategy was not randomized and was 65% percutaneous coronary intervention versus 35% CABG. Although these data are limited, they suggest that preoperative revascularization in general is not beneficial. They also suggest that if preoperative revascularization is to be performed, methods that do not involve stenting such as balloon angioplasty or CABG may be preferable.

The second situation, which is becoming increasingly common, occurs when a patient with previous coronary stenting requires noncardiac surgery. In this situation, it is important to know the indication for stenting, the date of implantation, and the type(s) of stent(s) used, as well as the patient’s current OAA therapy and proposed duration. An assessment of the risks of the proposed surgery by the surgeon and anesthetist and of the likelihood

### Table 1. Coronary Stent Thrombosis and Noncardiac Surgery

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Type</th>
<th>Time Period</th>
<th>Patients, n</th>
<th>DES, %</th>
<th>Time From PCI to Surgery</th>
<th>Mortality Rate,* % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilson et al¹⁰</td>
<td>2003</td>
<td>Retr, NR</td>
<td>1990–2000</td>
<td>207</td>
<td>0</td>
<td>&lt;60 d</td>
<td>3.4 (1.2–6.3)</td>
</tr>
<tr>
<td>Sharma et al¹¹</td>
<td>2004</td>
<td>Retr, NR</td>
<td>1995–2000</td>
<td>47</td>
<td>0</td>
<td>&lt;90 d</td>
<td>18.4 (8.6–30.4)</td>
</tr>
<tr>
<td>Reddy et al¹²</td>
<td>2005</td>
<td>Retr, NR</td>
<td>1999–2004</td>
<td>56</td>
<td>0</td>
<td>...</td>
<td>8.6 (2.3–17.5)</td>
</tr>
<tr>
<td>Leibowitz et al²²</td>
<td>2006</td>
<td>Retr, NR</td>
<td>1995–2002</td>
<td>94</td>
<td>0</td>
<td>&lt;90 d</td>
<td>14.6 (8.1–22.4)</td>
</tr>
<tr>
<td>Vicenzi et al²³</td>
<td>2006</td>
<td>Prosp, NR</td>
<td>2001–2004</td>
<td>103</td>
<td>...</td>
<td>&lt;1 y</td>
<td>5.7 (1.8–11.1)</td>
</tr>
<tr>
<td>Compton et al¹⁴</td>
<td>2006</td>
<td>Retr, NR</td>
<td>2003–2006</td>
<td>38</td>
<td>100</td>
<td>...</td>
<td>2.5 (0.0–7.9)</td>
</tr>
<tr>
<td>Schouten et al¹⁵</td>
<td>2007</td>
<td>Retr, NR</td>
<td>1999–2005</td>
<td>192</td>
<td>52</td>
<td>&lt;2 y</td>
<td>3.1 (1.0–6.1)</td>
</tr>
</tbody>
</table>

PCI indicates percutaneous coronary intervention; Retr, retrospective; Prosp, prospective; and NR, nonrandomized.

*Mortality rates were calculated using the adjusted Wald interval.

### Table 2. Assessing the Risk of Surgery and Possible Stent Thrombosis

<table>
<thead>
<tr>
<th>Risk of Stent Thrombosis</th>
<th>High</th>
<th>Moderate</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stop all OAs</td>
<td>Continue at least 1 OAA if possible</td>
<td>Continue all OAs</td>
</tr>
<tr>
<td></td>
<td>Consider short-acting IV antiplatelet agents while off OAs</td>
<td>Consider short-acting IV antiplatelet agents while off OAs</td>
<td>Proceed with surgery</td>
</tr>
<tr>
<td></td>
<td>Proceed with surgery</td>
<td>Proceed with surgery</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Restart OAs as soon as possible after surgery</td>
<td>Restart OAs as soon as possible after surgery</td>
<td>...</td>
</tr>
<tr>
<td>Moderate</td>
<td>Stop all OAs</td>
<td>Continue 1 OAA if possible</td>
<td>Continue all OAs</td>
</tr>
<tr>
<td></td>
<td>Proceed with surgery</td>
<td>Proceed with surgery</td>
<td>Proceed with surgery</td>
</tr>
<tr>
<td></td>
<td>Restart OAs as soon as possible after surgery</td>
<td>Restart OAs as soon as possible after surgery</td>
<td>...</td>
</tr>
<tr>
<td>Low</td>
<td>Stop all OAs</td>
<td>Stop all OAs</td>
<td>Continue 1 OAA if possible</td>
</tr>
<tr>
<td></td>
<td>Proceed with surgery</td>
<td>Proceed with surgery</td>
<td>Proceed with surgery</td>
</tr>
<tr>
<td></td>
<td>Restart OAs as soon as possible after surgery</td>
<td>Restart OAs as soon as possible after surgery</td>
<td>...</td>
</tr>
</tbody>
</table>

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and importance of possible stent thrombosis, preferably in conjunction with a cardiologist (ideally the cardiologist who implanted the stent), then needs to be undertaken. As an aid to this assessment, we have adapted a table from Albaladejo et al and incorporated the latest recommendations (Table 2). This table and the accompanying algorithm (the Figure) are based on consensus opinion and therefore may change as further data become available.

When possible, surgery should be delayed until the patient is outside the recommended period of dual antiplatelet therapy, as determined by the stent and lesion characteristics. This would mean that surgery should be delayed until 6 weeks after implantation of a bare-metal stent and 1 year after implantation of a DES. In reality, the recommendation for a 6-week delay with a bare-metal stent is to ensure that the patient completes a 4-week course of dual antiplatelet therapy because the patient will need 5 to 10 days (depending on the platelet half-life) for the effect of the antiplatelet agents to wear off before surgery. Patients in whom noncardiac surgery is carried out within these time periods are deemed at high risk of stent thrombosis. Patients in whom noncardiac surgery is performed outside these time periods may still be at high risk of stent thrombosis, depending on factors related to their coronary anatomy and their clinical characteristics. Stent thrombosis is more likely to occur in patients who have had stenting of ostial lesions, bifurcation lesions, lesions in small vessels, multiple lesions, or long lesions. In addition, patients with diabetes mellitus or renal impairment or patients in whom the indication for stenting was an acute myocardial infarction or an acute coronary syndrome are at higher risk of stent thrombosis.

When assessing the bleeding risk for an operative procedure, one needs to consider not only the actual blood loss associated with the procedure in terms of anemia and need for transfusion but also the consequences of the bleeding in terms of its site. For instance, even minor bleeding may be intolerable during or after certain ophthalmologic and intracranial surgery. Often, the decision to operate while antiplatelet agents are continued will be a matter of personal judgment. For many operative procedures, there is no evidence and no recommendations to aid in this decision. Dual OAA therapy usually consists of aspirin and a thienopyridine (most frequently clopidogrel). It is generally accepted that there are more perioperative bleeding problems with thienopyridines than aspirin; therefore, if 1 OAA is stopped, it is generally the thienopyridine.

When possible, OAAs should be continued throughout the perioperative period, and if stopped, they should be stopped for as short a period as possible before surgery and restarted as soon as possible after surgery. In addition, for patients at high risk of stent thrombosis, consideration should be given to substituting oral agents with a shorter-acting, intravenous glycoprotein IIb/IIIa inhibitor during the perioperative period. However, the evidence for this use of alternative antiplatelet agents is largely anecdotal. In patients for whom noncardiac surgery is planned soon but preoperative revascularization is considered...
essential, consideration should first be given to modes of revascularization that do not involve stents such as CABG\textsuperscript{23} or balloon angioplasty,\textsuperscript{26} although the latter may necessitate stenting if there is a complication. If stenting is deemed necessary, there may be a place for newer stents that encourage rapid endothelialization,\textsuperscript{27} thereby possibly allowing surgery to be performed earlier. However, there is no evidence yet that these theoretical benefits will be realized. Along the same lines, absorbable stents are currently in development.\textsuperscript{28} However, although these theoretical benefits will be realized. Along the same lines, absorbable stents are currently in development.\textsuperscript{28} However, although they may remove the risk of late thrombosis, they probably will not solve the problem of the risk of early surgery. If, despite all of the above precautions, stent thrombosis should occur during the perioperative period, the correct treatment is almost certainly urgent percutaneous coronary intervention\textsuperscript{29} because recent surgery usually rules out the use of thrombolytic therapy.

**How Should We Manage Our Patient’s Antiplatelet Therapy?**

Our patient’s clopidogrel was discontinued for 5 days before the biopsy and restarted with a loading dose of 300 mg on the first postoperative day. Aspirin was continued throughout.

**Conclusions**

The management of the increasingly common and complex group of patients with coronary stents who require noncardiac surgery requires a multidisciplinary approach to avoid potentially catastrophic outcomes. We have outlined some guidelines to aid in the decision-making process that should be undertaken before such patients are scheduled for surgery.

**Disclosures**

None.

**References**


**KEY WORDS:** angioplasty, stents, surgery, thrombosis
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Circulation. 2007;116:e378-e382
doi: 10.1161/CIRCULATIONAHA.107.726992
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2007 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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World Wide Web at:
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