To Clot or Not to Clot, that is the Question: ASA/Plavix in the Perioperative Period

Lisa T. Newsome, MD, DMD
Somnia, Inc

Recent PCI with stenting combined with discontinuation of antiplatelet therapy confers significant morbidity and mortality during noncardiac surgery. Because stent endothelialization may be incomplete, sudden discontinuation of clopidogrel and aspirin combined with the prothrombotic state induced by surgery increases the risk of acute perioperative stent thrombosis and abrupt vessel closure, leading to significant morbidity and mortality (1-3) (Figure 1). Despite the 2002 AHA/ACC guidelines which recommended a 4-6 week interval between bare metal stenting (BMS) and noncardiac surgery “to allow 4 full weeks of dual-antiplatelet therapy and re-endothelialization of the stent to be completed, or nearly so,” reports of perioperative morbidity and mortality continued to be published (3). The most powerful predictor of acute stent thrombosis in BMS is a time delay of <14 days between implantation and interruption of dual-antiplatelet therapy (4). The current 2007 ACC/AHA Perioperative Guidelines state bare metal stent thrombosis is exceedingly rare more than 4 weeks after insertion (5). However, Doyle et al. found a 2% cumulative incidence of BMS thrombosis at 10 years, which was increased among patients considered “off-label” for drug-eluting stent (DES) use (P=0.024). Very late BMS thrombosis (>12 months) was also associated with increased risk of death (P<0.001) (6). However, the authors did not mention whether any of these cases occurred perioperatively.

Numerous publications of perioperative morbidity and mortality in patients with DES coupled with clinical and pathological reports of incomplete stent endothelialization suggest acute stent thrombosis, myocardial infarction (MI), and death may be more prevalent than previously thought with these devices, particularly when dual-antiplatelet therapy is interrupted perioperatively (3,4). Artang and Dieter reviewed 36 cases of late stent thrombosis in patients receiving DES and found a strong association between late stent thrombosis (>30 days after deployment) and cessation of dual-antiplatelet therapy (7). Overall, 55% of patients discontinued both clopidogrel and aspirin treatment, and 86.3% of patients stopped clopidogrel after the initial recommended duration for dual-antiplatelet therapy (3 months for sirolimus-eluting stents; 6 months for paclitaxel-eluting stents). If clopidogrel alone was discontinued, the median time to an adverse clinical event was 30 days. In comparison, if both aspirin and clopidogrel were stopped, the median time to an adverse clinical event was 7 days (P<0.0001). Forty-two percent of events occurred in relation to a surgical procedure in which dual-antiplatelet therapy or clopidogrel alone were discontinued. The morbidity and mortality rates were 92% and 8%, respectively. There was no difference in occurrence between sirolimus- and paclitaxel-eluting stents. The authors recommended the perioperative continuation of aspirin.

Patients with coronary stents, particularly DES, who subsequently present for noncardiac surgery, pose a particular challenge for clinicians during the perioperative period. Clinicians must balance the risks of 1) discontinuing antiplatelet agents and increasing the possibility of perioperative stent thrombosis, MI, and cardiac death against 2) continuing clopidogrel and aspirin, thus increasing the potential for surgical bleeding, which in certain cases, may be life-threatening. Patients who prematurely discontinue dual-antiplatelet therapy have higher rates of re-hospitalization and mortality when compared with those who continue therapy (8). Surgery performed early after DES implantation is associated with a significantly increased incidence of perioperative MI and death, regardless if clopidogrel and aspirin are continued (9,10). However, a patient may complete the recommended 12-month duration of antiplatelet therapy and still be at risk for perioperative stent thrombosis, MI, and death. Some institutions treat patients with dual-antiplatelet therapy for 12-24 months, and in cases where additional stent complexities and comorbidities exist, clopidogrel and aspirin are continued indefinitely (9,11). This complicates management since 60-70% of patients are receiving DES for “off-label” or unapproved use (3,12,13-16). Chassot
et al. contend, based on the currently available data, the risks of withdrawing patients from antiplatelet agents are greater than continuing them, imposing a perioperative cardiac death rate that is increased 5- to 10-times (17).

Surgical intervention creates a prothrombotic and proinflammatory state conducive to the development of perioperative stent thrombosis, as illustrated in Figure 1 (18,19). Autopsy results have shown these mechanisms are responsible for at least half of all perioperative infarctions (3,20). Despite this milieu, surgeons often stop all antiplatelet drugs preoperatively—regardless of their patients’ comorbidities—in order to minimize intraoperative bleeding.

Withdrawal of oral antiplatelet agents is known to be an independent predictor of mortality in patients with ACS and those at risk for coronary artery disease (CAD) (21,22). Abrupt cessation of aspirin results in a rebound phenomenon whereby both cyclooxygenase-1 and thromboxane B2 (the product of thromboxane A2 [TxA2] hydrolysis) levels increase rapidly, not returning to baseline for 3-4 days (23). Complete recovery of platelet function occurs in 50% of patients by day 3, and 80% of patients by day 4 (24). These patients subsequently generate increased levels of thrombin and decrease fibrinolysis further enhancing platelet aggregation and worsening the risk for perioperative stent thrombosis, MI, and death. Collet et al. prospectively studied 1358 patients admitted with ACS and found a 2-fold increase in both death and death/MI among recent withdrawers compared with chronic users and nonusers (25). Of this group, 57.5% had known CAD, and 64% had discontinued aspirin for scheduled surgery. Multivariate analysis found aspirin withdrawal to be a strong independent predictor (OR=2.02, P=0.003) of mortality and death/MI at 30 days. Aspirin interruption was also found to be an independent predictor for bleeding events (OR=2.6, P<0.01). Ferrari et al. found, in 383 patients with established CAD hospitalized with recurrent ACS, 13.3% of events occurred 10.9±1.9 days (range 4-17 days) after abrupt aspirin withdrawal (26). Ten (20%) patients developed thrombosis of a BMS implanted 15.5±6.5 months earlier, which accounted for 50% of the ST-segment elevation MIs (STEMIs) diagnosed. Aspirin was interrupted in 20 patients (40%) for minor surgery or dental treatment. Biondi-Zoccai et al. performed a meta-analysis of 50,279 patients at risk for CAD and found aspirin non-adherence/withdrawal was associated with a 3-fold higher risk of death and MI (OR=3.14, P=0.0001) (22). The risk was significantly higher in patients with intracoronary stents (OR=89.78, P<0.001). Although the data from these studies are not specifically from perioperative patients, it is likely applicable. The loss of aspirin’s protective effect during the hypercoagulable surgical state confers an increased risk of stent thrombosis not fully appreciated by clinicians.

Recent studies suggest clopidogrel may provide anti-inflammatory protection, further attenuating the thrombotic process (27). Abrupt withdrawal may result in a proinflammatory and prothrombotic state (28). After 12 months of dual-antiplatelet therapy in diabetics with DES, significant increases in platelet aggregation (P<0.0001) and inflammatory biomarkers (P<0.05 for C-reactive protein, P<0.001 for P-selectin) were measured one-month after clopidogrel withdrawal (29). This may have serious perioperative implications, particularly for surgical patients with additional risk factors for stent thrombosis.

**Impact of Aspirin and Clopidogrel on Perioperative Bleeding**

The impact of aspirin on surgical bleeding has been primarily studied in cardiac and vascular surgery (30-34). While preoperative aspirin may increase chest tube drainage and re-exploration rates in cardiac surgery, these clinical endpoints were observed with larger doses (≥325 mg), prolonged duration of cardiopulmonary bypass, lack of antifibrinolytic use, and emergent/urgent surgery without a difference in operative mortality rates (35,36). In patients undergoing “off-pump” CABG, there was no difference in blood loss between aspirin users and nonusers (37). In vascular surgery, perioperative aspirin significantly improves long-term peripheral bypass graft patency, and appears to have a protective effect against transient ischemic attacks and stroke in patients undergoing carotid endarterectomy (32,33). Burger et al. performed a review and meta-analysis of the surgical and interventional literature to determine the risks of low-dose aspirin withdrawal versus the bleeding risks associated with aspirin continuation (34). Aspirin withdrawal preceded 10.2% of acute cardiovascular syndromes (MI, stroke, peripheral arterial occlusion, cardiac death). Although aspirin increased the incidence of bleeding by a factor of 1.5, it did not increase the severity or perioperative morbidity/mortality, except in intracranial surgery and, possibly, transurethral prostatectomy. The authors recommended discontinuing aspirin only if the risk of bleeding complications exceeds the cardiovascular risks.
of aspirin withdrawal. In their review, Merritt and Bhatt concluded aspirin monotherapy should be continued in elective noncardiac surgery (38).

The likelihood of increased bleeding and/or an increased requirement for blood transfusion in patients undergoing major noncardiac surgery while taking clopidogrel has largely been inferred from the cardiac surgical literature, which contains conflicting data (39). Patients who remain on clopidogrel and aspirin while undergoing CABG, particularly within 4 days of the scheduled procedure, have a significantly higher incidence of perioperative bleeding, re-exploration, blood-component transfusion, and extended intensive care/hospital stays (40-44). Although studies of on- and off-pump CABG reported significantly increased blood component-transfusion rates without increased morbidity/mortality in patients receiving clopidogrel, Koch et al. reported significant reductions in both early and long-term survival in patients receiving a perioperative blood transfusion with CABG (41,45,46).

There is little evidence to define the true impact of continuing thienopyridines on bleeding in noncardiac surgery, and the information available remains anecdotal and inconsistent (38,47). When compared to aspirin alone, the combination of clopidogrel and aspirin increases the absolute risk of major bleeding by 0.4-1.0% (48-50). In a multicenter registry, Vichova reported an 18.6% postoperative bleeding complication rate; aspirin and clopidogrel had been withheld in 26% and 24% of patients, respectively (51). Schouten et al. found transfusion was required in 24% of patients continuing and 20% of patients who discontinued antiplatelet therapy (P=0.50) (52). There was no difference in the number of units transfused between the two groups. In their review, Chassot et al. reported perioperative clopidogrel use increases surgical bleeding and transfusion rates by 50% without concomitant increased morbidity and mortality, except in intracranial surgery (17). In procedures where blood loss can be easily controlled, there may be no indication to stop antiplatelet drugs (39,53).

Despite concerns regarding perioperative bleeding, data suggest postoperative clopidogrel confers a protective effect against MI, stroke and death. The Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events (CAPRIE) trial indicates clopidogrel monotherapy was more effective than aspirin alone in reducing the combined risk of ischemic stroke, MI, and vascular death in high-risk patients with previous CABG (relative risk reduction 8.7%) (54,55). Fewer gastrointestinal side effects were observed with clopidogrel than with aspirin monotherapy.

**Current Recommendations for Perioperative Management of Patients with Coronary-Artery Stents**

The 2007 AHA/ACC/Society for Cardiovascular Angiography and Interventions/ American College of Surgeons/American Dental Association Science Advisory concluded premature discontinuation of dual-antiplatelet therapy markedly increases the risk of catastrophic stent thrombosis, MI and death (5,39). They recommend postponing all elective procedures for which there is a significant risk of bleeding until dual-antiplatelet therapy is completed (Table 1) (39). However, if patients with DES are “to undergo subsequent procedures that mandate discontinuation of thienopyridine therapy, aspirin should be continued if at all possible and the thienopyridine restarted as soon as possible after the procedure because of concerns about late stent thrombosis” (39). Aspirin should also be continued perioperatively for patients with BMS (39). Similar recommendations have been published in the ACC/AHA 2007 Perioperative Guidelines for Noncardiac Surgery and the 2009 American Society of Anesthesiologists Practice Alert for the Perioperative Management of Patients with Coronary Stents (5,17,56,57). However, Chassot and others contend dual-antiplatelet therapy is the cornerstone for stent thrombosis prevention, and the risk of discontinuing clopidogrel and aspirin preoperatively outweighs the benefit of reduced hemostasis, especially in patients with procedural complexities and comorbidities which place them at higher risk for developing stent thrombosis (17,58). The authors emphasized the importance of continuing aspirin throughout the perioperative period, except in instances when surgery is performed in a closed space (intracranial surgery, posterior chamber of the eye, spinal surgery in the medullary canal) (17).

Although case reports and series of perioperative management of patients with DES have been published, no universally accepted guidelines exist. Many advocate simply the perioperative continuation of clopidogrel and aspirin whenever possible (3,13). Others have recommended the substitution of non-selective non-steroidal anti-inflammatory drugs (NSAIDs) and low molecular weight heparin (LMWH) in surgical procedures with excessive hemorrhagic risk (59,60). However, this practice is controversial and there is no scientific evidence to support their efficacies in
preventing perioperative stent thrombosis as reports of ACS have been reported with this practice (3,5,17,25,59-61). The concomitant use of non-selective NSAIDs and aspirin significantly increases cardiac morbidity and mortality in patients with CAD and the incidence may be even higher in patients with coronary stents (3,62). Non-selective NSAIDs competitively inhibit aspirin binding to the serine residue at position 530 by binding to the catalytic site of cyclooxygenase-1 (3).

Although heparin therapy is often used perioperatively for thromboembolic prophylaxis, it does not possess any antiplatelet properties and is not protective against stent thrombosis (3,63). In fact, Vicenzi et al. described an association between perioperative heparin therapy and increased cardiac morbidity and mortality among patients with coronary stents (1). During UFH infusion, increases in thrombin and platelet activity have been measured and persist many hours after an infusion is discontinued, while any protective anticoagulant effect declines rapidly due to the short half-life of UFH (64,65). Webster et al. found the administration of UFH significantly and transiently increases platelet aggregation despite chronic aspirin therapy (150 mg/day) in patients undergoing carotid endarterectomy or lower extremity angioplasty, persisting into the immediate postoperative period (66). Abrupt cessation of enoxaparin results in rapid increases in prothrombotic activity with maximum levels measured 12 and 24 hours after discontinuation (67). Minor elevations in platelet activation are associated with LMWH (65).

Brilakis et al. recently summarized treatment options for patients with DES: 1) continue dual-antiplatelet therapy throughout the perioperative period for patients at low risk of bleeding; 2) implement “bridging therapy”, in which a short-acting GP IIb/IIIa inhibitor (tirofiban or eptifibatide) or thrombin inhibitor, or both, is substituted for clopidogrel during the perioperative period; or 3) discontinue clopidogrel preoperatively, restarting it as soon as possible postoperatively (58,68). Although empiric and without evidence-based data supporting its efficacy, multiple institutions utilize bridging therapy to prevent perioperative stent thrombosis (8,17,39,61,69,70). Glycoprotein IIb/IIIa inhibitors have been favored since this platelet receptor is the pivotal mediator for platelet aggregation and thrombus formation (71).

More recently published protocols, including from the Cleveland Clinic, recommend bridging therapy with GP IIb/IIIa inhibitors primarily 1) in patients who have not completed dual-antiplatelet therapy and 2) in patients whose stent complexities and comorbidities significantly increase their risk for developing catastrophic stent thrombosis and its sequelae (13,17,70). Tirofiban and eptifibatide are administered parenterally, have half-lives <2 hours, and are eliminated by renal clearance (72). The infusion rate is reduced by 50% in patients with reduced renal function (serum creatinine >2.0 mg/dl or creatinine clearance <50 ml/min). Platelet function returns to 60-90% of normal after the infusion is stopped for 6-8 hours. Figures 2 and 3 present management algorithms based on the currently available literature.

Reversible P2Y12 receptor antagonists are undergoing clinical trials, and may prove to be of value perioperatively (3,73-77). Cangrelor is a parenteral, reversible direct P2Y12 inhibitor whose half-life of 5-9 min allows 100% recovery of platelet function one hour after the infusion is discontinued (73,74). An infusion of 4 µg/kg/min achieves complete platelet inhibition when measured at 4 minutes (75). AZD6140 is an oral, reversible direct P2Y12 receptor antagonist. It provides more rapid and complete platelet inhibition than clopidogrel (73,76). AZD6140 has a half-life of 12 hours, making it efficacious in the perioperative setting (76). Current trials have found similar rates of bleeding (3).

Although success with bridging therapy has been reported, prospective studies are necessary to validate it as a viable management strategy. Opponents argue bridging therapy is 1) expensive, 2) logistically difficult, 3) exposes patients to risks associated with a prolonged hospitalization, and 4) confers no protection against intraoperative stent thrombosis (3,58). Resuming clopidogrel or a GP IIb/IIIa inhibitor as soon as possible postoperatively is paramount to protecting against stent thrombosis when the risk is greatest (3,58). Brilakis et al. recommend a postoperative 600 mg loading dose of clopidogrel, which reduces 1) the time to achieve maximal platelet inhibition (2 versus 6 hours with a 300 mg loading dose), and 2) the frequency of hyporesponsiveness to clopidogrel, particularly in patients whose platelets are activated secondary to surgical intervention (58,68,78-80). However, anesthetic drugs metabolized by CYP3A4 may irreversibly inhibit this isoenzyme and prevent the conversion of clopidogrel to its active state, modulating its antiplatelet effect (81-83). Midazolam irreversibly inactivates CYP3A4 after metabolism to 1-
hydroxymidazolam (81,82). Midazolam also exerts antiplatelet activity, the mechanisms of which are not fully elucidated; whether this counteracts clopidogrel modulation is unknown (84,85). Competitive (reversible) inhibitors – drugs that may not prevent clopidogrel activation – of CYP3A4 include fentanyl, alfentanil, and propofol (86-88).

If a patient presents for surgery with aspirin and clopidogrel inadvertently stopped by their surgeon or another physician, some advocate administering 325 mg of non-enteric coated aspirin the day of surgery, and delaying the procedure until later that day (70,89). Theoretically, the patient should have antiplatelet effects within 2 hours secondary to the rapid absorption of aspirin (70,89,90). A single dose of 160 mg has been shown to completely eliminate platelet TxA₂ production; however, this may not be the case in patients with aspirin resistance (62,90-93). Others have suggested administering aspirin 325 mg for 3-5 days in order to achieve a steady-state, which may overcome issues with resistance (3,70).

When stent thrombosis occurs, it acutely manifests as a STEMI or a sudden malignant dysrhythmia, and must be treated with immediate reperfusion to avoid a transmural myocardial infarction due to the abrupt interruption of coronary blood flow in a myocardial region that is neither collateralized nor preconditioned by recurrent chronic ischemia (58,68). Thrombolytic therapy (intravenous or intracoronary) is significantly less effective than PCI in treating stent thrombosis and restoring myocardial perfusion (58,94). Administration of thrombolytic therapy is often prohibitive in the perioperative period. Therefore, primary PCI is the definitive treatment for perioperative stent thrombosis, and restoration of coronary stent patency (3,17,58,70,95-97). Surgical procedures should be performed in institutions where 24-hour interventional cardiology is available to provide immediate and emergent intervention (3,17,70,95-97). Percutaneous coronary intervention carries an increased risk of bleeding when performed early after surgery because antiplatelet and antithrombin agents must be administered during the procedure (1,58).

Postoperative management should include admission to a higher-acuity unit with continued ECG monitoring and cardiology surveillance (68,96). Routine monitoring of cardiac biomarkers would be useful in detecting myocardial injury, recurrent ischemia, and for risk stratification, and should be drawn prior to emergent transfer to the cardiac catheterization laboratory (17). Elevated perioperative troponin levels are statistically significant independent predictors of morbidity and mortality one year after surgery (97). However, the occlusive nature of stent thrombosis, and ongoing myocardial necrosis, may quickly lead to hemodynamic instability, ventricular arrhythmias, cardiogenic shock, or cardiac arrest, necessitating emergent PCI (97).

Considerations for Regional Anesthesia for Patients with Coronary-Artery Stents

In patients with coronary-artery stents, particularly DES, the use of regional anesthesia (RA) must be carefully considered. Regional anesthesia, particularly neuraxial blockade, attenuates the hypercoagulable perioperative state by blunting the sympathetic response (98,99). Systemic absorption of local anesthetics provides antiplatelet effects by blocking TxA₂ and decreasing platelet aggregation (100-102). These benefits may be advantageous, and RA may seem the safest choice in certain situations (3). However, the potential for stent thrombosis with discontinuation of antiplatelet drugs and potential coagulation abnormalities must be taken into account when considering RA, particularly in patients considered higher-risk (13,17).

It is generally interpreted from the 2003 American Society of Regional Anesthesia (ASRA) guidelines the thienopyridines and dual-antiplatelet therapy are contraindications to neuraxial anesthesia or peripheral nerve blockade in noncompressible regions that cannot be observed for bleeding (103). Following the guidelines confers no guarantee neuraxial anesthesia will be free from bleeding complications (3,103). In fact, only about one-third of patients who developed neuraxial hematoma in a large series of spinal and epidural anesthetics had any coagulation abnormality (104). Aspirin alone does not appear to increase the risk of neuraxial hematoma, and should not interfere with the performance of neuraxial blockade (103,105). For patients receiving bridging therapy with eptifibatide, 8 hours must lapse before a neuraxial blockade can be performed (3,103,106).

Although perioperative platelet transfusions have been suggested in patients on dual-antiplatelet therapy when RA is considered safest, this practice cannot be justified (70,96,107-109). Theoretically, apheresis platelets administered to patients with stents who then receive clopidogrel and aspirin may not develop antiplatelet effects to provide
adequate protection from stent thrombosis for hours to days (70). The administration of platelets should probably be avoided except in instances of life-threatening bleeding (6,70). If platelet administration is considered absolutely necessary, Doyle et al recommend waiting 12 hours (3 half-lives) after the last dose of clopidogrel (half-life = 4 hours) when serum levels of the drug are no longer detectable to ensure normal platelet function (6). However, Cornet et al. published a case series of 3 patients with gastrointestinal bleeding or who were scheduled for emergency surgery and who received platelet transfusions shortly after bare-metal stent insertion (108). Dual-antiplatelet therapy was discontinued in one patient 14 hours prior to transfusion, while the other 2 patients remained on clopidogrel and aspirin. In all 3 patients, stent thrombus formation with donor platelets occurred in both the presence and absence of dual-antiplatelet therapy, suggesting therapeutic serum levels of clopidogrel and aspirin may not affect transfused platelets. Moreover, the thrombogenic surfaces of stents may attract and activate donor platelets to a even greater extent than endogenous platelets, further increasing the risk of stent thrombosis, MI, and death (70,108).

A further dilemma with RA, particularly neuraxial blockade, in patients with stents is that postoperative PCI cannot be delayed to allow for catheter removal and prevent spinal cord compromise. Performance of neuraxial instrumentation, whether a single-shot technique or involving catheter insertion, significantly increases the risk of a neuraxial hematoma in patients who must subsequently receive antiplatelet/antithrombotic therapy during PCI for acute stent thrombosis (3). Indwelling catheters should not be removed in the presence of therapeutic anticoagulation (103). If a surgical patient requires PCI, catheters should be removed prior to antithrombotic/antiplatelet/thrombolytic therapy, and PCI must be undertaken urgently. Vigilant and intensive monitoring of sensorimotor function should be performed to detect any evidence of spinal cord compromise. The appropriate time delay between catheter removal and clopidogrel administration remains undefined. There are no guidelines for catheter removal preceding antiplatelet/antithrombotic administration (103,110). Douketis et al. recommend administering clopidogrel or GP IIb/IIIa inhibitors 2-3 hours after epidural catheter removal, although longer time delays have been suggested (108). Longer time delays increase the risk and complications of postoperative stent thrombosis if clopidogrel is withheld; this must be a mutual decision between the anesthesiologist and cardiologist. There are no guidelines regarding peripheral nerve blockade and catheters. Ultrasound-guided blockade with and without catheter placement may be safest in preventing potential bleeding complications, particularly in the setting of dual-antiplatelet therapy (111). Based on the current information available, the decision to perform RA should be made case-by-case with consideration given to all potential complications (96,108).

**References:** Upon request to the Author

**Table 1. Duration of Antiplatelet Therapy and Timing of Noncardiac Surgery**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Timing of Noncardiac Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilatation without stenting</td>
<td>2-4 weeks of dual-antiplatelet therapy</td>
</tr>
<tr>
<td>PCI and BMS</td>
<td>4-6 weeks minimum of dual-antiplatelet therapy</td>
</tr>
<tr>
<td>PCI and DES</td>
<td>12 months of dual-antiplatelet therapy</td>
</tr>
</tbody>
</table>

In patients in whom coronary revascularization with PCI is appropriate for mitigation of cardiac symptoms and who need elective noncardiac surgery in the subsequent 12 months, a strategy of balloon angioplasty or bare-metal stent placement followed by 4 to 6 weeks of dual-antiplatelet therapy is probably indicated.

Aspirin: lifelong therapy, whichever is the revascularization technique

(From 2007 AHA/ACC Science Advisory and Society of Cardiovascular Angiography DES Task Force Recommendations for Timing of Noncardiac Surgery after PCI and 2007 ACC/AHA Recommendations for

Figure 1 - Diagram of the pathophysiology of acute perioperative stent thrombosis.
Figure 2 - Proposed Algorithm for Perioperative Management of Patients with Bare-Metal Stents Based on Current Literature.

- Perform surgery where 24 hour interventional cardiology coverage is available
- Restart clopidogrel/ aspirin as soon as possible after surgery
Figure 3 - Proposed Algorithm for Perioperative Management of Patients with Drug-Eluting Stents Based on Current Literature

- Perform surgery where 24 hour interventional cardiology coverage is available
- Restart clopidogrel/aspirin as soon as possible after surgery
- Please refer to Table 4 for additional risk factors for stent thrombosis