# ANESTHESIA FOR PATIENTS WITH CORONARY STENTS FOR NON CARDIAC SURGERY

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## INTRODUCTION

Approximately 5% of patients with intracoronary stents will undergo non cardiac surgery within the first year after stenting, and an increasing number will continue to present for surgery thereafter. As the success of the stents requires long-term antiplatelet therapy, management of patients with these devices poses a dilemma to the anesthesiologist. Discontinuation of antiplatelet therapy relatively soon after PCI (percutaneous coronary intervention) with stenting confers significant mortality during non cardiac surgery. As stent endothelialization may not yet be complete at the time of surgery, abrupt discontinuation of antiplatelets combined with the prothrombotic state induced by surgery increases the risk of acute perioperative stent thrombosis (Fig.1) and myocardial infarction. Continuation of antiplatelet medications may be associated with an increased risk of intraoperative bleeding and also prevent administration of regional anesthesia.

As per the 2007 Focused Update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention, all post-PCI stented patients receiving a DES (drug eluting stent), clopidogrel 75 mg daily should be given for at least 12 months if patients are not at high risk of bleeding. For post-PCI patients receiving a BMS (bare metal stent), clopidogrel should be given for a minimum of 1 month and ideally up to 12 months (unless the patient is at increased risk of bleeding; then it should be given for a minimum of 2 weeks). *(Level of Evidence: B)*

They also recommend postponing all elective procedures for which there is a significant risk of bleeding until dual-antiplatelet therapy is completed (Table 1).

Aspirin is continued throughout the perioperative period, except in instances where surgery is performed in closed space (intracranial surgery, posterior chamber of the eye, spinal surgery in the medullary canal).

The substitution of non selective NSAIDs and LMWH for dual-antiplatelet therapy is controversial and there is no scientific evidence to support their efficacies in preventing perioperative stent thrombosis. The concomitant use of non selective NSAIDs and aspirin significantly increases cardiac morbidity and mortality in patients with coronary artery disease and the incidence may be even higher in patients with coronary stents. Although heparin therapy is often used perioperatively for thromboembolic prophylaxis, it does not have antiplatelet properties and is not protective against stent thrombosis.

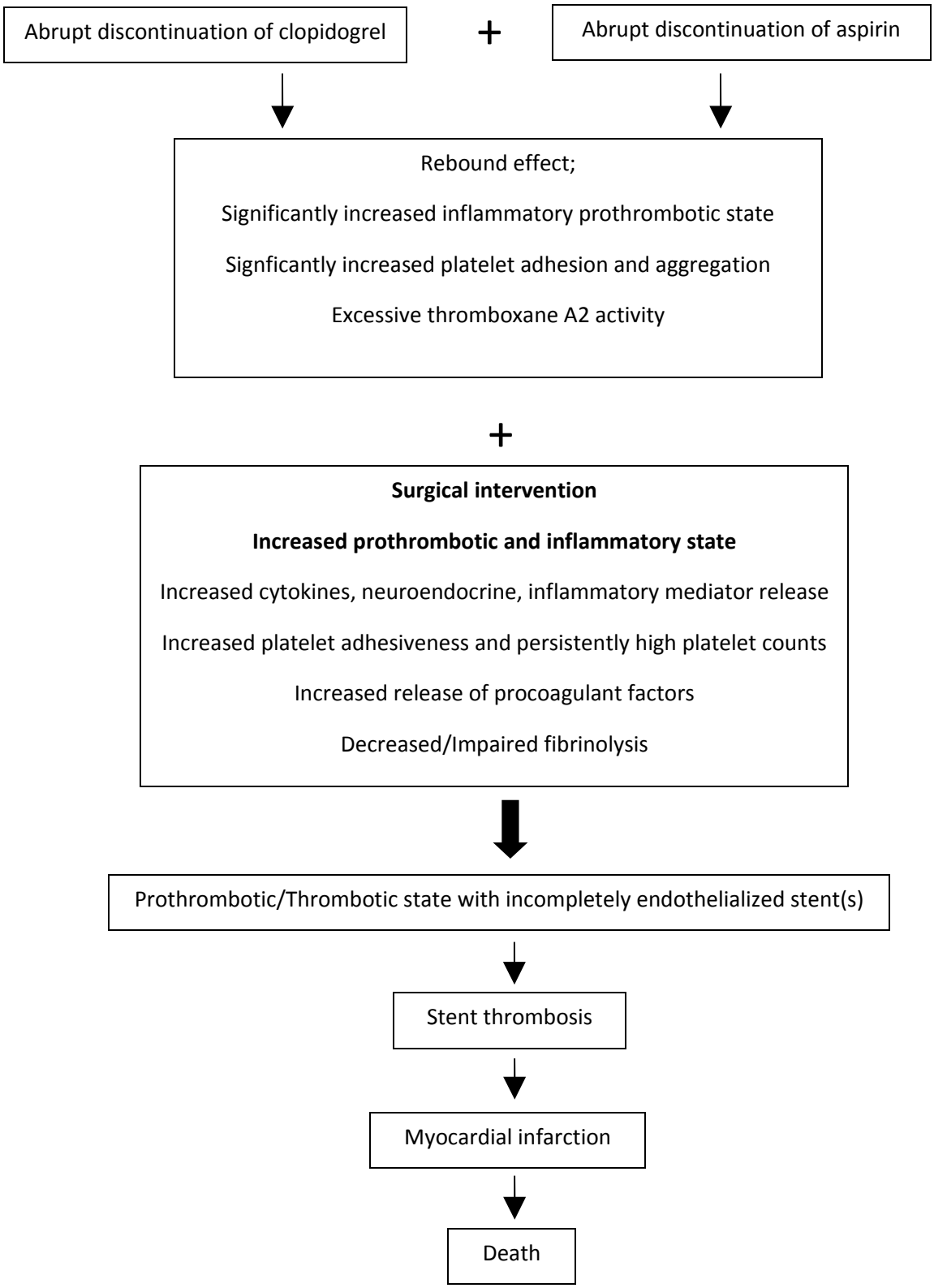


*Fig.1. Diagram of the pathophysiology of acute perioperative stent thrombosis*

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| **Dilatation without stenting**: 2 – 4 week of dual-antiplatelet therapy |
| Surgery postponed for 2 – 4 week (vital surgery only) |
| **PCI and BMS**: 4 – 6 week minimum of dual- antiplatelet therapy |
| Elective surgery postponed ≥ 6 week, but not for more than 12 week, when restenosis may begin to occur |
| **PCI and DES**: 12 months of dual-antiplatelet therapy |
| Elective surgery postponed for ≥ 12 months |
| In patients in whom coronary revascularization with PCI is appropriate for mitigation of cardiac symptoms and who need elective non cardiac surgery in the subsequent 12 months, a strategy of balloon angioplasty or BMS placement followed by 4 to 6 weeks of dual-antiplatelet therapy is probably indicated |
| Aspirin: Lifelong therapy, whichever is the revascularization technique |

*Table 1. Duration of antiplatelet therapy and timing of noncardiac surgery*

To summarize 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. GP IIb/IIIa inhibitors have been favored since this platelet receptor is the pivotal mediator for platelet aggregation and thrombus formation. They are more potent than the combination of aspirin and a theinopyridine. GP IIb/IIIa inhibitors are recommended as bridging therapy 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. Tirofiban and eptifibatide are administered parenterally, have half lives < 2 h, and are eliminated by renal clearance. Platelet function returns to 60% - 90% of normal after the infusion is stopped for 6 – 8 h.

*Fig.2. Algorithm for perioperative management of patients with bare metal stents*

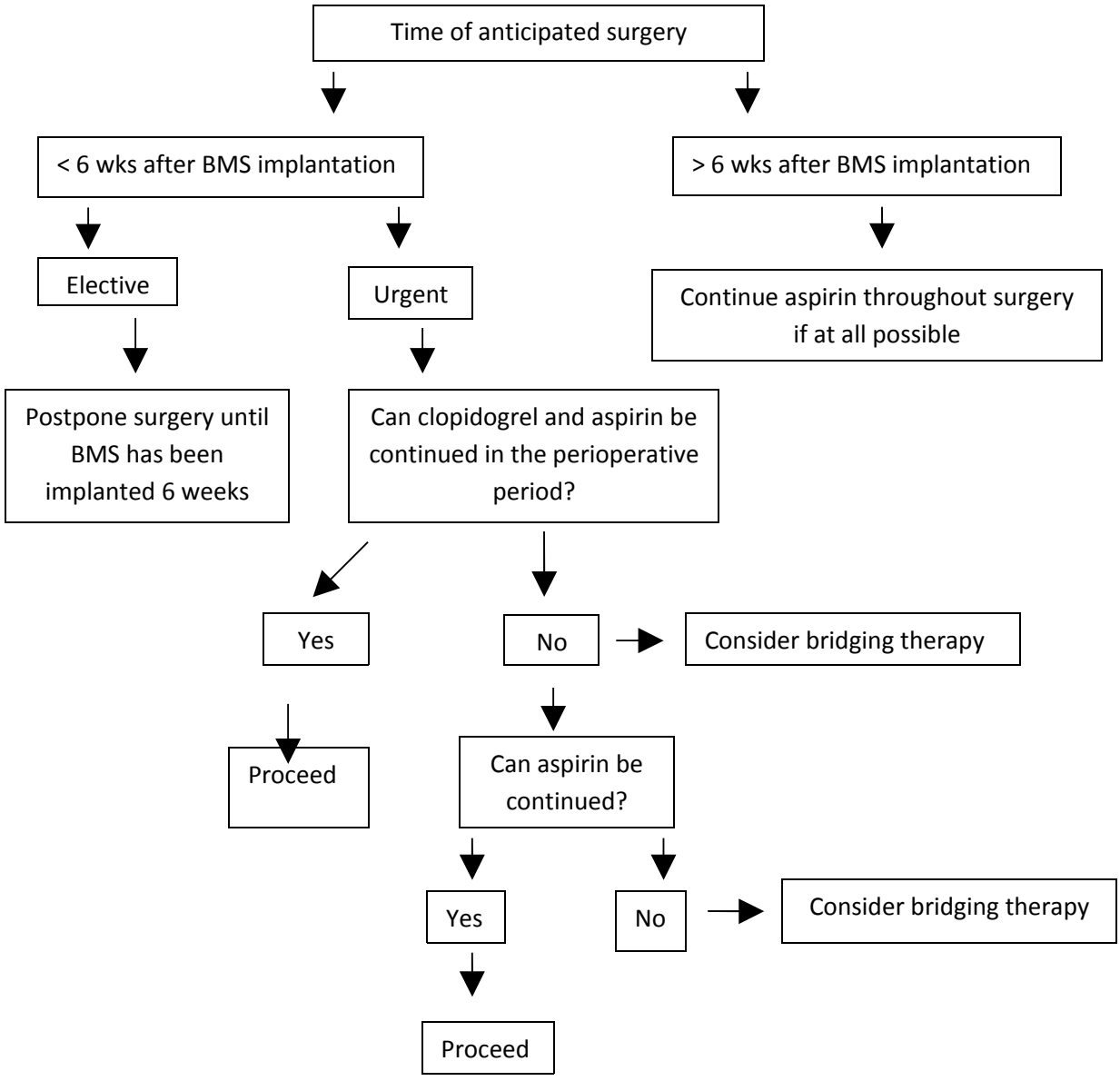
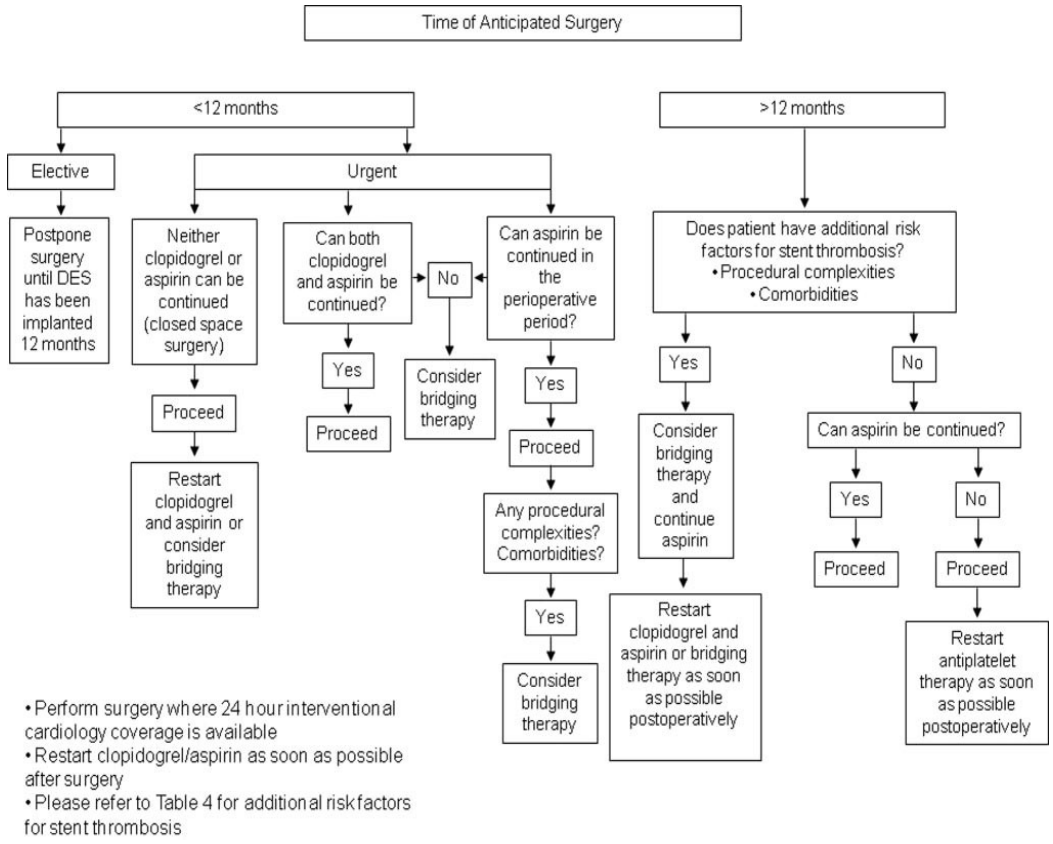


*Fig.3. Algorithm for perioperative management of patients with drug eluting stents*




Management of stent thrombosis:

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 MI due to the abrupt interruption of coronary blood flow in a myocardial region that is neither collateralized nor preconditioned by recurrent chronic ischemia. Thrombolytic therapy (IV or intracoronary) is significantly less effective than PCI in treating stent thrombosis and restoring myocardial perfusion. 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. Surgical procedures should be performed in institutions where 24-h interventional cardiology is available to provide immediate and

emergent intervention. Postoperative management should include admission to a higher-acuity unit with continued electrocardiogram monitoring and cardiology surveillance.

Regional anesthesia (RA):

Neuraxial blockade attenuates the hypercoagulable perioperative state by blunting the sympathetic response. Systemic absorption of local anesthetics provides antiplatelet effects by blocking TxA2 and decreasing platelet aggregation. These benefits of regional anesthesia are advantageous in patients with intracoronary stents. ASRA recommendations have to be followed to decide when RA can be administered in patients on antiplatelets.

The role of perioperative platelet transfusions in patients on dual-antiplatelet therapy when RA is considered cannot be justified. Ex vivo studies have shown that transfused platelets may not be inhibited by the presence of adequate serum levels of antiplatelet drugs. 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.

CONCLUSION

The management of patients with coronary artery stents during the perioperative period is an important safety issue. Communication between the patient's cardiologist, surgeon and anesthesiologist is essential to minimize the risk of catastrophic stent thrombosis.