

Perioperative Cardiovascular Risk Evaluation and Care for Noncardiac Surgery – Part II

BY MARIE-CLAUDE PARENT, MD, MSc, FRCPC AND STÉPHANE RINFRET, MD, MSc, FRCPC

Systematic perioperative cardiovascular risk evaluation for noncardiac surgery is essential. Indeed, patients who undergo this type of surgery are at a substantial risk for cardiac events. Annually, 500,000 to 900,000 patients worldwide will experience a major perioperative cardiac complication, such as death, myocardial infarction (MI) or nonfatal cardiac arrest.¹ The number of patients undergoing noncardiac surgery is constantly increasing and patients with coronary artery disease (CAD) live longer; as a result, they are likely to require all types of surgical operations.^{1,2}

Another critical aspect to consider is that of informed consent. The patient has the right to know the level of risk associated with a specific surgical procedure, even more so when surgery is elective and does not affect vital prognosis. Appropriate risk evaluation also makes it easier for the physician to make certain decisions (eg, choice of the operative procedure, intensity of postoperative care, etc).

This issue of *Anesthesiology Rounds* is published in two parts and attempts to answer the following questions: In a given clinical context, how can one carry out the best possible perioperative risk evaluation? Which patients benefit from a more thorough investigation of their cardiovascular state? When should one consider revascularization? For which patients should β -blockers be prescribed in preparation for surgery? Are there other agents that could reduce the risk? Having discussed the diagnosis and stratification of cardiovascular risk in the first part, this second part deals with the prevention of cardiac events, antiplatelet therapy management, and the administration of β -blockers, statins, and/or acetylsalicylic acid (ASA - Aspirin®).

THE PREVENTION OF CARDIAC EVENTS

Revascularization (either percutaneous or surgical with coronary artery bypass surgery or CABG) must be performed only if it is justified *independently* of a need to prepare for noncardiac surgery. The decision to perform preoperative revascularization must take into account the expected risks vs the benefits. At the present time, the role of preoperative revascularization is in itself questioned. Specifically, the Coronary Artery Revascularization Prophylaxis (CARP) trial, published in 2004 by McFalls et al³ evaluated the impact of coronary revascularization before major elective vascular surgery. The results revealed a similar postoperative increase of troponin I in both groups (11.6%, revascularization group vs 14.3%, control group; $P=0.37$). There was no difference in intrahospital mortality or at 2.7 years (22%, revascularization group vs 23%, control group; $P=0.92$).

Several criticisms were raised regarding this study, mainly because its size was limited and a number of high-risk patients were eventually excluded. Nevertheless, the CARP trial strongly questions the impact of preoperative revascularization on early postoperative outcome and on long-term mortality.

Committee for Continuing
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University of Montreal

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Another recent pilot study, the Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echo (DECREASE) V trial, selected high-risk patients (≥ 3 risk factors) scheduled to undergo vascular surgery.⁴ Only patients whose performance test indicated extended ischemia were randomized, either into the invasive approach group (coronary angiography and revascularization) or in the medical therapy alone group (without coronary angiography). All received β -blockers. No significant difference (mortality and MI) was observed between the 2 groups at 30 days and at 1 year.

These data suggest that revascularization is beneficial only for a carefully selected patient population. Currently, the preoperative role of percutaneous coronary intervention (PCI) seems limited to patients with unstable and active coronary heart disease (CHD); eg, MI with or without ST segment elevation or unstable angina. In such a situation, a strategy using angioplasty alone or angioplasty with the insertion of a bare metal stent is preferred.⁵ Prophylactic revascularization is not recommended for patients with asymptomatic ischemia or stable angina (Class I or II of the Canadian Cardiovascular Society [CCS]), unless the coronary anatomy presents a high risk. It is not clear whether patients with Class III angina according to the CCS classification would benefit from PCI. The majority of evidence available to date seems to suggest that preoperative revascularization does not modify risk in this patient population.⁵

ANTIPLATELET THERAPY MANAGEMENT

Antiplatelet therapy management represents a challenge in the context of upcoming noncardiac surgery. In fact, in patients who underwent PCI, early interruption of dual antiplatelet therapy is associated with a significant risk of stent thrombosis. In studies evaluating drug-eluting stents, thrombosis is associated with a 20%-45% mortality rate, obviously a catastrophic clinical outcome.⁶⁻⁸ A recent meta-analysis revealed an incidence of early thrombosis (ie, within 30 days of implantation) of 4.4/1000 (0.44%) with drug-eluting stents and 5/1000 (0.5%) with bare metal stents ($P=0.74$).⁹ The incidence of late thrombosis (ie, > 30 days after implantation) reached 5/1000 for drug-eluting stents vs 2.8/1000 for bare metal stents ($P=0.22$). The median occurrence time for thrombosis was 15.5 months for the sirolimus-eluting stent (173-773 days) and 18 months for paclitaxel-eluting stents (40-548 days). As for bare metal stents, the median was between 3.5 and 4 months. Very-late thrombosis, ie, over a year after implantation, appears to occur exclusively with drug-eluting stents. This risk was evaluated at 5/1000 (0.5%) for drug-eluting stents vs 0% for bare metal

stents (relative risk [RR] 5.02; $P=0.02$). Clearly, these recent data affect the desired length of antiplatelet combination therapy.

In the case of an angioplasty performed with a balloon catheter alone, it is recommended to continue the preoperative administration of ASA and to delay surgery for 2 to 4 weeks. It has been demonstrated that postponing noncardiac surgery for >8 weeks increases the risk of restenosis in the treated vessel.¹⁰

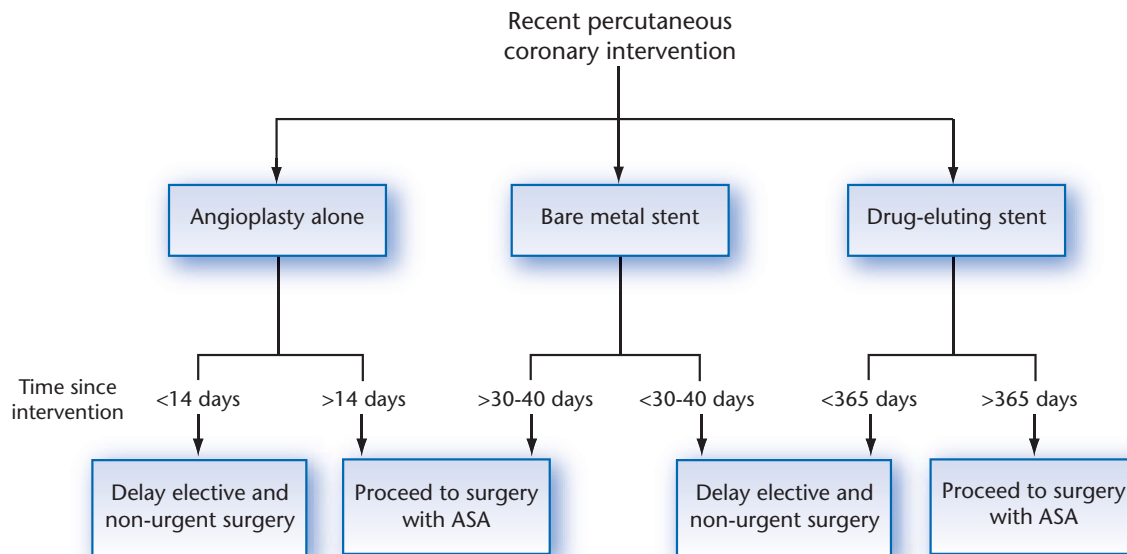
The use of drug-eluting stents should be avoided as much as possible in the preoperative setting. Guidelines currently support at least 1 month of antiplatelet combination therapy (ASA and a thienopyridine) for bare metal stents and at least 1 year of therapy for drug-eluting stents.⁵ The early interruption of antiplatelet medication is a major risk factor for thrombosis with all types of stents, but particularly for late thrombosis with drug-eluting stents. In the setting of an upcoming noncardiac surgery, the implantation of a bare metal stent is clearly preferred, provided it is clinically justified outside the perioperative setting.

Other risk factors are associated with an increased risk of stent thrombosis, namely a history of stent thrombosis, multiple stents, a stent of considerable length, or when the stent is implanted at a bifurcation, the presence of incompletely revascularized 3-vessel disease, and diabetes or heart failure with impaired ejection fraction.¹¹

Operative risk management and care must take into account the risk of bleeding associated with the continuation of antiplatelet therapy vs the risk arising from a potential stent thrombosis after interruption of antiplatelet therapy. One study found that the Aspirin[®] and clopidogrel combination therapy increased the absolute risk of a major bleed from 0.4% to 1.0%.¹² In order to choose the best possible option, it is crucial to promote discussion among the various decision makers, ie, the cardiologist, the surgeon, and the anesthesiologist. The patient must then be informed of the suggested approach.

Should antiplatelet combination therapy be suspended prematurely after the implantation of a drug-eluting stent, the thrombotic risk becomes very high. It is strongly recommended to at least maintain ASA. Taking into account platelet lifespan (7 days), a maximal interruption of 5 days for clopidogrel could be considered, in order to maintain some antiplatelet effect. Conversely, a complete interruption of at least 5 days is needed in order to avoid hemorrhagic complications (risk of major bleeding or major complications of even a minor bleeding event, eg, in neurosurgery). Antiplatelet combination therapy should be resumed as soon as possible after surgery.⁵ Perioperative stent thrombosis frequency is currently

FIGURE 1: Proposed approach to the management of patients with percutaneous coronary intervention prior to a noncardiac surgical procedure



unknown. Recent guidelines, based on expert recommendations, have suggested an antiplatelet therapy management strategy for patients with previous PCI requiring noncardiac surgery (Figure 1).⁵

ADMINISTRATION OF β -BLOCKERS

There is much controversy surrounding the perioperative use of β -blockers. In fact, the role β -blockers play in low- or intermediate-risk patients is not well defined. Few studies have evaluated the significance of drug titration for a given heart rate and determined which β -blocker should be preferred. The optimal starting time and length of use also remain undetermined. Theoretically, β -blockers present several benefits. They enhance the reduction of myocardial O_2 demand, prolong diastolic filling time, reduce arrhythmias, and provide protection against plaque rupture in a setting of activation of the sympathetic nervous system.

Table 1 summarizes the methodology and results of 5 randomized controlled studies addressing the use of β -blockers published in recent years.¹³⁻¹⁷ An observational study used the Lee index to assess the efficiency of β -blockers with respect to risk.¹⁸ Higher risk patients benefited from the use of β -blockers, whereas patients with a lower risk (< 2 risk factors) seemed to be disadvantaged by them.

Despite the variability encountered in the results of the various studies, current recommendations suggest the use of β -blockers in intermediate- to high-risk patients. This would include patients with ≥ 2 or 3 risk factors.^{5,19} It also seems preferable to

initiate treatment several days or weeks before surgery and to titrate the dose to reach a heart rate of 50-60 beats/min. Cardioselective and prolonged action drugs may be more effective; therefore, atenolol or bisoprolol could be recommended. Currently, there are no data specifying the optimal duration of treatment that can vary between 1 month postoperatively to indefinitely, if there is an underlying CHD.

Table 2 summarizes the latest recommendations published in 2007 by the ACC/AHA regarding the use of β -blockers.⁵

Very recently, the results of the PeriOperative Ischemic Evaluation (POISE) trial were presented; they question the benefit and even the safety of preoperatively administered β -blockers.²⁰ POISE was a randomized, double-blind clinical trial that included 4,174 patients in the metoprolol group (orally, 200 mg/day of the sustained-release [SR] formula) and 4,177 patients in the placebo group. Previous CAD was found in 43% of patients, arterial vascular disease (AVD) in 41% of patients and a history of stroke (cerebrovascular accident [CVA]) in 15% of patients. Vascular surgery was the type of surgery performed in 42% of patients, intraperitoneal surgery in 22% of patients, and orthopedic surgery in 21% of patients. The incidence of primary outcome (combination of cardiovascular mortality, MI or cardiac arrest) was lower in the metoprolol group vs the placebo group (5.8% vs 6.9%, hazard ratio [HR] 0.83; 95% confidence interval [CI], 0.70-0.99; $P=0.04$). This positive outcome was primarily

TABLE 1: Methodology and results of major randomized controlled trials assessing the perioperative use of β -blockers

	Mangano et al. ¹³	DECREASE ¹⁴	MaVS ¹⁵	DIPOM ¹⁶	POBBLE ¹⁷
STUDY DESIGN	<ul style="list-style-type: none"> • 200 known CAD or risk patients undergoing noncardiac surgery • Atenolol vs placebo • 10 mg IV dose immediately before and after surgery • Subsequently, 50-100 mg PO die for length of hospital stay 	<ul style="list-style-type: none"> • 112 patients undergoing vascular surgery • ≥ 1 risk factor and evidence of reversible ischemia at stress echocardiography • Bisoprolol preoperatively (initiated ≥ 7 days before) vs standard care • Preoperative target HR 60 bpm and < 80 bpm intra- and postoperatively • Patients with extensive ischemia, suspected 3 vessel disease or left main = excluded 	<ul style="list-style-type: none"> • 497 patients • Vascular surgery • Metoprolol vs placebo • Initiated 2 hours before surgery and continued $\times 5$ days 	<ul style="list-style-type: none"> • Patients with Type 2 diabetes undergoing major noncardiac surgery • 921 patients aged > 39 y • Metoprolol 100 mg die (SR) vs placebo • Therapy initiated 24 hours before surgery and to 8 days postoperatively 	<ul style="list-style-type: none"> • 103 patients undergoing infrarenal vascular surgery • Median age 73 y • Metoprolol 50 mg PO bid vs placebo • Therapy initiated from admission to 7 days postoperatively
CONCLUSIONS	<ul style="list-style-type: none"> • Overall mortality significantly lower in atenolol group at 6 months (0% vs 8%), 1 year (3% vs 14%) and 2 years (10% vs 21%) • Benefit related mainly to reduction of cardiac cause mortality during the first 6-8 months • Results become non-significant if inclusion of all deaths 	<ul style="list-style-type: none"> • Reduction of cardiac mortality incidence or MI at 30 days (3.4% vs 34%) • 90% relative reduction of risk... non-plausible according to several experts! 	<ul style="list-style-type: none"> • Lack of difference in mortality at 30 days (10% vs 12% placebo) 	<ul style="list-style-type: none"> • Assessment of all-cause mortality, acute MI, unstable angina or heart failure (21% metoprolol vs 20% placebo; HR 1.06, 95% CI, 0.8-1.41) • All-cause mortality 16% in both groups • Limitations: short treatment duration, no titration for HR, protocol violations 	<ul style="list-style-type: none"> • Cardiovascular events occurred in 32 patients at 30 days (MI, VT, CVA, death) • 17 patients (32%) metoprolol group vs 15 patients (34%) placebo group (NS) • Significant reduction of the mean hospitalization time by 2 days

Mangano DT, et al. *N Engl J Med.* 1996; Poldermans D, et al. *N Engl J Med.* 1999; Yang H, et al. *Can J Anaesth.* 2004; Juul AB et al. *BMJ.* 2006; Brady AR, et al. *J Vasc Surg.* 2005. CAD = coronary artery disease; CVA = cerebrovascular accident; MI = myocardial infarction; HR = heart rate; bpm = beats/minute; SR = sustained release; VT = ventricular tachycardia.

driven by a diminution in nonfatal infarcts (3.6% vs 5.1%; HR 0.70; $P=0.0007$). However, total mortality increased in the metoprolol group (3.1% vs 2.3%; HR 1.33; $P=0.03$), as were CVAs (1.0% vs 0.5%; HR 2.17; $P=0.005$). Patients in the metoprolol group also presented with more hypotension (15.0% vs 9.7%; $P<0.0001$)

and significant bradycardia (6.6% vs 2.4%; $P<0.0001$).

A major criticism of this study is that the dose of metoprolol was possibly too high with no consideration of blood pressure. Nevertheless, these results remain very troubling and could lead to modifications of the ACC/AHA guidelines.

TABLE 2: Level of recommendations for beta-blocker use according to the ACC/AHA 2007⁵

	Low cardiovascular risk (no risk factor)	Intermediate cardiovascular risk (≥ 1 risk factor)	High cardiovascular risk or CAD	Patients already treated with β -blockers
Vascular surgery	Class IIb	Class IIb	Class I (if presence of ischemia at stress test) Class IIa (If CAD+)	Class I, evidence level: B
Intermediate/high-risk surgery	Insufficient data	Class IIb	Class IIa	Class I, evidence level: C
Low-risk surgery	Insufficient data	Insufficient data	Insufficient data	Class I, evidence level: C

Class I: Evidence that therapy is beneficial; Class II: Conflicting evidence / Differences of opinions; Class IIa: Evidence / opinion in favor of beneficial effect; Class IIb: Efficiency / Evidence not as well established; Class III: evidence / general opinion that therapy is deleterious.

ADMINISTRATION OF STATINS

Statin administration could be beneficial perioperatively in vascular surgery and could reduce the risk of perioperative mortality. The anti-inflammatory property of statins could be the operative factor, in addition to the effects of statins on plaque stabilization and thrombogenesis reduction.²¹ Cohort studies and larger clinical trials will be needed in order to validate the positive effect of statins, although it is obviously an interesting treatment to consider. New recommendations still specify that statins should be continued perioperatively, if the patient already takes one type of statin (Class I), and that the use of a statin is reasonable in patients undergoing vascular surgery (Class IIa). Finally, for patients with at least 1 clinical risk factor undergoing an intermediate-risk surgery, the use of a statin should be considered (Class IIb).⁵

USE OF ASA

A meta-analysis compared ASA to placebo in vascular surgery (infrainguinal bypass). A tendency towards a reduction of events was observed, but the results were not statistically significant.²² In contrast, the Pulmonary Embolism Prevention (PEP) trial suggested that there was an increase in the risk of cardiac ischemic events with the use of ASA in hip fracture patients.²³ The objective of the PEP trial was to demonstrate that ASA could prevent the occurrence of pulmonary embolism and deep venous thrombosis in orthopedic surgeries. Overall, very few cardiovascular events were observed and the statistical power to detect the impact of ASA on the occurrence of such events was therefore reduced.

On the other hand, in cases of carotid endarterectomy, the guidelines published in 2004 by the American College of Chest Physicians (ACCP) recommended the initiation of ASA preoperatively.²⁴ The ideal timing to initiate ASA in other types of vascular surgeries remains unclear. According to a recent review, ASA as monotherapy should not be discontinued routinely in cases of elective noncardiac surgeries.²⁵ For the time being, it is the clinician's task to assess whether the potential benefits outweigh the risk of bleeding secondary to ASA when treatment is continued in the perioperative period.

CONCLUSIONS

Patients no doubt benefit from the adequate assessment of cardiac risk. This allows the clinician to obtain an informed consent that is all the more important, since most noncardiac

surgeries are elective and rarely affect vital outcome. The presence of risk factors should be evaluated together with the patient's functional performance status and the type of surgery considered. The use of noninvasive tests are preferred in patients scheduled for high-risk surgery, such as vascular surgery, if test results are likely to influence therapy. Depending on the intensity of detected anomalies or symptoms, coronary angiography should be considered together with revascularization, but only if this is beneficial for the patient, independently of the upcoming noncardiac surgery. In cases of lower risk surgeries, the use of functional tests to predict risk should be limited to patients presenting with a very high risk of cardiac complications.

Finally, the administration of a β -blocker should be considered, especially in high-risk patients or patients with known CAD who are undergoing vascular or high- to intermediate-risk surgery and those already treated with β -blockers. In patients with lower risk, there is insufficient evidence to recommend initiation of β -blockers. Furthermore, in light of the recent results of the POISE study, high doses of β -blockers cannot be recommended. Iatrogenic hypotension associated with β -blockers is definitely to be avoided. Statins are a promising option (although not yet validated) to reduce the risk of perioperative cardiac events. The results of clinical trials exploring the use of statins in the perioperative period will have to be favorable before their use can be recommended specifically in this context.

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Drs. Parent and Rinfret have stated that they have no disclosures to announce in association with the contents of this issue.

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This publication is made possible by an educational grant from
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