Guidelines

for

Pre- operative Cardiovascular Assessment

By

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Clinical Standards & Guidelines

2009 AD 2709 K

Kurdistan Board
For Medical Specialties

Bordi Kurdistan Bo Psporayati Pziski

Setting Clinical and Professional Excellence

Surgery increases myocardial oxygen demand & causes alterations in the balance between prothrombotic and fibrinolytic factors, potentially resulting in increased coronary thrombogenicity. These factors, together with patient position, fluid balance, temperature shift, bleeding, and type of anaesthesia, may all contribute to hemodynamic derangements, leading to myocardial ischaemia and heart failure.
General, regional, and neuraxial anaesthesia differ in terms of the stress response evoked by surgery.
Surgical interventions can be broadly divided into low-risk, intermediate-risk and high-risk groups, with estimated 30-day cardiac event rates (cardiac death and myocardial infarction) of 1%, 1 – 5%, >5%, respectively (Table.1).
Surgical factors that influence cardiac risk are related to the urgency, invasiveness, type and duration of the procedure, as well as the change in body core temperature, blood loss, and fluid shifts; all clarified by the surgical Apgar score (Table.2).

Functional capacity
Determine functional capacity which is measured in metabolic equivalents (METs) using exercise testing or the ability to perform daily activities.
▪ *taking care of self, such as eat, dress, or use the toilet (1 MET)*
▪ *walking up a flight of steps or a hill (4 METs)*
▪ *doing heavy work: scrubbing floors, lifting or moving heavy furniture (4 - 10 METs).*
▪ *participating in strenuous sports (>10 METs)*
Inability to climb two flights of stairs or to run a short distance equal <4 METs indicating poor functional capacity and is associated with poor prognosis

Cardiac risk factors
The Lee risk index model & the NSQIP model provide complementary prognostic perspectives as cardiac-risk prediction index in non-cardiac surgery to help the clinician in the decision-making process:
1. *The Lee index* or 'revised cardiac risk index designed to predict post-operative myocardial infarction, pulmonary oedema, ventricular fibrillation or cardiac arrest, and complete heart block.
It comprises six variables: type of surgery, history of IHD, history of heart failure, history of cerebrovascular disease, pre-operative treatment with insulin and pre-operative creatinine >170mmol/L= >2 mg/dL or creatinine clearance of < 60ml/min/1.73m^2).
2. *NSQIP index* involves five predictors of peri-operative myocardial infarction/cardiac arrest: age, type of surgery, functional status, elevated creatinine (>130mmol/L= >1.5 mg/dL) and the American Society of Anesthesiologists (ASA) class:
Class I: patient is completely healthy;
Class II: patient has mild systemic disease;
Class III: patient has severe systemic disease that is not incapacitating.
Class IV: patient has incapacitating disease that is a constant threat to life.

Class V: moribund patient who is not expected to live for 24 hours with/out the surgery.
ClassV1: declared brain-dead patient whose organs being removed for donor purposes.
The NSQIP index performed better than the Lee risk index; but some peri-operative cardiac complications such as pulmonary oedema and complete heart block were not considered in the NSQIP model, by contrast, the Lee index allows estimation of the risk of peri-operative pulmonary oedema, complete heart block, myocardial infarction, death.

Biomarkers

Cardiac troponins (T & I) sensitive, specific & are independent of and complementary to other important cardiac indicators of risk (ST deviation and LV function).
Assessment of cardiac troponins before and 48–72 hours after major surgery is imperative but the diagnosis of non-ST-segment elevation myocardial infarction should never be made solely on these biomarkers.

B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) are produced in cardiac myocytes in response to increases in myocardial wall stress & occur at any stage of heart failure, independently of the presence or absence of myocardial ischaemia & are considered as important prognostic indicators.

Non-invasive testing

provides information on three cardiac risk markers: LV dysfunction, myocardial ischaemia and heart valve abnormalities.

● ECG (with/out treadmill test), echocardiography and radionuclide ventriculography aimed to detect myocardial ischaemia, evaluate blood pressure & heart rate response & provide an estimate of functional capacity.

● Myocardial perfusion imaging is well established pre-operative risk stratifications in patients with limited exercise capacity using pharmacological stress (dipyridamole, adenosine, or dobutamine); it has high negative predictive value; but low positive predictive value.

Imaging stress testing is recommended before intermediate or high risk surgery in those with poor functional capacity (<4 METs) and clinical cardiac risk factors. (Table.3).

● Cardiovascular magnetic resonance (CMR) imaging can be used for detection of ischaemia; both perfusion and wall motion can be detected during stress and at rest with high sensitivity and specificity. Computed tomography with angiography can be used to detect coronary calcium. Both CMR & CT angiography are used for selected cases only for preoperative assessment.

Risk-reduction strategies: Pharmacological

● ß-blocker: used to decrease myocardial oxygen consumption by reducing heart rate, leading to a longer diastolic filling period and decreased myocardial contractility.
Peri-operative *continuation* of ß-blockers is recommended in patients currently receiving this medication. Pre-operative *initiation* of ß-blockers is considered in patients scheduled for high risk surgery with ≥ 2 clinical risk factors or ASA status (American Society of

Anesthesiologists) ≥3 & in patients who have known IHD or myocardial ischaemia.
When oral ß-blockade is initiated in patients who undergo non-cardiac surgery, use as a first choice preferably atenolol or bisoprolol, starting at a low dose with target of a resting heart rate 60–70 bpm, systolic blood pressure >100 mm Hg and intra-operative mean arterial pressure should remain >55 mm Hg. Treatment should ideally be initiated between 30 days and (at least) 2 days before surgery, and should be continued post-operatively for several months. However; for patients testing positive for pre-operative stress, long-term therapy should be used.

● Peri-operative continuation of statins is recommended. Pre-operative initiation of statin therapy should be considered in patients undergoing vascular surgery, ideally at least 2 weeks before surgery.

● Continuation of ACEIs or ARBs, in stable patients with heart failure and LV systolic dysfunction during non cardiac surgery should be considered with close monitoring. Initiation of ACEIs or ARBs should be considered at least 1week before surgery in cardiac stable patients with heart failure and LV systolic dysfunction.
Consider transient discontinuation of ACEIs or ARBs 24 hrs before surgery in hypertensive patients especially with concomitant ß-block and should be resumed after surgery as soon as blood volume and pressure are stable

● Although heart rate-reducing calcium channel blockers (diltiazem or verapamil) are not indicated in patients with heart failure and systolic dysfunction, the initiation or continuation of these drugs may be considered in patients who do not tolerate ß-blockers and in patients with vasospastic angina.

● Alpha$_2$ receptor agonists (e,g clonidine) should not be administered to patients undergoing non-cardiac surgery as it may increase the risk of hypotension and non-fatal cardiac arrest.

● Diuretics for hypertension &/or heart failure should be continued to the day of surgery and resumed orally when possible with close monitoring of volume status, fluid end electrolyte balance (precisely potassium level) in the peri-operative period.

● The use of low-dose aspirin or dual anti-platelet therapy in patients undergoing non-cardiac surgery should be individualized weighing the peri-operative bleeding risk against the risk of thrombotic complications.
To reduce risk of bleeding especially in patient with stent, it is recommended to delay elective non-cardiac surgery (minimum of 4 weeks and ideally for up to 3 months) if possible until completion of the full course of dual anti-platelet therapy and performing surgery then after without discontinuation of aspirin.
In patients needing surgery within a few days, it is recommended to withhold clopidogrel and ticagrelor for 5 days and prasugrel for 7 days prior to surgery unless there is a high risk of thrombosis and to use (if available) platelet function tests for optimal timing of surgery.
Dual anti-platelet therapy should be resumed as soon as possible after surgery and, if

possible, within 48 hours. Transfusion of platelets is recommended should excessive or life-threatening peri-operative bleeding occur.

● If the international normalized ratio (INR) is ≤1.5, surgery can be performed safely; however, in anti-coagulated patients with a high risk of thrombo-embolism; bridging therapy is recommended; vitamin K antagonists VKAs must be stopped 3 – 5 days before surgery, with daily INR measurements until 1.5-2.0 is reached, then start therapeutic doses of unfractionated heparin (UFH) or LMWH (preferable); the last dose of LMWH should be administered no later than 12 hours before the procedure. However;
patients with mechanical prosthetic heart valves, the evidence in favor of IV UFH; these patients should be hospitalized and treated with UFH until 4 hours before surgery. Depending on the patient's haemostatic status; LMWH or UFH is resumed 12 hours after surgery at the pre-procedural dose but VKAs can be resumed only 1- 2 days after surgery with the pre-operative maintenance dose plus a boosting dose of 50% for 2 consecutive days; the maintenance dose should be administrated thereafter.

In patients treated with the non-VKA direct oral anticoagulants (NOACs) i.e. direct thrombin inhibitor (dabigatran) , direct factor Xa inhibitors (rivaroxaban, apixaban, edoxaban) and as all of which have short biological half-lives & well-defined 'on/off' action; bridging to surgery is mostly unnecessary . An exception to this rule is the patient with high thrombo-embolic risk, whose surgical intervention is delayed for several days. It is recommended to stop NOACs for 2–3 times their respective biological half-lives prior to surgery in surgical interventions with 'normal' bleeding risk, and 4–5 times the biological half-lives before surgery in surgical interventions with high bleeding risk. Reduced kidney function or moderate-high bleeding risk should lead to earlier cessation. Because of the fast 'on' effect of NOACs, resumption of treatment after surgery should be delayed for 1-5 days, until post-surgical bleeding tendency is diminished.

In patients who are receiving VKAs and who require reversal of the anticoagulant effect for an urgent surgical procedure, low-dose (2.5 – 5.0 mg) IV or oral vitamin K is recommended. The effect of vitamin K on INR will first be apparent after 6 – 12 hours. For more immediate reversal of the anticoagulant effect of VKAs is needed, treatment with fresh-frozen plasma (FFP) or prothrombin complex concentrate (PCC) is recommended, in addition to low-dose intravenous or oral vitamin K.

In patients receiving UFH and requiring reversal of the anticoagulant effect for an urgent surgical procedure, cessation of therapy is sufficient, because coagulation is usually normal 4 hours after cessation. When UFH is given subcutaneously, the anticoagulant effect is more prolonged. For immediate reversal, the antidote is protamine sulphate.
1 mg of protamine sulphate will usually neutralize at least 100 international units of heparin; the dose of protamine sulphate should be reduced if more than 15 minutes have elapsed since intravenous injection.

In patients who are receiving LMWHs, the anticoagulant effect may be reversed within 8 hours of the last dose. If immediate reversal is required, IV protamine sulphate can be

used, but anti-Xa activity is never completely neutralized (maximum 50%).
When severe bleeding complications occur under the influence of NOACs, symptomatic treatment should be initiated because of the lack of specific antidotes with a potential benefit for the use of PCC or FFP. Haemodialysis is an effective method for eliminating direct thrombin inhibitor (dabigatran) from the circulation but does not help when a direct factor Xa inhibitor (rivaroxaban, apixaban, edoxaban) has been used.

Risk-reduction strategies: Revascularization
Perform non-urgent, non-cardiac surgery in patients with recent bare metal stent BMS after a minimum of 4 weeks and ideally 3 months following the implantation.
Perform non-urgent, non-cardiac surgery in patients who have had recent drug eluting stent DES no sooner than 12 months following the implantation but may be reduced to 6 months for the new generation DES.
Perform non-urgent, non-cardiac surgery in patients with recent balloon angioplasty after at least 2 weeks after the implantation
Prophylactic myocardial revascularization before high-risk surgery may be considered depending on the extent of a stress-induced perfusion defect.

Heart failure
Pre-operative CXR, ECG & echocardiography should be considered. The assessment of natriuretic peptides (BNP or NTproBNP) should form part of a pre-operative evaluation comparing the pre-operative and post-operative levels of natriuretic peptides as they are strongly correlated to the prognosis of heart failure and enhancing risk stratification for the composite outcomes.
Although continuation of ACEIs/ARBs until the day of surgery has been associated with an increased incidence of hypotension, it is recommended that all heart-failure medications, such as ACE inhibitors, ARBs, and beta-blockers, be continued and that the patient's haemodynamic status be carefully monitored. In patients considered susceptible to hypotension, ACEIs/ARBs may be omitted on the morning of surgery, whereas β-blockade should be continued throughout the peri-operative period if possible.
Heart failure medications should be re-instituted post-operatively, as soon as clinical conditions allow.
In patients with newly diagnosed heart failure, it is recommended that intermediate- or high-risk surgery be deferred, preferably for at least 3 months after initiation of heart failure therapy, to allow time for therapy up-titration and improvement of LV function.

Arterial hypertension
When raised blood pressure is discovered in a pre-operative evaluation, it is advisable to search for target organ damage (ECG, renal function and evidence of heart failure) and to initiate therapy to lower the blood pressure to an appropriate level.
During the induction of anaesthesia, sympathetic activation can cause an increase in

blood pressure of 20–30 mm Hg and a heart rate increase of 15–20 bpm in normotensive individuals, this response is more pronounced in patients with untreated hypertension.
As the period of anaesthesia progresses, patients with pre-existing hypertension are more likely to experience lability of intra-operative blood pressure, which may lead to myocardial ischaemia. Hypertensive patient may also be unstable, and profound hypotension especially when associated with baroreflex-mediated tachycardia may be equally detrimental. It is recommended that peri-operative blood pressure be kept at 70–100% of baseline, avoiding excessive tachycardia.
Post-surgical elevation of blood pressure is frequently brought about by anxiety and pain after awakening, and may return to normal after treating these factors.
In patients with grade 1 or 2 hypertension (systolic blood pressure <180 mm Hg; diastolic blood pressure <110 mm Hg) there is no evidence of benefit from delaying surgery to optimize therapy; in such cases, antihypertensive medications should be continued during the peri-operative period.
In patients with grade 3 hypertension (systolic blood pressure ≥180 mm Hg and/or diastolic blood pressure ≥110 mm Hg), the potential benefits of delaying surgery to optimize the pharmacological therapy should be weighed against the risk of delaying the procedure.
There is no clear evidence favoring one mode of antihypertensive therapy over another in patients undergoing non-cardiac surgery and patients with arterial hypertension should be managed according to the existing guidelines for treatment of hypertension.

Arrhythmias

Arrhythmias may indicate underlying structural heart disease; the discovery of pre-operative arrhythmias should lead to thorough evaluation before surgery with echocardiography, coronary angiography /revascularization and in selected cases; invasive electrophysiological study and treatment steps include identifying and correcting the reversible causes (e.g. hypoxia, electrolyte disturbance).
There is no evidence that VPBs or non-sustained VTs alone are associated with a worse prognosis or that suppressive therapy is beneficial; however and regardless of the cause, sustained monomorphic VT with haemodynamic compromise must be treated promptly with electric cardioversion. Amiodarone can be used for initial treatment of patients with stable sustained monomorphic VT to prevent recurrences. Beta-blockers are useful in patients with recurrent sustained polymorphic VT, especially if ischaemia is suspected
No medication is recommended to suppress supraventricular premature beats.
For SVT; Vagal manoeuvres , adenosine or verapamil are helpful. In cases with incessant or commonly recurring SVT in the perioperative setting; beta-blockers, calcium channel blockers, or amiodarone treatment can be used. In rare cases (and taking into account the urgency and nature of planned surgery), pre-operative catheter ablation of the arrhythmia substrate may be considered, e.g. for patients with Wolff-Parkinson- White syndrome and pre-excited AF.

The objective in managing perioperative AF is usually ventricular rate control, β-blockers and calcium channel blockers (verapamil, diltiazem) are the drugs of choice for rate control. Amiodarone can be used as a first line drug in patients with heart failure, since digoxin is frequently ineffective in high adrenergic states such as surgery.
Anticoagulation must be based on the individual clinical situation.
Peri-operative brady-arrhythmias usually respond well to short term pharmacological therapy; temporary cardiac pacing & prophylactic pacing before non-cardiac surgery is not commonly indicated apart from patients with complete heart block or symptomatic asystolic episodes.
Patients with a permanent pacemaker can safely undergo surgery if appropriate precautions are taken; keeping the electro-cautery device away from the pacemaker, giving only brief bursts, and using the lowest possible amplitude to decrease the interference. The pace maker should be set in an asynchronous or non-sensing mode in patients who are pacemaker-dependent. This is most easily done in the operating room by placing a magnet on the skin over the pace maker.

Valvular heart disease VHD

It is recommended that a clinical and echocardiographic evaluation of a patient with VHD be performed and, if necessary, treated before non-cardiac surgery. The evaluation aimed to assess the severity of VHD, the symptoms and their relationship to VHD, the estimated risks of valvular intervention and of cardiac complications according to the type of non-cardiac surgery. The usual classification of non-cardiac surgery, using the three risk groups defined in Table 1, should also be used in patients with VHD**.**
Aortic stenosis: Severe aortic stenosis is defined as valve area (<1.0 cm^2 or < 0.6cm^2/m^2 body surface area, except in obese patients), and flow dependent indices (maximum jet velocity 4 m/sec and mean aortic pressure gradient ≥40 mm Hg).
Severe aortic stenosis constitutes a well-established risk factor for perioperative mortality and myocardial infarction.
In case of urgent non-cardiac surgery in patients with severe aortic stenosis, such procedures should be performed under more invasive haemodynamic monitoring, avoiding rapid changes in volume status and heart rhythm as far as possible.
In the case of elective non-cardiac surgery, the presence of symptoms is essential for decision-making. In *symptomatic* patients, aortic valve replacement should be considered before elective surgery. Patients who are not candidates for valve replacement, due either to high risks associated with serious comorbidities or contra-indicated for aortic valve replacement or refusal to undergo the operation, non-cardiac surgery should be performed only if is essential otherwise; balloon aortic valvuloplasty or, preferably, transcatheter aortic valve implantation (TAVI) may be a reasonable therapeutic option before surgery.
In *asymptomatic* patients, non-cardiac surgery of low to intermediate risk can be performed safely & if possible, the absence of symptoms should be confirmed by exercise testing. If high-risk surgery is planned, further clinical assessment is necessary to

assess the risk of aortic valve replacement as it is considered as the initial procedure. Those at high risk for aortic valve replacement, elective surgery under more invasive haemodynamic monitoring should be performed only if strictly necessary.

Mitral stenosis: Non-cardiac surgery can be performed with relatively low levels of risk in patients with non-significant mitral stenosis (valve area >1.5 cm^2) and in asymptomatic patients with significant mitral stenosis (valve area <1.5 cm^2) and systolic pulmonary artery pressure <50 mm Hg. Pre-operative surgical correction of mitral stenosis in these patients is not indicated. Control fluid status & heart rate is essential to avoid tachycardia, which may cause pulmonary oedema. Development of AF may cause serious clinical deterioration. Due to high risk of embolism, anticoagulation is important. In *asymptomatic* patients with significant mitral stenosis and systolic pulmonary artery pressure >50 mm Hg, and in *symptomatic* patients, the risk related to the non-cardiac procedure is significantly higher, and these patients may benefit from percutaneous mitral commissurotomy (or open surgical repair) particularly before high-risk surgery.

Primary aortic regurgitation and mitral regurgitation: Non-significant aortic regurgitation and mitral regurgitation do not independently increase the risk of cardiovascular complications during non-cardiac surgery.

In *asymptomatic* patients with severe aortic or mitral regurgitation and preserved LV function, non-cardiac surgery can be performed without additional risk.

Symptomatic patients and those who are asymptomatic with severely impaired LVEF (<30%) are at high risk of cardiovascular complications, and non-cardiac surgery should be performed only if necessary. If so; they may benefit from optimization of pharmacological therapy to produce maximal haemodynamic stabilization before undergoing high-risk surgery.

Patients with prosthetic valve(s): can undergo non-cardiac surgery without additional risk, provided that there is no evidence of valve or ventricular dysfunction. The main problem is the need for antibiotic prophylaxis & for modification of the anticoagulation regimen in the perioperative period, with oral anticoagulants being temporarily replaced by UFH (preferable) or LMWH at therapeutic doses.

Renal disease

Impaired renal function with cut-off GFR value of < 60 mL/min/1.73 m^2 is associated with a significantly increased risk of CVD and is an independent risk factor for adverse post-operative cardiovascular outcomes, including myocardial infarction, stroke, and progression of heart failure.

Risk factors for the development of post-operative acute kidney injury AKI following non-cardiac surgery include: age >56 years, male, active cardiac failure, ascites, hypertension, emergency surgery, intra-peritoneal surgery, pre-operative creatinine elevation and diabetes mellitus. Identification of patients at risk of perioperative worsening of renal function is important to initiate supportive measures; maintenance of

adequate intravascular volume for renal perfusion and use of vasopressors.
In patients with stage 4 or 5 CKD, prophylactic hemofiltration may be considered before complex intervention or high-risk surgery.
The most frequent causes for AKI in hospitalized cardiac patients relate to the combination of a low cardiac output/high venous pressure, and/or the administration of iodinated contrast media during diagnostic and interventional vascular procedures.
Pre-procedural hydration with isotonic fluids is the most effective method of reducing the risk of CI-AKI. Normal saline or isotonic sodium bicarbonate (1.26%) may be used.
N-acetyl cysteine may be considered for prophylaxis of CI-AKI; however, the evidence for its benefit remains inconclusive.
Alkalinize urine using bicarbonate, sodium/potassium citrate or acetazolamide is helpful.
The use of high-dose statins in preventing CI-AKI is promising.
Although there are theoretical benefits from the use of loop diuretics in early or established AKI, these have not been supported by data in studies, and diuretics are therefore not recommended for the prevention or treatment of AKI.

Cerebrovascular disease CVD

The followings are independent predictors of perioperative stroke:
age, history of myocardial infarction within 6 months prior to surgery, acute renal failure, history of stroke, history of TIA, dialysis, hypertension, chronic obstructive pulmonary disease (COPD), and current smoking.
Peri-operative strokes are mainly ischemic and cardio-embolic and AF is often the underlying leading condition. To attenuate the risk of peri-operative stroke, anti-platelet/anticoagulant & statin treatments should be continued whenever possible throughout the perioperative period. Alternatively, the period of drug withdrawal should be kept as short as possible while weighting thrombo-embolic and haemorrhagic risks with adequate selection of the anaesthetic technique (regional, neuraxial, and general anaesthesia), prevention and treatment of AF, euglycaemic control & meticulous perioperative blood pressure control. Patients undergoing non-cardiac surgery should be questioned about previous neurological symptoms, and those with symptoms suggestive of TIA or stroke in the preceding 6 months should undergo pre-operative neurological consultation as well as neuro-vascular and brain imaging, where appropriate.

Peripheral artery disease PAD

Patients with PAD (ankle–brachial ratio of <0.9 or previously re-vascularized with surgery or percutaneous transluminal angioplasty) usually have advanced atherosclerotic disease affecting most vascular beds in varying degrees and considered risk factor for non-cardiac surgery; it is reasonable to assess the presence of IHD through clinical evaluation, exercise or imaging test in PAD patient with clinical symptoms or has more than two of the clinical risk factors detailed in Table 3.
All patients with PAD should be treated with statins and platelet inhibitors with blood pressure control and lifestyle measures should be attended as recommended.

Pulmonary disease

Pre-existing pulmonary disease especially in smokers & after abdominal or thoracic surgery increase the risk of post-operative pulmonary complications which are in part a consequence of the development of atelectasis during general anaesthesia; however, factors that result in post-operative hypoventilation, reduced tidal volumes, and impaired lung expansion may cause persistent lung collapse and increase the risk of respiratory infection. Certain respiratory conditions are associated with cardiovascular pathology and may require special cardiac risk assessment and management, in addition to dealing with pulmonary disease per se. Of these are: chronic obstructive air way disease COPD, obesity hypoventilation syndrome (OHS) and pulmonary artery hypertension (PAH).

COPD is a risk factor for IHD and sudden death, apart from several shared risk factors for both types of disease (smoking, diabetes, hypertension, systemic inflammation, increased plasma fibrinogen). Reduced (FEV_1) is considered a marker for cardiovascular mortality & nonfatal coronary events independent of age, gender, and smoking history.

COPD patients should stop smoking (>2 months before surgery) with instruction in chest physiotherapy and lung expansion maneuvers.

β-adrenergic agonists, anticholinergic agents and short-term systemic/inhaled steroids should be continued until the day of surgery in symptomatic patients, appropriate antibiotics for pulmonary infection should be given for at least 10 days before surgery.

Obesity hypoventilation syndrome OHS is defined as the triad of obesity, daytime hypoventilation, and sleep-disordered breathing. Although distinct from simple obesity and sleep apnea, but most patients with OHS also have obstructive sleep apnea.

OHS is associated with CAD, obesity-related cardiomyopathy, heart failure, stroke, pulmonary hypertension, cor pulmonale and metabolic syndrome. Pre-operative evaluation includes the use of screening questionnaires, peripheral oxygen saturations, serum bicarbonate levels and at times; positive airway pressure therapy.

Pulmonary hypertension PH is defined as an increase in mean pulmonary arterial pressure >25 mm Hg at rest, as assessed by right heart catheterization & is associated with increased post-operative complications, including right ventricular failure, myocardial ischaemia, and post-operative hypoxia and mortality.

The outcome predictors of patients with pulmonary hypertension undergoing non-cardiac surgery include New York Heart Association functional Class >III, intermediate to high-risk surgery, right ventricular dysfunction, and long duration of anaesthesia.

During peri-operative state; any specific therapy must be continued with facilities for appropriate monitoring. In case of progression of right heart failure in the post-operative period, the diuretic dose should be optimized and, if necessary, inotropic support with dobutamine needs to be initiated.

Disturbed glucose metabolism

Insulin should be used to control hyperglycemia with the trigger for therapy at 10.0 mmol/L (180 mg/dL) and relative trigger at 8.3 mmol/L (150 mg/dL).

Early diagnosis of post-operative complications

Consider to stratify the risk of post-operative complications and mortality with a simple surgical 'Apgar' score (Table 2) to allow redirecting patients to higher intensity units or selected postoperative measurements of natriuretic peptides and troponin.

Pre-operatively and post-operatively, patients who could most benefit from BNP and troponin measurements are those with METs ≤ 4 or with a revised cardiac risk index value >1 for vascular surgery, >2 for non-vascular surgery and post-operative patients with a surgical Apgar score <7 in order to detect complications early, independently of their revised cardiac risk index values.

There is no universal 'target blood pressure value' to define intra-operative arterial hypotension, but percentage decreases >20% in mean arterial pressure, or mean arterial pressure values < 60 mm Hg for cumulative durations of > 30 minutes, are associated with a statistically significant increase in the risk of post-operative complications that include myocardial infarction, stroke, and death. Similarly, increased duration (> 30 minutes) of deep anaesthesia (bispectral index scale values <45) was statistically associated with an increased risk of post-operative complications.

Post-operative pain management

Severe post-operative pain increases sympathetic drive and delays recovery.

Neuroaxial analgesia with local anesthetics or opioids and/or alpha2-agonists and IV opioids, alone or in combination with non-steroidal anti-inflammatory drugs, seem to be the most effective regimens.

Epidural analgesia is associated with a significant decrease in mortality and risk of AF, SVT, deep-vein thrombosis, respiratory depression, atelectasis, pneumonia, ileus, and post-operative nausea and vomiting, and also improved recovery of bowel function, but significantly increased the risk of arterial hypotension, pruritus, urinary retention, and motor blockade.

Local or regional analgesia, gabapentin or pregabaline, or IV lidocaine, might have a preventive effect against persistent postsurgical pain and could be used in a high-risk population.

Non-steroidal anti-inflammatory drugs and cyclo-oxygenase-2 inhibitors have the potential for promoting heart and renal failure, thrombo-embolic events, and should be avoided in elderly, myocardial ischaemia, diffuse atherosclerosis, patients with renal and heart failure, on diuretics, or those with unstable hemodynamic.

Summary of pre-operative cardiac risk evaluation

1. In urgent cases, patient or surgery specific factors dictate the strategy and do not allow further cardiac testing or treatment. In these cases, the consultant provides recommendations on perioperative medical management, surveillance for cardiac events, and continuation of chronic cardiovascular medical therapy.

2. If the patient is unstable (*unstable angina pectoris, acute heart failure, significant cardiac arrhythmias, symptomatic valvular heart disease, recent myocardiac infarction and residual myocardiac ischaemia*); surgery should be cancelled or delayed to clarify and treat appropriately before surgery until stabilization Treatment options should be discussed by a multidisciplinary team, because interventions might have implications for anaesthesia and surgical care.

3. In cardiac stable patients, determine the risk of the surgical procedure (Table.1).

If the estimated 30-day cardiac risk of the procedure in cardiac stable patients is low; it is unlikely that test results will influence management and it would be appropriate to proceed with the planned surgical procedure. One can identify risk factors and provide recommendations on lifestyle and medical therapy to improve long-term outcome.

4. Consider the functional capacity of the patient. If asymptomatic or cardiac stable patient has good functional capacity (>4 METs), perioperative management is unlikely to be changed on the basis of test results irrespective of the planned surgical procedure. It is appropriate to refer the patient for surgery even in the presence of clinical risk factors with recommendations for lifestyle and appropriate medication.

5. In patients with a moderate or poor functional capacity, consider the risk of the surgical procedure (Table1). Patients scheduled for low or intermediate-risk surgery can proceed for surgery. Patients scheduled for high-risk surgery, consider non-invasive testing especially if more than two clinical risk factors present(Table.3):

if non-invasive stress test revealed no stress-induced ischaemia or mild-moderate ischaemia suggestive of one or two vessel disease then proceed to the planned surgical procedure; if non-invasive stress test revealed stress-induced ischemia individualized perioperative management is recommended, taking into consideration the potential benefit of the proposed surgical procedure weighed against the predicted adverse outcome. Also, the effect of medical therapy and/or coronary revascularization must be assessed for immediate postoperative outcome and for long-term follow-up.

Table.1 Surgical risk estimate according to type of surgery

Low-risk: < 1%	Intermediate-risk: 1–5%	High-risk: > 5%
• Superficial surgery • Breast • Dental • Endocrine: thyroid • Eye • Reconstructive • Carotid asymptomatic (CEA or CAS) • Gynaecology: minor • Orthopaedic: minor (meniscectomy) • Urological: minor (transurethral resection of the prostate)	• Intraperitoneal: splenectomy, hiatal hernia repair, cholecystectomy • Carotid symptomatic (CEA or CAS) • Peripheral arterial angioplasty • Endovascular aneurysm repair • Head and neck surgery • Neurological or orthopaedic: major (hip and spine surgery) • Urological or gynaecological: major • Renal transplant • Intra-thoracic: non-major	• Aortic and major vascular surgery • Open lower limb revascularization or amputation or thromboembolectomy • Duodeno-pancreatic surgery • Liver resection, bile duct surgery • Oesophagectomy • Repair of perforated bowel • Adrenal resection • Total cystectomy • Pneumonectomy • Pulmonary or liver transplant

Table.2 Surgical Apgar score

	0	1	2	3	4
Estimated blood loss (mL)	> 1000	601-1000	101-600	≤ 100	
Lowest mean arterial pressure (mmHg)	< 40	40-54	55-69	≥ 70	
Lowest heart rate (beats/min)	> 85	76-85	66-75	56-65	≤ 55
Calculation of the surgical Apgar score, range from 0 to 10 points, with lower scores associated with worse postoperative outcomes. Abbreviations: ml = milliliters; MAP = mean arterial pressure; HR = heart rate					

Table.3 Clinical cardiac risk factors

- Ischemic heart disease (defined as a history of MI, pathologic Q waves on the ECG, use of nitrates, abnormal stress test, or chest pain secondary to ischemic causes)
- Congestive heart failure
- History of cerebrovascular disease
- Diabetes requiring insulin therapy
- Preoperative serum creatinine level higher than 2 mg/dL

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