

How to improve outcomes for patients sustaining myocardial injury during and after noncardiac surgery

Perioperative myocardial infarction: invasive vs conservative approach

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Summary

Patients with a myocardial injury during and after noncardiac surgery have an elevated risk of death. They may have significant coronary artery disease amenable to revascularisation, but over 50% of these patients develop an infarct in territories without significant stenosis of the corresponding vessel, and the clinical presentation does not easily allow differentiation between these situations. Coronary angiography and revascularisation in surgical patients suffering perioperative cardiac events is therefore controversial. Coronary angiography with a view to possible revascularisation may be appropriate preoperatively in cases of significant left main coronary artery stenosis and moderate to severe inducible myocardial ischaemia in intermediate risk vascular patients. In the peri- and postoperative setting, coronary angiography is indicated with ST elevation and possibly persistent haemodynamic instability following a significant troponin rise. Late coronary angiography after surgery may be indicated in vascular patients at a high risk for subsequent cardiac events. Coronary angiography may be contraindicated in patients with comorbidities associated with poor intermediate term survival or uncertain acute coronary syndromes. All other generally accepted indications for coronary angiography in medical patients are far more controversial in surgical patients, because of the risk of perioperative bleeding. In these patients, the benefits of medical therapy alone may outweigh the risks of coronary angiography to identify coronary lesions amenable to revascularisation. The medical therapy of all surgical patients with a myocardial injury following noncardiac surgery should be intensified.

Key words: myocardial infarction; coronary revascularisation; surgery

Introduction

Perioperative acute coronary syndromes have a significantly higher mortality than spontaneous acute coronary syndromes [1]. However, despite their clinical importance, there is little evidence to guide clinical therapy. This review considers the anatomy and pathophysiology of perioperative myocardial infarction (PMI) and myocardial injury after noncardiac surgery (MINS), and potential medical and invasive coronary therapeutic options to manage these patients. PMI is

defined as a myocardial infarction that fulfils the Universal Definition criteria for myocardial infarction [2], and MINS is defined as “prognostically relevant myocardial injury due to ischaemia that occurs during or within 30 days after noncardiac surgery” but does not necessarily fulfil the criteria for myocardial infarction [3]. When considering the appropriateness of diagnostic coronary angiography, the principle that the incremental information gained should exceed the negative consequences of the intervention should be maintained [4]. As there are currently no accepted or evidence-based indications for coronary catheterisation and an invasive strategy in the peri-/postoperative noncardiac setting, it is reasonable to consider whether the indications for appropriate diagnostic catheterisation in nonsurgical patients are applicable to surgical patients [4, 5].

Perioperative myocardial infarction and preoperative coronary revascularisation

The two commonly considered types of myocardial infarction in the perioperative period are type 1 (plaque rupture) and type 2 (supply-demand imbalance). A type 2 myocardial infarction may be secondary to a number of potential pathophysiological mechanisms including endothelial dysfunction, vasospasm and stenotic lesions, among others [6]. It has been proposed that the incidence of type 2 PMI increases as the degree of coronary stenosis increases [7]. The implications of this proposal are that if one targets fixed stenotic lesions, then perioperative outcomes should be improved. This approach appears reasonable when one considers the reported prevalence of documented coronary artery disease potentially amenable to coronary revascularisation in high-risk preoperative surgical patients. A recent prospective observational study using preoperative computed tomography (CT) coronary angiography in surgical patients at cardiac risk showed that 1.3% (14/1067) had left main stem stenosis of >50%, and 40.3% (385/955) has at least one lesion causing >70% stenosis [8].

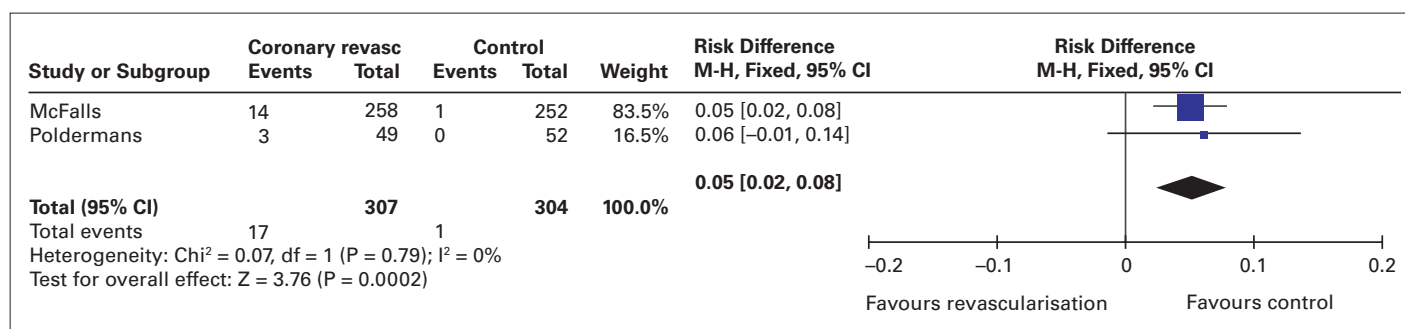


Figure 1: Major adverse cardiac events prior to noncardiac surgery following preoperative coronary revascularisation.

Despite the prevalence of surgically correctable coronary artery disease in these patients, randomised trials of preoperative coronary artery revascularisation have not shown intermediate-term cardiovascular benefit [9–11]. There are potentially three reasons for this. Firstly, preoperative coronary revascularisation prolongs the time to noncardiac surgery, with limited cardiovascular protection in the early period following coronary revascularisation, resulting in a significant increase in the incidence of major adverse cardiac events (MACE; defined as death or nonfatal myocardial infarction) preceding the noncardiac surgery (5.5% vs 0.3%, odds ratio [OR] 12.32, 95% confidence interval [CI] 2.30–66.03, $p = 0.003$, $I^2 = 0\%$) (fig. 1). Therefore, preoperative coronary revascularisation is associated with a number needed to harm of 20 prior to noncardiac surgery.

Table 1: The prediction of perioperative myocardial infarction in patients with preoperative coronary angiography demonstrating coronary stenoses of >70%.

Study	Pretest probability	PPV	NPV	Sensitivity	Specificity
VISION CTA [8]	0.087	0.12	0.94	0.56	0.62
Hwang et al. [55]	0.032	0.14	0.98	0.37	0.93

PPV = positive predictive value; NPV = negative predictive value

Table 2: The severity of the coronary stenosis and the associated type of perioperative myocardial infarction in surgical patients [14].

	Type 1 Rupture	Type 2 Supply-demand	Likelihood ratio for a type 2 PMI
LMS >50%	3/17 (18%)	7/36 (19%)	1.02
3 vessels >50%	8/17 (47%)	20/36 (56%)	1.1
2 vessels >50%	4/17 (24%)	8/36 (22%)	0.9
1 vessels >50%	3/17 (18%)	4/36 (11%)	0.6
0 vessels >50%	0/17 (0%)	2/36 (6%)	Ref

Secondly, 56% of PMIs have been shown to occur in coronary territories other than those identified by preoperative inducible ischaemia [12], suggesting that a site of significant coronary stenosis is not necessarily the site of a PMI. Indeed, the degree of coronary stenosis as seen on preoperative coronary artery screening does not accurately predict the patients at risk of perioperative myocardial infarction (table 1).

Thirdly, the need for dual antiplatelet therapy in patients with coronary stent insertion may increase the risk of perioperative bleeding, and of stent thrombosis if there is discontinuation or bridging of the therapy in the subsequent noncardiac surgery [13].

Stratifying patients by preoperative coronary artery screening of stenoses of >70% appears to be futile. Coronary CT angiography overestimates cardiac risk associated with subsequent noncardiac surgery. This would have resulted in 186 patients/1000 screened being stratified at a higher risk, yet only 15 of these patients would have been of an appropriately increased risk stratification [8]. The incorrect higher risk classification of 171 patients/1000 screened [8] could potentially lead to harm (as discussed previously, due to a delay of their noncardiac surgery and potential preoperative coronary revascularisation). It appears that preoperative coronary angiography is better at excluding disease than identifying disease (table 1). Finally, the degree of coronary stenosis appears to have no relationship with the type of PMI (table 2) [14]. It is therefore controversial whether the degree of coronary stenosis is a determinant of the type of PMI.

What may be more important in the future is a focus on clinical research that documents the potential haemodynamic significance of coronary stenosis in the preoperative period. There are some data supporting evaluation of the haemodynamic effects of a coronary stenosis, where stress echocardiography may possibly provide better prognostication than other modalities [15]. The use of modalities such as fractional flow reserve need investigation.

The only preoperative coronary characteristic which may separate the pathophysiological type of PMI may be the association between more complex lesions and type 1 PMIs [16].

Patients who may benefit from preoperative coronary revascularisation

Randomised clinical trials demonstrating the usefulness of preoperative revascularisation are lacking. It is possible that there are only two groups of patients in whom preoperative coronary revascularisation may be cardioprotective, although it is important to understand that neither of the groups have been subjected to a randomised clinical trial. There is a selection bias in the data from the Coronary Artery Revascularization Prophylaxis (CARP) trial, as exclusion criteria included at least 50% stenosis of the left main coronary artery and a left ventricular ejection fraction <20% [9]. However, revascularisation of left main stem stenosis in these vascular surgical patients has been associated with a significantly improved 2.5 year survival (hazard ratio 0.19, 95% CI 0.05–0.66) [17]. A second group of intermediate risk vascular surgical patients who demonstrated moderate to severe reversible ischaemia on preoperative thallium scanning also had significantly improved survival following preoperative coronary revascularisation (OR 0.52) [18].

The indications for coronary angiography and revascularisation are similar to those in the “non-presurgical” setting. Medical and interventional treatment of myocardial ischaemia is recommended whenever noncardiac surgery can be delayed. The same criteria for an invasive approach should be used as in the “non-presurgical” setting. Diagnostic coronary angiography could be considered acceptable in medical patients who present with intermediate-risk clinical findings and demonstrable reversible myocardial ischaemia [4, 19].

The pathophysiology of perioperative myocardial infarction

It appears, therefore, that it is important to understand the pathophysiology of the perioperative myocardial infarction, as opposed to the coronary anatomy, in order to determine the appropriate therapy.

The ischaemia precedes troponin elevation in the majority of vascular surgical patients by approximately 18 hours [20]. Importantly, nearly all these patients with myocardial ischaemia after noncardiac surgery present with the ischaemia within the first 3 days after surgery [20], emphasising the importance of the oxy-

gen supply-demand imbalance rather than complete coronary occlusion in the early postoperative period. Myocardial oxygen supply-demand imbalance may be secondary to (i) the presence of significant stenoses [12, 21–23], (ii) partial occlusion from plaque rupture [22], (iii) low flow states associated with thrombosis [22], (iv) increased postoperative myocardial oxygen demand [24], (v) decreased global oxygen supply (anaemia, hypoxaemia, low cardiac output, hypotension), and (vi) procoagulation [25].

The association between the surgical procedure and postoperative cardiac events is therefore dependent on (i) the duration of operation [26], (ii) the haemodynamic physiological insult [27], (iii) the degree of perioperative bleeding and the need for transfusion [26], and (iv) the associated inflammatory response secondary to the surgery [28]. The data suggest that patients undergoing intermediate and major surgery are at increased risk of postoperative myocardial events [3].

Perioperative myocardial ischaemia has been documented and/or proposed to occur through a number of mechanisms. Classically, peri- and postoperative oxygen supply-demand imbalance has been shown to be aggravated by an increase in heart rate and/or a relative tachycardia [29–31]. Recently, the importance of the development of intracoronary thrombosis without plaque rupture has been proposed as a pathophysiological mechanism of PMI [32]. Distal to a coronary atheromatous lesion, an intracoronary thrombus may form owing to poststenotic blood flow stasis induced by hypotension and platelet activation [32]. Indeed, hypotension is more commonly associated with perioperative adverse cardiac outcomes than is hypertension [33–35]. Coronary atheroma causes the boundary layer between blood and vessel wall to separate distal to the coronary lesion, resulting in a low pressure area behind (distal to) the atheroma with regions of reverse flow or recirculation. Periods of hypotension may further decrease coronary blood flow and provide a physiological environment conducive to thrombus formation [32]. The absence of evident thrombus does not exclude this mechanism, as subsequent thrombolysis prior to coronary angiography following PMI is likely [1]. Clinically important hypotension has been shown to be an independent predictor of PMI [27]. Platelet activation occurs at the site of coronary stenoses, and these activated platelets may contribute to thrombus formation in the loss pressure, low flow recirculation zone found distal to the coronary atheroma [32].

In summary, PMI is most likely to occur in patients with significant coronary artery stenoses. In the first few postoperative days, these patients may suffer a

relative flow-mediated hypoperfusion, which precedes myocardial infarction. This is most commonly at the site of significant coronary stenoses (>80%) [21, 22]. Hypoperfusion may be aggravated by hypotension, and/or intracoronary thrombosis secondary to hypercoagulability and inflammation. Hypotension that has been associated with perioperative myocardial infarction is defined as a systolic blood pressure less than

90 mm Hg requiring fluid resuscitation, intra-aortic balloon pump, an inotropic agent or vasopressors [27]. A mean arterial pressure of <55 mm Hg has been associated with MINS [36]. There are few data on the degree of perioperative hypercoagulability and inflammation and PMI. A myocardial oxygen demand imbalance may be associated with the surgical stress response, volume status changes, pain and sympathetic stimulation [25, 28]. This process precedes troponin elevation in 80% of patients by approximately 18 hours. It is possible that even a rapid rise in troponins within the first 2 postoperative days [37], is more likely to represent an oxygen supply-demand imbalance (possibly aggravated by thrombus formation) than plaque rupture. PMIs are probably more evenly distributed across the surgical hospital stay [32]. The pathophysiology of fatal PMIs are shown in table 3.

Table 3: The characteristics of fatal perioperative myocardial infarctions (adapted from ref [32]).

Characteristic	Type 1 Rupture	Type 2 Supply-demand
Coronary stenoses >50%	>95% of all PMI	
Incidence	44–55%	45–56%
Multivessel coronary stenoses	92%	86%
Presence intraluminal thrombus	52–66%	0–7%
Mean postoperative day of presentation	Later	Earlier
Pathophysiology	Complex perioperative physiology	

Diagnosis and management of perioperative myocardial infarction

A relevant number of patients (84%) do not report any specific ischaemic symptoms postoperatively [3]. Indicative symptoms and signs (angina, dizziness, shortness of breath, hypotension) of myocardial infarction in the postoperative period can easily be masked for the following reasons: intubation and/or sedation, delirium, difficulty in distinguishing between surgery-related pain and “cardiac pain”, differential diagnosis between hypotension and/or hypoxaemia due to surgical complications or myocardial infarction and associated haemodynamic instability.

It is known that postoperative myocardial infarction most frequently occurs in the first 3 days, occurring most commonly (>50%) in the first 24 hours after surgery [1, 38]. Still, many cases are not identified up to 3–5 days after surgery, resulting in a comparatively large number of patients suffering cardiogenic shock (approximately 40%) or even fatal outcome (up to 70%) [7]. As clinical findings are often not indicative, the diagnostic pathway differs from that for patients presenting with a classical myocardial infarction. The 12-lead ECG is a cheap and easily available tool for the detection of myocardial ischaemia. If possible, this should be compared with a previous ECG to rule out confounding signs. Postoperative ECG changes may be missed, as they are often transient or subtle [3]. According to the VISION trial, new postoperative ST-elevation, left bundle-branch block and ECG changes of the anterior wall are associated with an elevated 30-day mortality [3]. Perioperative myocardial ischaemia with ST depression is nearly 40 times more common than ST elevation in perioperative Holter monitoring [20]. ST

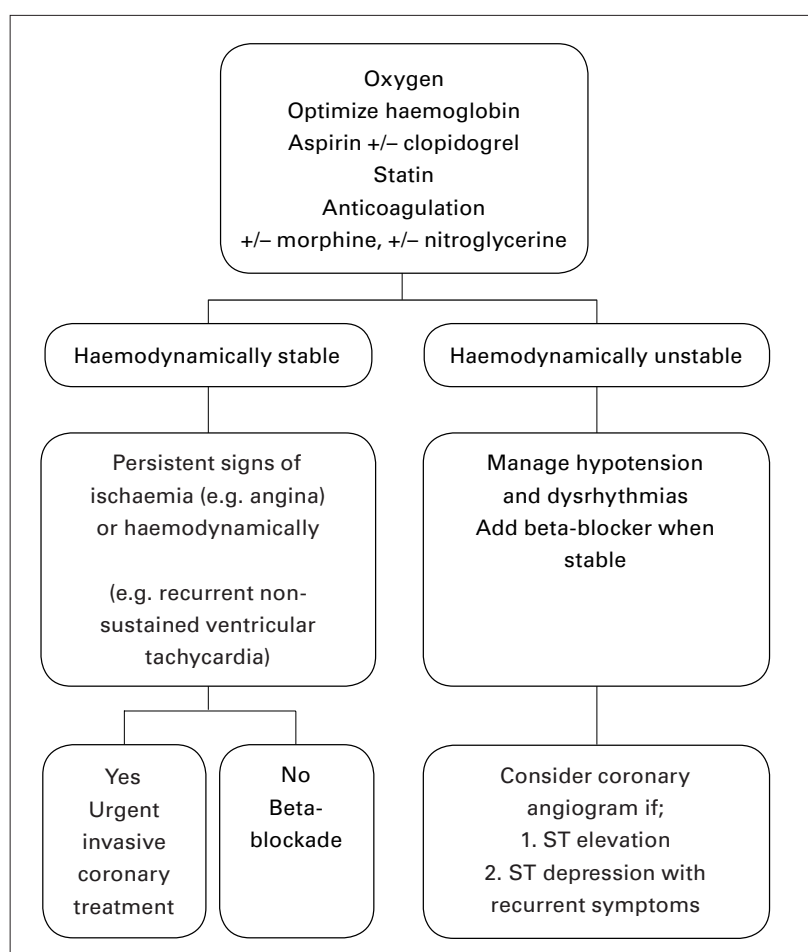


Figure 2: Proposed management algorithm for a perioperative myocardial infarction (adapted from [57]).

elevation occurs infrequently (3% of patients) [38]. Non-ST-segment-elevation myocardial infarction (NSTEMI) occurs frequently in the postoperative period. Although there are no data directly reporting the outcomes associated with type 1 and type 2 PMIs, data suggest that type 1 PMI is probably significantly less frequent than type 2 PMI, but associated with a significantly worse outcome.

Based on the pathophysiology and coronary anatomy of PMIs, the following management plan is suggested, as there is no evidence from randomised trials to guide the management of perioperative myocardial infarction (fig. 2). For example, a case-control study suggested that addition or intensification of beta-adrenergic blockade improves outcomes following PMI [39], but there are no data on the role of beta-blockers in haemodynamically unstable PMI patients. Based on the PeriOperative ISchemic Evaluation (POISE) trial data and the association between hypotension and mortality [40], we would not recommend beta-blockade in unstable surgical patients who have suffered a PMI.

Pharmacological management

The PeriOperative ISchemic Evaluation (POISE) trial showed that the use of statin and aspirin therapy was associated with cardiovascular protection in patients who had had a perioperative cardiovascular event [41]. This is important, as approximately 50% of patients with postoperative troponin elevations do not have these medications prescribed during the hospital admission [41] and there is a 24-hour window of a sustained low troponin leak prior to further troponin elevation in approximately half of the patients [37]. A core principle is to minimise the extent of the troponin leak, as the peak troponin level is associated with mortality [42]. There is case-control observational evidence that intensification of aspirin, statin, beta-blockers and angiotensin converting-enzyme (ACE) inhibitors improves survival in patients with a postoperative troponin leak [39]. This therapy would require a team approach that includes management of antithrombotics, which should be modified according to the surgical circumstances (and the postoperative bleeding risk).

There are no randomised controlled trials on the therapeutic management of patients with MINS. All therapeutic options need to be discussed at an interdisciplinary level as soon as possible. Suggested management might include the following;

- Aspirin: 100 mg daily, usually with no loading dose,
- Heparin: Start with 10 000 to 15 000 (up to 20 000) international units per day without a bolus,

- Dual antiplatelet therapy should be individualised according to surgical bleeding risk and interdisciplinary discussion between cardiologist, surgeon and anaesthesiologist or intensivist.

Antiplatelet therapy

Treating acute coronary syndrome (ACS) patients in the setting of noncardiac surgery requires a balance between bleeding and thrombosis. The current guidelines on ST-elevation myocardial infarction (STEMI) [43] and NSTEMI [44] have to be adapted to the bleeding risk associated with the surgical intervention. In accordance with the 2014 guidelines on cardiovascular assessment and management of patients undergoing noncardiac surgery [19], aspirin as primary preventive agent should be discontinued preoperatively because the risk of bleeding exceeds the risk of MACE [45]. In patients with a history of myocardial infarction or cerebrovascular disease, the risk of bleeding has to be balanced against the ischaemic risk, and aspirin should be withheld or continued accordingly [19]. Should patients suffer a perioperative MI, then aspirin should be started as soon as possible [41], and dual antiplatelet therapy should be considered based on the indication, surgery and pharmacokinetic properties of the agents. Currently, there are no available data about the new oral antiplatelet drugs. On the basis of their pharmacodynamic and pharmacokinetic properties, they are initially not advisable perioperatively because of an increased risk of bleeding. GPIIa/IIIb inhibitors theoretically have better antiplatelet properties, although data on their use in a high-risk perioperative situation are lacking. Cangrelor, a new reversible, intravenous, short acting P2Y₁₂ inhibitor seems to be a promising option for initial, controllable antiplatelet treatment in the event of postoperative MACE; however clinical experience in the setting of PMI is lacking [46]. Initiation of dual antiplatelet therapy is only advisable if the bleeding risk is either very low or a potential haemorrhage can be easily controlled.

Heparins

Many patients in the postoperative setting will be receiving heparin thromboprophylaxis. As coronary artery thrombosis is a primarily platelet-mediated pathology, the use of heparins alone does not provide full antithrombotic protection. In cases of acute myocardial infarction, the possibility of dose increase or initiation of treatment should be evaluated by an interdisciplinary team. If management includes percutaneous coronary intervention, then intravenous anticoagulants are mandatory [47]. The combination of dual antiplatelet therapy and anticoagulants in the

perioperative period has not yet been properly investigated. Pharmacological properties suggest that an individualised, cautious approach, depending on comorbidities and indications, is advisable.

The role of coronary revascularisation following perioperative myocardial infarction

Although we cannot predict in the preoperative period which patients will benefit from coronary interventions, once a patient has sustained a PMI, we know that a coronary angiogram is indicated. The majority of patients who sustain a PMI have lesions that exhibit more than >50% stenosis, reported from 70.2% in CTA VISION [8] to 94.2% in another study of PMIs [1]. In non-surgical patients without prior noninvasive stress imaging, but suspected coronary artery disease, a pretest probability of 90% for coronary artery disease is an acceptable indication for coronary angiography [4]. The pretest probability for coronary artery disease in CTA VISION following PMI was 92% [8].

Timing of coronary angiography where revascularisation may improve outcome

As all patients who have had a PMI potentially fulfil the criteria for a diagnostic coronary angiogram, the question then becomes: when should the coronary angiography be done with a view to coronary revascularisation?

An immediate invasive strategy should be considered for STEMI [48] or cardiogenic shock due to suspected ACS [4]. The evidence for this approach is, however, poor. In a systematic review of the case series on the management of PMI, haemodynamically unstable short-term mortality was reported to be 32.3% (95% CI 20.6–43.9%) [49]. Patient management differed significantly according to haemodynamic presentation, with haemodynamically unstable patients (appropriately) receiving significantly more acute coronary interventions than haemodynamically stable patients (75.8% vs 23.1%, respectively, $p = 0.0006$), although the analysis was underpowered to determine whether acute coronary interventions improved short-term mortality (29.8% [14/47] for acute coronary intervention and 40% [6/15] for no intervention, $p = 0.53$) [49]. A trial of 361 patients per group would be needed to show a relative risk reduction in mortality of 25% with 80% power, and an alpha error of 0.05. This systematic review also suggested publication bias [49], as the proportion of reported haemodynamically unstable patients was significantly more than expected [49]. In a case series of an early invasive strategy, thrombus was seen in 63%

of patients [50], although the sensitivity to visualise thrombus with coronary angiography is low. These patients underwent angiography within 11.1 ± 17.4 hours after symptom onset [50]. The extent of the occlusion is, however, unclear. In the acute postoperative situation, where the bleeding risk may be prohibitive, it is important to consider strategies to restore flow in coronary arteries without the need for long-term dual antiplatelet therapy (e.g. balloon dilatation or thrombus aspiration only).

In patients who do not fulfil criteria for an early invasive strategy, a decision needs to be taken as to how early a diagnostic coronary angiogram should be considered. For a “deferred angiogram” in nonsurgical (medical) patients, a 12- to 48-hour window is suggested for high-risk patients [44, 51]. In surgical patients, it is unlikely that coronary angiography would be realistically conducted within this time period as the mean time to coronary angiography has been reported as 5.5 days (± 8 days) from PMI presentation [1]. In these patients, however, the proportion of thrombotic lesions seen (7.5%) was less than in the post-mortem studies [21, 22], suggesting that antiplatelet and antithrombotic therapy may have resulted in a significant reduction in thromboses [1]. It is possible, therefore, that in patients without an indication for immediate invasive therapy, it is crucial to optimise haemodynamics and medical therapy because coronary angiography is certainly delayed in surgical patients, and these interventions may decrease the number and severity of coronary thromboses.

However, it is important to risk-stratify all patients who have been acutely stabilised in order to identify those who are at a high risk of clinical events in the short term [52]. In these patients, coronary angiography would be indicated. Currently, the only risk stratification tool we have is from the myocardial injury in noncardiac surgery (MINS) study [3]. Patients scoring two or more points have a significantly higher 30-day mortality than patients with fewer than two points [3]. The reported 30-day mortality rates were 5.2% (95% CI 3.3–7.4%) with zero points, 10.2% (95% CI 6.5–11.9%) with one point, 19.0% (95% CI 8.7–24.3) with two points, 32.5% (95% CI 10.6–45.9%) with three points, and 49.8% (95% CI 12.0–65.5%) with four points [3]. The mortality for the entire MINS cohort was 9.8% [3]. Points are scored on three clinical risk factors: (i) age ≥ 75 years old (one point), (ii) ST elevation or new left bundle-branch block (two points) and (iii) anterior ischaemic ECG findings (one point). Patients with two or more points should be considered for early coronary angiography (before 30 days). However, a risk-benefit decision needs to be made about an appropriate time for this inter-

vention, as the role of bleeding resulting in an adverse cardiac outcome with an early invasive strategy in these patients is unknown. Coronary angiography alone can be used with a negligible bleeding risk in the postoperative patient as a risk stratification tool in these patients. In a prospective study, the bleeding associated with coronary angiography following PMI and subsequent antiplatelet therapy in postoperative patients was not different from that in medical patients with an acute myocardial infarction [1]. In acute invasive postoperative coronary interventions, 22% of reported patients required transfusion [50]. Percutaneous revascularisation should be deferred or postponed if the bleeding risk is considered prohibitive.

The prevalence of coronary artery disease after PMI with accepted indications for coronary revascularisation (per the coronary artery bypass grafting guidelines) [53] is shown in the table 4.

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Table 4: Prevalence of surgically correctable coronary artery disease in patients who have sustained a perioperative myocardial infarction (PMI).

Lesion	Procedure	COE	Preoperative	Post PMI
Unprotected LMS	CABG/ PCI	I/ IIa	(45/1000) 4% [56]	(10/66) 15% [14]
3 vessel	CABG	I	11% [56]*	NR
2 vessel with proximal LAD	CABG	I	89/948 (9.4%) [8]	(20/74) 27% [8]
2 vessel without proximal LAD and extensive ischaemia	CABG	IIa	39/122 (32%) [18]	NR
1 vessel with proximal LAD	CABG	IIa	NR	NR
LV dysfunction	CABG	IIa	NR	NR
Survivors of sudden cardiac death presumed ischaemic-mediated VT	CABG/PCI	I	NR	NR

LMS: left main stem; LAD: left anterior descending; COE: class of evidence; CABG: coronary artery bypass grafting; PCI: percutaneous coronary intervention; PMI: perioperative myocardial infarction; LV: left ventricle; VT: ventricular tachycardia; NR: not reported; * absolute numbers not reported

In patients who have not had coronary angiography within a month of a PMI or MINS, later coronary angiography following surgery would be indicated in vascular patients at a high risk for subsequent cardiac events on the basis of preoperative inducible myocardial ischaemia [54]. In other patients in whom the indication appears less clear, it would be appropriate to perform a stress test to determine the appropriateness of coronary angiography [52].

Patients who should not have an early invasive strategy include patients with extensive comorbidities (which is common with cancer surgery patients, for example), or patients with a low likelihood of acute coronary syndromes [52]. These are patients who have other reasons for postoperative troponin elevation including sepsis and pulmonary embolism [3].

Conclusion

Patients at risk of PMI carry a significant coronary artery disease burden; however, the ability to improve outcome in the preoperative period through coronary revascularisation is limited. Patients who have sustained a PMI should have their medical therapy optimised and then the timing of coronary angiography should be considered on the basis of early risk stratification. Randomised controlled trials are urgently needed in these patient population.

Disclosure statement

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The full list of references is available in the online article at cardiovascmed.ch