The management of combined coronary artery disease and peripheral vascular disease

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Coronary artery disease (CAD) and peripheral vascular disease (PVD) remain highly prevalent in the population due to population ageing, smoking, diabetes, unhealthy lifestyles, and the epidemic of obesity, and frequently coexist. The management of combined CAD and PVD is a common challenge and brings with it numerous clinical dilemmas. The goal of this article is to review the prevalence of PVD and its major impact upon prognosis in patients with known CAD and in turn to review the impact of CAD upon the prognosis of patients with PVD. This review will also highlight issues related to the peri-operative evaluation and management of patients going to vascular surgery, including medical optimization as well as the performance and timing of coronary revascularization.

Keywords
Coronary artery disease • Peripheral vascular disease • Peri-operative management • Non-cardiac vascular surgery

Introduction
It seems . . . to be one of those simple cases which are so extremely difficult. —Sherlock Holmes

Both coronary artery disease (CAD) and peripheral vascular disease (PVD) remain highly prevalent in the population despite a decline in mortality from cardiovascular diseases. Possible causes include population ageing, smoking, diabetes, unhealthy lifestyles, and the epidemic of obesity. As a result, the management of combined CAD and PVD is a common clinical challenge which raises many controversial issues in regard to the optimal therapeutic strategy.

The goal of this review is to emphasize the prevalence of PVD (defined in this paper as lower extremity, aortic or carotid artery disease) and its major impact upon prognosis in patients with known CAD and in turn to review the impact of CAD upon the prognosis of patients with PVD. This review will also highlight issues related to the peri-operative management of patients going to vascular surgery, including medical optimization as well as the performance and timing of coronary revascularization.

Epidemiology of combined coronary artery disease and peripheral vascular disease

Prevalence of combined coronary artery disease and peripheral vascular disease
The high prevalence of combined CAD and PVD has been confirmed in two large international studies—the REACH (Reduction in Atherothrombosis for Continued Health) registry and the AGATHA (A Global Atherothrombosis Assessment) study in which 16–35% of patients (with established atherosclerotic disease or three or more risk factors) had polyvascular disease. Significant CAD in at least one vessel has been reported in ~60% of patients with severe lower extremity PVD requiring surgery. This is not surprising considering the common risk factors for atherosclerosis—smoking, diabetes, hyperlipidaemia, hypertension, and elevated C-reactive protein—with smoking and diabetes being most strongly (two- to four-fold) correlated with lower extremity PVD.
Long-term outcomes of patients with combined coronary artery disease and peripheral vascular disease

The presence of combined lower extremity PVD and CAD is associated with nearly doubled all-cause mortality, to 4.6% per year, compared with either disease alone.2 The 1-year risk of cardiovascular death, myocardial infarction (MI), stroke, or hospitalization for atherothrombotic events among these patients was 23.1% (compared with 13–17% in either disease alone) in the REACH registry.2 Peripheral vascular disease emerged as a more important predictor of mortality than previous MI or severity of angina in the CASS registry6 (Figure 1).

Impact of peripheral vascular disease in acute coronary events

Multiple studies have shown that PVD is also a risk factor for worse outcomes in patients suffering an acute coronary syndrome.7–9 Data from the Global Registry of Acute Coronary Events (GRACE) showed an increase of in-hospital mortality from 4.5 to 7.2% and a 6-month mortality from 3.9 to 8.8% in patients with lower extremity PVD, and these differences persisted after accounting for differences in baseline characteristics.9 Other studies showed that patients having an acute coronary event with combined PVD have an increased risk for atrial fibrillation, heart failure, recurrent ischaemia,8 and requirement of blood transfusions.7

Impact of peripheral vascular disease in coronary revascularization outcomes

Patients with lower extremity PVD undergoing percutaneous coronary interventions (PCI) for CAD have lower procedural success rates, higher in-hospital cardiovascular complication rates, and higher long-term rates of MI, target vessel revascularization, and mortality10,11 (Figure 2). At 30 days post-coronary artery bypass grafting (CABG), patients with lower extremity PVD exhibited higher rates of mortality in the CASS registry12 as well as increased neurological,13 systemic, renal, and pulmonary complications than patients without PVD.14 Five-year mortality rates after CABG were five times higher in patients with symptomatic or asymptomatic [ankle-brachial index (ABI) <0.9] lower extremity PVD compared with patients without PVD in the Bypass Angioplasty Revascularization Investigation (BARI) cohort.15 Similarly, another prospective study showed a more than three-fold increased risk for the composite endpoint of death, acute coronary syndrome, or cerebrovascular events in patients undergoing CABG with clinical or subclinical (ABI <0.85) lower extremity PVD compared with patients without PVD.16

Impact of coronary artery disease in patients undergoing vascular surgery

Peri-operative and long-term prognosis after non-cardiac vascular surgery is predominantly determined by the presence and severity of underlying CAD17 (Table 1).

Peri-operative outcomes

Peri-operative MI is thought to be the result of two basic pathophysiological approaches: coronary plaque rupture leading to thrombus formation and subsequent vessel occlusion, or secondary to a sustained oxygen supply–demand mismatch perioperatively.18 The peri-operative milieu may also be detrimental in that the metabolic response to surgery is characterized by sympathetic over-activity, and an inflammatory and pro-thrombotic environment.19 Moreover, peri-operative cardiac events are asymptomatic in ≏80% of patients.20 Patients scheduled for non-cardiac vascular surgery are at significant risk of peri-operative cardiovascular morbidity and mortality with MI or cardiac death occurring in ≏6.2% (range 2.2–19.0%) of patients.21 This is due to the high prevalence of CAD in patients with PVD undergoing surgery.1 Peri-operative cardiac events were significantly increased from 3 to 8.5% in patients with concomitant CAD undergoing vascular surgery in the CASS registry.22 Cardiac death (particularly fatal MI) accounted for ≏40% of 30-day all-

![Figure 1](https://example.com/figure1.png)  
Kaplan–Meier survival curves in patients with and without vascular disease in the CASS study. Adapted from Eagle et al.6 with permission from Elsevier (Copyright © 1994).
cause mortality. In patients with suspected CAD who underwent abdominal aortic aneurysmal (AAA) surgery, the peri-operative mortality risk increased three-fold from $\approx 3$ to $9\%$.23

**Long-term outcomes**

In population-based studies from Olmsted County, Minnesota, CAD was associated with a nearly two-fold decreased chance of survival after long-term follow-up in patients undergoing elective AAA repair ($34\%$ vs. $59\%$ at 8 years)22 and lower extremity revascularization ($24\%$ vs. $51\%$ at 10 years).24 Similarly, the cumulative incidence of cardiac events at 8 years was 2--4-fold higher in patients with CAD when compared with patients without CAD undergoing elective AAA ($61\%$ vs. $15\%$)23 or carotid endarterectomy (CEA) ($61\%$ vs. $25\%$).25 Another study26 showed that, for patients undergoing AAA repair with pre-operative evidence of heart disease or hypertension, the 5-year mortality rate from MI was $11.7\%$, compared with $3.7\%$ for those without a history of hypertension or heart disease, which was similar to that expected in the general population with the same age and sex composition.

A recent population study in Rotterdam27 compared long-term prognosis of propensity-matched vascular surgery patients and patients with CAD undergoing PCI and found that the patients undergoing vascular surgery were at increased relative risk for long-term mortality. Cardiovascular death was the major cause of peri-operative and long-term mortality among vascular surgical patients with PVD ($76\%$ and $46\%$, respectively). Patients undergoing elective AAA repair and lower limb revascularization were found to have worse long-term survival when compared with patients undergoing CEA (Figure 3).

**Optimal management of patients with coronary artery disease and peripheral vascular disease**

**Principles of secondary prevention in patients with coronary artery disease and peripheral vascular disease**

As outlined earlier, patients with combined CAD and PVD have increased morbidity and mortality and they should thus be treated aggressively according to national guidelines for secondary prevention.28,29 Life-style modification is key including, as a priority, complete cessation of smoking and avoidance of exposure to passive smoke, 30 min of exercise at least five times a week and weight loss to a BMI of $<25$ kg/m$^2$, and instruction regarding a prudent diet which is low in saturated fats ($<7\%$ of total calories), trans-fatty acids, and cholesterol ($<200$ mg/day) as well as limited in sodium intake and high in fresh fruits and vegetables. Reduction of LDL cholesterol to $<100$ mg/dL (and if possible $<70$ mg/dL) with statins as first-line drugs is highly recommended. Diabetics should aim for an HbA1c $<7\%$ and should be educated in proper foot care. Blood pressure should be $<140/90$ mmHg in all patients and...
130/80 mmHg in patients with diabetes or chronic kidney disease. The use of beta-adrenergic blockers in patients with PVD is not contraindicated, and their use, as well the use of angiotensin-converting enzyme-inhibitors (ACE-I) [or angiotensin receptor blockers (ARB)], is encouraged to reduce the risk of adverse cardiovascular events. Monitoring of renal function should be performed after starting an ACE-I or ARB since patients with bilateral renal artery stenosis may develop renal failure. Antiplatelet therapy is indicated in all patients with CAD and PVD. Aspirin 75–325 mg daily is first-line therapy, but clopidogrel is an effective alternative and was found to be superior to aspirin in patients with atherosclerotic disease in the CAPRIE trial. Furthermore, it has been demonstrated that optimal risk control was associated with fewer 1-year cardiovascular ischemic symptoms or events in patients with combined CAD and PVD in the large REACH registry. Despite the known benefits of antiplatelet therapy and the treatment of hypertension, hyperlipidaemia, and possibly diabetes to reduce cardiovascular event rates in patients with CAD or at risk for CAD, patients with PVD are often less intensively treated than patients with other cardiovascular diseases. Similarly, patients with lower extremity PVD suffering an acute coronary event are often less aggressively treated (both medical therapy and revascularization) despite being at increased risk for morbidity and mortality. The reason for under-treatment remains largely unexplained but may possibly be due to insufficient knowledge and a lack of awareness among physicians and patients of the associated atherothrombotic risks in the presence of PVD.

**Management of patients with coronary artery disease undergoing vascular surgery**

The pre-operative evaluation offers a unique opportunity to identify patients at increased peri-operative risk and to initiate risk reduction therapy. The history and examination should be aimed at identifying serious active cardiac conditions (acute coronary syndromes, arrhythmias, decompensated heart failure, or severe valvular disease) which require immediate therapy, as well as assessing functional capacity and co-morbidities that help risk-stratify the patient. The Lee revised cardiac risk index (RCRI) uses six risk factors (ischaemic heart disease, congestive heart failure, cerebral vascular disease, high-risk surgery, insulin-treated diabetes, and pre-operative creatinine >2 mg/dL) to discriminate patients undergoing non-cardiac surgery at low risk (0 and 1 risk factors, risk 0.4 and 0.9%) from patients at high risk (two and three or more risk factors, risk 7 and 11%) of major cardiac events. Furthermore, the risk of peri-operative all-cause mortality increases with the type of peripheral vascular surgery performed and in patients with hypertension and COPD. Predictors of increased peri-operative risk of MI in patients undergoing vascular surgery...
were age >70 years, AAA surgery, diabetes, angina, and baseline ST-T abnormalities on ECG in the CARP (Coronary Artery Revascularization Prophylaxis) trial. Elevated troponins and brain natriuretic peptide levels pre-operatively have been associated with increased short-term events.

Peri-operative cardiac risk is directly related to the extent of jeopardized myocardium on non-invasive stress testing. Dobutamine stress echocardiography and stress nuclear imaging have been shown to have similar sensitivity (85 vs. 83%) for predicting peri-operative events, but stress echocardiography has better specificity (70 vs. 47%) and has the advantage of assessing valvular heart disease. However, the most important question here is not which stress test is better but rather whether stress testing should be performed routinely before vascular surgery. According to the 2007 ACC/AHA guidelines as well as the 2009 ESC/ESA guidelines, pre-operative non-invasive stress testing should be considered for patients undergoing vascular surgery if they have poor functional capacity (<4 metabolic equivalents) and they have three or more risk factors (Class I ESC/ESA and Class IIa ACC/AHA) and may be considered for patients with one or two clinical risk factors if it is felt that test results will change peri-operative management (Class IIb). The DECREASE-II trial demonstrated conclusively that, in intermediate-risk patients undergoing vascular surgery, among whom heart rate was tightly controlled by beta-blockers, there appeared to be no benefit from routine pre-operative non-invasive stress testing.

Importantly, although beta-blockers are a cornerstone of therapy in patients with chronic stable angina, post-MI, and congestive heart failure, their routine use in the peri-operative setting is a topic of considerable debate. Initial trials performed before the year 2000 showed a decreased incidence of peri-operative death and non-fatal MI with selective beta-blockers such as atenolol and bisoprolol, but these trials were small and some measured endpoints went way beyond the peri-operative period. However, more recent trials showed no difference in peri-operative cardiovascular events when using pre-operative metoprolol. A large retrospective study of >120,000 patients showed that the effect of beta-blockers in reducing death was directly related to the RCRI—higher risk patients (RCRI ≥2) had reduced risk of death with beta-blockers, whereas patients at low risk (RCRI of 0 or 1) did not benefit and were possibly harmed by beta-blockers. The POISE trial published in 2009 is the largest trial studying peri-operative beta-blocker use and further intensified the debate. This trial resulted in a 17% decrease in the primary endpoint of cardiovascular death, non-fatal MI, and non-fatal cardiac arrest in the patients who were treated with 100 mg of extended release metoprolol pre-operatively (repeated during the first 6 h after surgery if systolic blood pressure was >100 mmHg) and 200 mg post-operatively for 30 days compared with placebo. However, the 30% decrease in non-fatal MI (3.6 vs. 5.1%) was partially offset by a 33% increase in total mortality (3.1 vs. 2.3%) and a two-fold increase in stroke (1.0 vs. 0.5%). Post hoc analysis showed that hypotension, which was more common in the metoprolol group, was the largest attributable risk for death and stroke. A recent meta-analysis concluded that beta-blockers result in 16 fewer non-fatal MIs per 1000 patients treated, but at the expense of three non-fatal disabling strokes and (possibly) three fatal cardiac or non-cardiac complications.

The main rationale for using beta-blockers in patients undergoing vascular surgery is to decrease myocardial oxygen demand by decreasing the heart rate and myocardial contractility in the peri-operative setting that often results in a catecholamine surge. However, beta-blockers have also been described as having coronary plaque stabilization and anti-inflammatory qualities, but this effect occurs after several days of treatment in contrast to the immediate haemodynamic changes with beta-blockers. Ideally, treatment should be started with a highly selective β-1 long-acting beta-blocker (such as bisoprolol or metoprolol succinate) 7–30 days before scheduled surgery and titrated up to aim for a heart rate of 60–70 b.p.m. while keeping the systolic blood pressure >100 mmHg. This should provide the maximal possible cardiac protection to decrease peri-operative MI while avoiding the increased risk of stroke or death associated with bradycardia and hypotension. Recently published guidelines on peri-operative beta-blocker use recommend continuation of beta-blockers for patients undergoing high-risk vascular surgery who are already receiving chronic beta-blocker therapy (Class I), or starting beta-blockers if they have known CAD or myocardial ischaemia on pre-operative non-invasive stress testing (ESC/ESA Class I and ACC/AHA Class IIa). The ESC/ESA guidelines also recommend beta-blockers for all patients undergoing high-risk vascular surgery (Class I), but the ACC/AHA guidelines recommend them only for high-risk surgery patients who have more than one risk factor (Class IIa). Routine administration of high-dose beta-blockers in the absence of dose titration may be harmful to patients who are undergoing non-cardiac surgery (Class III).

Patients with PVD with or without CAD should be on a statin for secondary prevention of cardiovascular events independently of vascular surgery. Furthermore, a meta-analysis showed that the postulated pleiotropic effects of statins to induce coronary plaque stabilization reduced peri-operative mortality by 59%, as well as reducing peri-operative MI and stroke in patients undergoing vascular surgery. The recent DECREASE III randomized trial demonstrated that the addition of fluvastatin XL to beta-blocker therapy in high-risk patients undergoing elective non-cardiac vascular surgery is associated with a significant reduction in ischaemic endpoints, compared with placebo, with a 53% reduction in cardiovascular death. The magnitude of reduction in mortality is surprising and needs to be confirmed in much larger trials.

One common dilemma the physician faces when taking care of patients with combined PVD and CAD peri-operatively is regarding the timing of surgery while beta-blockers and statins are initiated. We would recommend against delaying surgery since the heart rate control by beta-blockers is immediate and the pleiotropic effects of statins also start shortly after administration. If there is only a short period of time for medical optimization before surgery, one should resist the temptation of starting high doses of beta-blockers so as not to over beta-block the patient with the associated increased risk of stroke and death. A better approach is to start low-dose oral beta-blockers pre-operatively with intra-operative use of short-acting intravenous beta-blockers (such as esmolol) titrated to goal heart rates.
The evidence for antiplatelet therapy use in the peri-operative setting has not been very well studied except in patients undergoing CEA in which aspirin has been shown to decrease peri-operative cerebrovascular events, but not MI or death. In most patients, it would be reasonable to continue aspirin in patients with combined CAD and PVD unless the risk of bleeding during surgery outweighs the risk of cardiovascular events.

The issue regarding prophylactic coronary revascularization prior to PVD surgery is controversial. Early retrospective studies had shown prophylactic CABG to be protective against peri-operative death and MI in patients undergoing vascular surgery, but the protective shield of prophylactic CABG comes at a price, in that several studies demonstrated that the morbidity and mortality of both CABG and PCI are increased in patients with PVD. Moreover, a protective effect of revascularization has not withstood the more rigorous scrutiny of some recent randomized trials, although the results have been discordant. The CARP trial randomized patients with mostly one- or two-vessel CAD to either revascularization with PCI or CABG or to no revascularization prior to major vascular surgery. There were no differences between the two groups at 30 days with regard to MI, death, or length of hospital stay. Furthermore, long-term outcome at 2.7 years was also not statistically different (Figure 4). These results were confirmed in the DECREASE-V Pilot trial in which patients with extensive stress-induced ischaemia (including three-vessel as well as left main stenosis) were randomized to revascularization or no revascularization, but yet there was no advantage in the revascularized patients with regard to all-cause death or MI at 30 and 365 days. The reason for the lack of decreased cardiac morbidity and mortality after revascularization in patients undergoing vascular surgery may be due to the fact that revascularization may restore supply-demand mismatch but has no effect on reducing the inflammatory instability of coronary plaques. Also, the benefits of revascularization are known to be manifest in the long term (≥5 years), with morbidity and mortality being actually higher in the short term (<1 year) after CABG.

Different conclusions were drawn from a recent prospective randomized study which was undertaken to determine the impact of a strategy of systematic coronary angiography in medium-high-risk patients undergoing vascular surgery vs. selective coronary angiography based on results of non-invasive tests. This resulted in increased revascularization rates in the systematic coronary angiography group with improved long-term survival as well as freedom from major coronary events after a mean follow-up of 58 months. Possible reasons for this difference in outcome of this study include longer follow-up (6 vs. 1–2 years) and the revascularization of patients considered at high-risk on anatomic grounds, who might not have been identified by stress echocardiography in the absence of significant degrees of ischaemia. The number of patients enrolled in this study was relatively small, so larger multicentre trials to confirm these findings are warranted.

The decision to perform angiography and coronary revascularization in patients undergoing vascular surgery is made independently of the need for a vascular surgical procedure. Non-cardiac vascular surgery is not an indication for coronary revascularization, but it may have an impact upon the timing and nature of the procedure; whether CABG or PCI and, in the case of CABG, the number of grafts to be performed. The following text describes the results of the CARP trial, a randomized trial comparing CABG or PCI to no revascularization in patients with one- or two-vessel CAD undergoing major vascular surgery. The primary endpoint of the trial was a composite of all-cause death, MI, or stroke at 30 days. At 3 years, the rates of these events were similar in the CABG and PCI groups (19.7% vs. 18.9%, p=0.58). The 5-year survival rates were 78.9% vs. 82.6%, respectively (p=0.37). The authors concluded that routine revascularization with CABG or PCI prior to major vascular surgery is not associated with improved outcomes compared to no revascularization.

Figure 4: Long-term survival among patients assigned to undergo coronary artery revascularization or no coronary artery revascularization before elective major vascular surgery in the CARP trial. Adapted from McFalls et al. with permission. Copyright © 2004 Massachusetts Medical Society. All rights reserved.
of the latter, whether drug-eluting stents will be used or not. A key management issue is to decide whether coronary revascularization is warranted ‘in its own right’ based upon symptoms, the severity of ischaemia, co-morbidities, LV function, and upon the severity of the coronary anatomy in those patients who undergo angiography. The ACC/AHA38 and ESC/ESA42 recommendations for prophylactic revascularization prior to vascular surgery are similar to the recommendations for revascularization in patients with CAD not undergoing surgery: acute coronary syndrome, stable angina with left main, three-vessel or two-vessel disease with significant proximal left anterior descending with either an ejection fraction <50% or ischaemia during non-invasive testing; and/or symptoms despite optimal medical management. Once the decision is made that the patient is a candidate for coronary revascularization, then the intended vascular procedure is brought back into the picture, which will help to determine the timing and nature of both procedures (Figure 5).

If revascularization by PCI is performed, postponing non-urgent vascular surgery for 14 days after balloon angioplasty,38,42 at least 6 weeks but preferably 3 months after bare metal stent61 or 1 year after drug-eluting stent,62 is recommended to decrease the risk of coronary/stent thrombosis peri-operatively (Figure 6). In patients who require non-elective surgery within 12 months of drug-eluting stent placement, dual antiplatelet therapy should be continued if bleeding risk is low. If the risk of peri-operative bleeding is significantly high, then clopidogrel should be discontinued 3–5 days before surgery, and aspirin should be maintained. Clopidogrel (300 mg loading dose) should be restarted ideally on the day of (or the day after) surgery. In the highest risk patients (recent ST-elevation MI or history of stent thrombosis), intravenous small-molecule glycoprotein IIb/IIIa inhibitors may be used as bridging therapy pre-operatively even though this approach has not been proven to improve outcomes.

Intra-operative and post-operative surveillance for myocardial ischaemia, infarction, and arrhythmias is important since peri-operative MI has been associated with 30–50% peri-operative morality and reduced long-term survival.38 Intra-operative and post-operative computerized ST-segment monitoring should be performed in all vascular surgery patients since ST-segment change (especially >30 min duration) is an independent predictor of peri-operative cardiac events. If monitoring is not available, electrocardiograms should be taken at baseline and post-operatively immediately and daily for 2 days. Post-operative troponin measurement (more sensitive and specific than CK-MB) is recommended in patients with ECG changes or chest pain typical of acute coronary syndrome. Post-operative elevation of cardiac biomarkers has been shown to be associated with significant two-fold increased long-term mortality,63 but routine biomarker measurement post-operatively is not recommended due to difficulties in distinguishing peri-operative myocardial damage from acute MI.38

Management of patients with combined carotid and coronary artery disease

The combination of carotid stenosis and CAD deserves special attention. A systematic review34 found the risk of stroke after CABG to be ~2%, and two-thirds of these strokes occurred >24 h post-operatively. Stroke risk increased to 3% in predominantly asymptomatic patients with a unilateral 50–99% stenosis,
5% in those with bilateral 50–99% stenoses and 7–11% in patients with carotid occlusion. However, >50% of stroke sufferers did not have significant carotid disease, and 60% of territorial infarctions were contralateral to significant carotid disease. Thus, even assuming that prophylactic CEA or percutaneous stenting carried no additional risk, it could only prevent ∼40–50% of procedural strokes. Other possible sources of post-CABG stroke include proximal aortic atherosclerosis (embolic due to surgical manipulation), cerebral hypoperfusion, or pump-related thromboembolism. Carotid artery disease was found to be a univariate risk factor for stroke. Most surgeons agree that CEA or stenting should be performed on symptomatic patients with >50% stenosis if the operative risk of stroke or death is <6% and in asymptomatic patients with >60% stenosis if the perioperative risk is <3%. The role of endarterectomy vs. stenting is still the subject of ongoing trials.

In patients with severe carotid stenosis as well as severe CAD requiring intervention in both vascular beds, the correct timing of the two surgeries is of utmost importance. In patients with severe but stable carotid disease, CABG or PCI for severe stable or unstable CAD should be performed first and the carotids operated on later. For patients with unstable carotid disease but stable and severe CAD, then CEA or carotid stenting should be performed first and CABB/PCI later. For patients with unstable CAD as well as unstable carotid disease, consideration for simultaneous CABB/PCI and CEA/stenting is recommended. This simultaneous surgical strategy is an option also for patients with severe CAD with critical carotid disease, but there are insufficient data to declare this strategy superior to CEA before CABB (Figure 7).}

**Conclusion**

In contrast to the Sherlock Holmes quote, the management of patients with combined CAD and PVD is never easy and is in general extremely complex. Early diagnosis of lower extremity PVD is essential for starting risk reduction therapy so as to reduce cardiovascular morbidity and mortality. Careful perioperative evaluation of the severity of disease in both the coronary and the peripheral circulation (lower extremity, aorta, or carotid artery) plus a clear identification of associated co-morbidities (such as diabetes, renal artery stenosis, etc) is required so as to optimally medically manage patients and if necessary plan timing of revascularization for patients with severe CAD undergoing vascular surgery. Close collaboration between internists, cardiologists, vascular surgeons, and anaesthetists is the key to successful management of such patients.

**References**


