Chula Med J Vol. 65 No. 1 January - March 2021;87-101 DOI : 10.14456/clmj.2021.14

Review article

Surgical management of lower extremity peripheral arterial disease

Natawat Narueponjirakul, Papawee Chennavasin

Department of Surgery, Faculty of Medicine, Chulalongkorn University and King Chulalongkorn Memorial Hospital, Bangkok, Thailand

Becoming to aging society era, numbers of patients with peripheral arterial disease (PAD) have been continuously increasing. PAD is a presentation of atherosclerotic diseases. Most of the time, the patients are accompanied with underlying diseases that increase risks of atherosclerosis. Treatments of PAD essentially require multidisciplinary team supports. Smoking cessation, controlled exercise program, medical control for underlying diseases must be performed at the beginning of treatments.

Revascularizations are indicated in the patients with unbearable symptoms and limb threatening ischemia. The reason why modern endovascular techniques have expeditiously been grown up is its advantage of less invasive and not to burn the bridge for future surgical bypass. However, its drawbacks are higher reintervention rates, contrast-induced nephropathy, radiation exposure, and lower long-term patency.

Keywords: Peripheral arterial disease, surgical management, lower extremity.

The term peripheral arterial disease (PAD) is classically defined as the various diseases that affect noncardiac, nonintracranial arteries. The most common cause of PAD is atherosclerosis; less common causes include inflammatory disorders of the arterial wall (vasculitis) and noninflammatory arteriopathies, such as fibromuscular dysplasia.[1] In this topic, PAD is confined as a chronic occlusive disease of lower extremities' arteries due to atherosclerosis. PAD of the lower extremities is the third leading cause of atherosclerotic cardiovascular morbidity, only following coronary artery disease and stroke.

In 2010, estimated 202 million people around the world are living with PAD. PAD usually associated with severe comorbidities especially coronary artery disease (CAD) and cerebrovascular disease (CVD) or both. CAD and CVD are presented in more than half of the patients diagnosed with PAD.[1]

***Correspondence to:** Natawat Narueponjirakul**,** Department of Surgery, Faculty of Medicine, Chulalongkorn University and King Chulalongkorn Memorial Hospital, Bangkok 10330, Thailand.
E-mail: natawat.n@chulahospital.org, natawat.n@chula.ac.th
Received: November 5, 2019
Revised: February 10, 2020
Accepted: March 20, 2020

Increasing the prevalence of PAD by age is recognized by a study from the United Nations (UN) in 2010. Of the 40 - 60 year age group, the prevalence of PAD is 3.0 – 10.0% and increases up to 20.0% in the population older than 60 years old.[2] In term of location, half of the PAD lesions occurred in the femoropopliteal artery. Patients with aortoiliac occlusive lesions are usually concomitant with the femoropopliteal disease.[3]

Risk factors

PAD is one of the clinical presentations of systemic atherosclerosis. As such, it shares many risk factors with other clinical atherosclerotic diseases such as CAD and CVD. However, some differences among PAD, CAD, and CVD profiles have been characterized and the strength of associated specific risk factors varies across the clinical syndromes, as well as across anatomic patterns.

Demographic

Age is the strongest risk factor of PAD. The disease is rare among individuals less than 40 years old and rises in prevalence in the sixth to eighth decades. Sex-related differences in PAD appear to be different around the world. Although some studies showed women in some age groups tend to have a lower ankle-brachial index (ABI) than men,

symptomatic PAD still predominates in men. Several studies have demonstrated higher prevalence and poorer outcome of PAD in the black populations compared to the non-Hispanic or Hispanic white populations.

Smoking

Cigarette smoking is a strong risk factor of PAD across the population. Several studies revealed an odds ratio in the range of twofold to fourfold. Also, secondhand smoking appears to also carry an increased risk for PAD, although the data in this regard are limited. Smoking cessation is associated with a reduced risk of PAD but it is likely to take more than 20 years of cessation to reduce the level of risk to individuals who have never smoked.[4]

Diabetes

Diabetes has been strongly related to PAD prevalence and severity. The association is directly related to the severity and duration of diabetes.[5] For every 1.0% increase in hemoglobin A1c level, the risk of PAD is increased by 26.0%.[6]

Hypertension

Hypertension has been associated with most epidemiological studies with an increased risk of PAD, but the relationship has not been as strong as those with smoking or diabetic. Although, systolic blood pressure (SBP) has been associated with PAD, diastolic blood pressure is often not associated with PAD. [7, 8]

Dyslipidemia

Elevated total cholesterol (TC) and reduced high-density lipoprotein (HDL) levels have been associated with PAD in multiple studies.[7, 9] In one study, the TC/HDL ratio has been associated with PAD.[10, 11]

Obesity

To date, the evidence failed to show a consistent relationship between obesity and PAD.[12] The reason for inconsistent is unclear, although it has been postulated that PAD in older people is often associated with other chronic illnesses that might contribute to weight loss.

Inflammation

Elevated biomarkers of systemic inflammation have been consistently associated with atherosclerosis in general and PAD specifically. Plasma levels of C-reactive protein and fibrinogen have shown consistent associations with PAD.[11, 13, 14] Circulating cytokines such as interleukin-6 and adhesion molecules are also elevated in PAD patients. [15]

Homocysteine

Some evidence has demonstrated a significant relationship between elevated plasma levels of homocysteine and PAD.[16] However, the strength of the association is moderate when controlled for other risk factors.[11]

Socioeconomic status

PAD has been associated with lower socioeconomic status, including lower levels of income and education.[17, 18] These associations span race/ethnicity and are only partly explained by traditional risk factors such as smoking.

Pathophysiology

Atherosclerosis is the chronic inflammatory process of the endothelial layer of the blood vessel. After an injury of endothelial cells, the inflammatory process is started with lipid, inflammatory cells such as monocyte, and fibrous material migrates into a wall of blood vessel forming of atherosclerotic plaque. It causes thickening of the blood vessels' walls and narrowing their lumens. Hence, blood flow to the end organs is reduced. This process is generalized all over the body. In consequence, the clinical syndrome can vary upon which end organs are affected, such as PAD, CAD, or CVD.[19] In term of PAD, patients with lower limb ischemia can have the following signs and symptoms, namely:

Pain

Pain occurs when blood flow reduction reaches the stage that until oxygen supply does not match oxygen demand. Anaerobic metabolism results in an increase in substance P. Small unmyelinated A-delta and C sensory fibers are stimulated, causing pain. Opposite to oxygen supply reduction, oxygen demand can be increased when patients have activities such as walking or running. Pain on activity is called "intermittent claudication (IC)", typically should be relieved when activity is stopped. IC can be aggravated at the buttock, thigh, calf or foot regarding the severity and location of atherosclerotic lesions (e.g., infrarenal aorta, iliac arteries, and superficial

femoral artery or lower, respectively). Whenever the disease progresses, the pain could occur even during the resting phase, so-called "ischemic rest pain". Ischemic rest pain may occur during sleeping while the cardiac output is decreased.

Tissue loss

Tissue loss[20] is a more severe form of PAD, which occurs when ischemia is high severity that makes the necrotic tissue. The two forms are described below, namely:

"Ischemic ulcer" is a result of repeated microtrauma in the ischemic area. A small wound cannot be healed because of inadequate blood flow. Shallow nonhealing pallid erosion is usually found on distal of the foot.

"Ischemic gangrene" mostly occurs on toes when blood flow is insufficient for tissue to survive and results in tissue necrotic. Rutherford category is used to characterize the severity of PAD (Table 1).[20]

Clinical presentations

Asymptomatic patients, the most common form of PAD, have 3 - 4 times more than the symptomatic group.[21] Asymptomatic patients had no difference in morbidity and mortality from a patient with IC.[22] Due to underlying diseases, such as congestive heart failure which can limits patient activity, diabetic neuropathy which blunts pain sensation, can lead to severe PAD or critical limb ischemia without IC.[21] Only 7.0% of asymptomatic patients progress to IC. Every 0.1 level dropping of ABI is associated with an increase in the risk of a major cardiovascular event by 10.0 - 13.0%.[23]

Normally, IC has slow progress. Five years after the onset of symptoms, 70.0 - 80.0% of the patients have stable claudication, only 10.0 - 20.0% will get worsen. Limb amputation will be warranted in 4.0 - 27.0%. Mortality is between 15.0 - 30.0%. Cardiovascular event is a cause of death in half of the patients.[21, 24]

Table 1. Rutherford category.[20]

Rutherford category	Clinical description	Objective criteria
0	Asymptomatic—no hemodynamically significant occlusive disease	Normal treadmill or reactive hyperemia test
1	Mild claudication	Completes treadmill exercise†; AP after exercise > 50 mmHg but at least 20 mmHg lower than resting value
2	Moderate claudication	Between categories 1 and 3
3	Severe claudication	Cannot complete standard treadmill exercise† and AP after exercise < 50 mmHg
4	Ischemic rest pain	Resting AP < 40 mmHg, flat or barely pulsatile ankle or metatarsal PVR; TP < 30 mmHg
5	Minor tissue loss—nonhealing ulcer, focal gangrene with diffuse pedal ischemia	Resting AP < 60 mmHg, ankle or metatarsal PVR flat or barely pulsatile; TP < 40 mmHg
6	Major tissue loss—extending above TM level, functional foot no longer salvageable	Same as category 5

Categories 4, 5, and 6 are embraced by the term chronic *critical* ischemia
†5 minutes at 3 km per hour on a 12.0% incline.

AP: ankle pressure, PVR: pulse volume record, TP: toe pressure, TM: transmetatarsal.

Critical limb ischemia (CLI) is defined when the patients present with ischemic rest pain, ischemic ulcer, or ischemic gangrene. Without intervention, amputation is required in 35.0% of the patients. One-fourth of CLI will die within one year, the most common cause of death is a cardiovascular event.[25]

Diagnosis

The diagnosis of PAD is based on clinical signs and symptoms. History taking and careful physical examination are essential for the diagnosis. Around 20.0 - 50.0% of the patients are asymptomatic.[3] Ankle-brachial index (ABI) is either a screening or diagnostic tool. The sensitivity and specificity of ABI for diagnosis are 79.0 - 95.0% and more than 95.0%.[3, 26] ABI less than 0.9 or ankle pressure less than 50 mmHg is a criterion for diagnosis in asymptomatic patients.[27, 28] The normal range of ABI is between 1.0 - 1.4.

Individuals who have ABI between 0.9 - 1.0 are borderline and require further investigation normally with a treadmill test. Patients would be tested by walking on a treadmill at the speed of 3 km per hour at 12.0% incline for 5 minutes. Normal response is a slight increase or no change in the ABI compared with baseline. If the patient develops no symptom while walking and does not have a corresponding decrease in ankle pressure, PAD is essentially ruling out. Other causes of leg symptoms should be investigated. A falling in ABI more than 20.0% of baseline or dropping ankle pressure below 60 mmHg that requires more than 3 minutes to recover, is considered abnormal.

As for patients who have ABI more than 1.4, the non-compressible vessels are suspected. So, the patients should be organized for further investigation such as toe-brachial pressure index (TBPI) or toe pressure measurement. PAD can be established when TBPI is less than 0.7. Furthermore, Toe pressure less than 30 mmHg indicates advanced ischemia.

Treatment

The goal of treatments of lower extremities PAD is quality of life improvement such as slowing disease progression in asymptomatic patients, alleviating claudication, and prevention of limb loss. Lifestyle modification is mandatory in all PAD patients. Atherosclerotic risk modification must be adopted. The target of blood pressure less than 130/80 mmHg, level lipoprotein cholesterol (LDL) level less than 70 mg/dL, and hemoglobin A1C level less than 6.5% are the goals of treatment.[26, 27]

Time to revascularization depends on the degree of severity of disease. Incapacitating claudication means the claudication causing loss of ability to perform any occupation or basic activity of daily living and critical limb ischemia (CLI) are indications for revascularization.[21] A recently wound classification is from the Society of Vascular Surgery, Risk stratifications is based on wound, ischemia, and foot infection (WIfI).[29] Each WIfI category (wound, ischemia, foot infection) is classified into 4 subgroups (grade 0 - 3); then calculates into 4 clinical staging (I - IV). Each stage carries a different level of risk of amputation and the chances of benefit from revascularization procedure. For instance, patients with a high grade of ischemia but no infection are likely to get benefit from revascularization in contrast to patients with high grade of infection without ischemia have higher chance of amputation and less likely benefit from revascularization.

Revascularization

Two different ways of revascularization are open surgery and endovascular therapy. The former is more invasive than the latter but it has fewer reintervention rates. The Bypass versus Angioplasty in Severe Ischemia of the Leg (BASIL) trial[30], a recent landmark study, comparing open vs. endovascular procedure, showed non-statistically significant difference in early mortality rate (open 5.0% vs. endovascular 3.0%) and more early morbidity in open repair group (57.0% vs. 41.0% in open and endovascular group). These morbidities were mainly infective, wound, and cardiovascular complications. The in-hospital cost of first 12-months follows-up in open-surgery group was estimated as £23,322 (£20,096 of hospital stay and £3,225 of procedure costs), which was about one-third time higher than for those enrolled an endovascular-first group (£17,419 (£15,381, £2,039)).[30] As an interim analysis, if considering as a whole follow-up period, there was no difference of survival between each group. In addition, for patients who survive longer than two years of randomizations, open surgery group was associated with a significant increase in subsequent overall survival and a trend of improved amputation free survival.[31]

According to the result of the BASIL trial, open surgical repair is usually recommended in PAD patients, who have estimated life expectancy.

Surgical risk

Many pre-operative assessment tools were established such as Revised Cardiac Risk Index (RCRI), American College of Surgeons National Surgical Quality Improvement Program (NSQIP) Myocardial Infarction and Cardiac Arrest (MICA), and Vascular Study Group of New England Cardiac Risk Index (VSG-CRI). As for infrainguinal bypass surgery, VSG-CRI has been proved an accurate tool for prediction, whereas NSQIP-MICA and RCRI provided underpredicted major cardiovascular events.[32]

The other concern for the endovascular procedure is contrast media exposure. One study showed a significant decline in renal function in patients who were treated with the endovascular intervention compared to those with surgical procedure.[33]

Localization of disease

The localization of disease and extent of occlusion are crucial factors for considering surgical revascularization. TransAtlantic Inter-Society Consensus for management of peripheral arterial disease (TASC-II) divided PAD lesions according to 3 regions; including aorto-iliac (Figure 1), femoral-popliteal (Figure 2), and below knee regions (Figure 3).[3, 34] Each region is sub-divided into 4 groups, i.e., TASC type A, B, C, and D lesions. Type A is a short or focal stenosis; type D is a chronic total occlusion; type B and C are intermediate lesions.

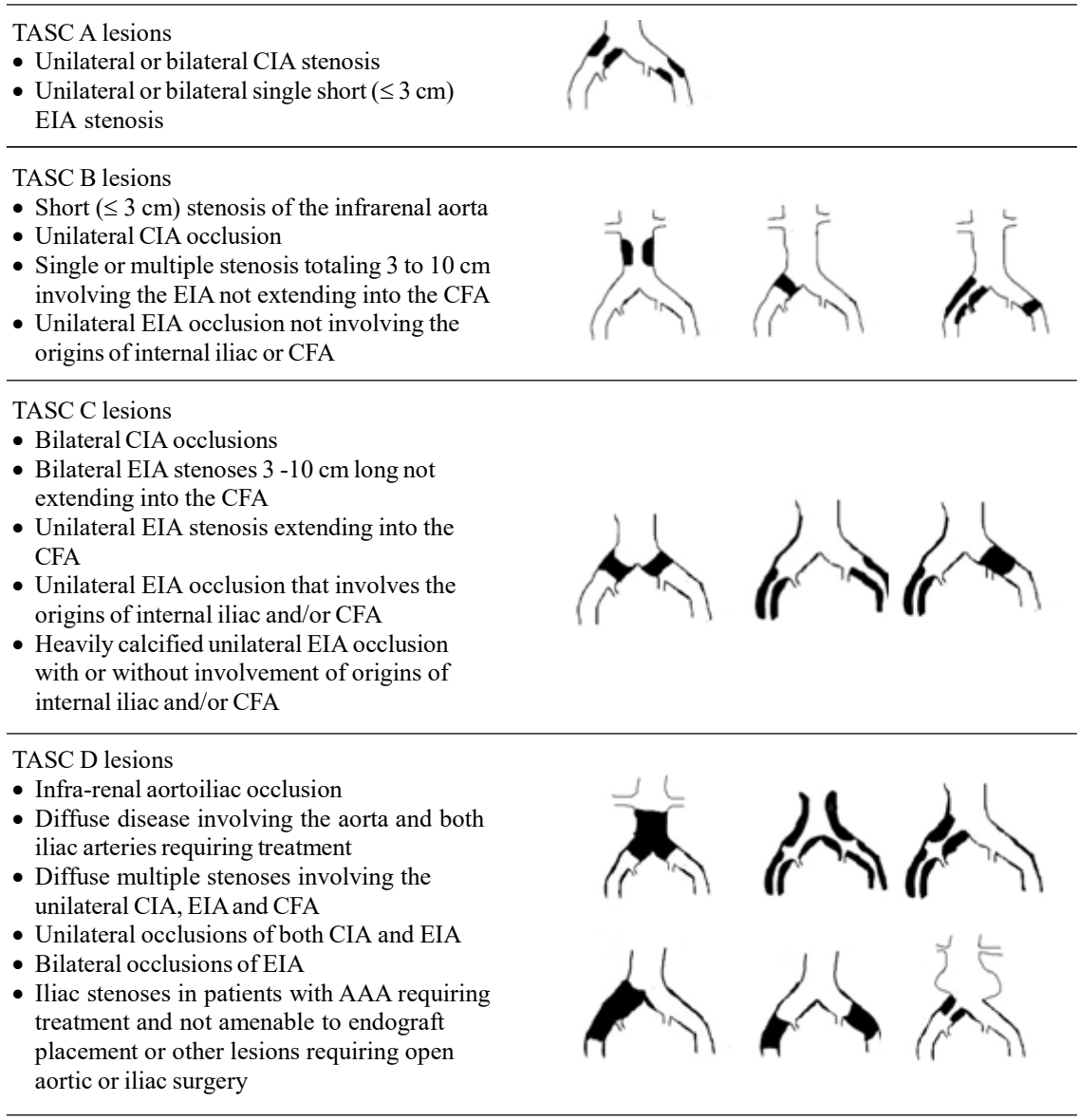


Figure 1. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC) classification of aortoiliac lesions. AAA, abdominal aortic aneurysm; CFA, common femoral artery; CIA, common iliac artery; EIA, external iliac artery.[3]

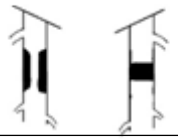
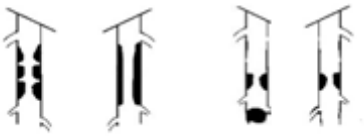
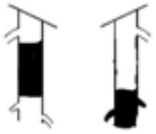
TASC A lesions • Single stenosis ≤ 10 cm in length • Single occlusion ≤ 5 cm in length	
TASC B lesions • Multiple lesions (stenoses or occlusions), each ≤ 5 cm • Single stenosis or occlusion ≤ 15 cm not involving the infra geniculate popliteal artery • Single or multiple lesions in the absence of continuous tibial vessels to improve inflow for a distal bypass • Heavily calcified occlusion ≤ 5 cm in length • Single popliteal stenosis	
TASC C lesions • Multiple stenoses or occlusions totaling >15 cm with or without heavy calcification • Recurrent stenoses or occlusions that need treatment after two endovascular interventions	
TASC D lesions • Chronic total occlusions of CFA or SFA (> 20 cm, involving the popliteal artery) • Chronic total occlusion of popliteal artery and proximal trifurcation vessels	

Figure 2. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC) classification of femoral-popliteal lesions. CFA, common femoral artery; SFA, superficial femoral artery.[3]

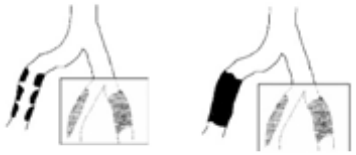
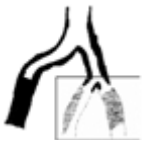
TASC A lesion • Single focal stenosis, ≤ 5 cm in length, in the target tibial artery with occlusion or stenosis of similar or worst severity in the other tibial arteries.	
TASC B lesion • Multiple stenosis, each ≤ 5 cm in length, or total length ≤ 10 cm or single occlusion ≤3 cm in length, in the target tibial artery with occlusion or stenosis of similar or worst severity in the other tibial arteries.	
TASC C lesion • Multiple stenosis in the target tibial artery and/or single occlusion with total lesion length > 10 cm with occlusion or stenosis of similar or worst severity in the other tibial arteries.	
TASC D lesion • Multiple occlusion involving the target tibial artery with total lesion length > 10 cm or dense lesion calcification or non-visualization of collaterals. The other tibial arteries occluded or dense calcification	

Figure 3. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC) classification of infrapopliteal lesions. The unshaded area represents the target lesion; the area inside the rectangle represents typical background disease.[3]

To date, the patients with TASC type A, B lesions have been recommended for endovascular intervention first while those with TASC type C and D are accompanied with a chance of failure by endovascular revascularization. According to new devices and higher technical (endovascular) skills, angioplasty first strategy or hybrid procedure may be extended to patients with type C and D lesions especially in the patients who are not fit for surgery.[34, 35]

The American College of Cardiology/American Heart Association (ACC/AHA) guideline[26] recommends open surgical procedure for patients with acceptable preoperative risk and technical factors advantage over endovascular treatment.

The European Society of Cardiology (ESC) guidelines[27] recommend as follows. For aortoiliac lesion, open surgery should be considered for a fit patient with an aortic occlusion extending up to the renal artery. Endovascular first is recommended for short occlusive lesion less than 5 cm. As for femoropopliteal occlusive lesions, open surgery is recommended for fit patients with long lesions more than 25 cm of superficial femoral artery lesions when available autologous vein and longer than two year life expectancy.

TASC classification focuses on lesion or involved arterial segment, however, lacking integrating complexity of disease pattern. The global limb anatomic grading system (GLASS) was developed to facilitate decision-making in CLI patient. By incorporating of a baseline assumption, GLASS is less complex and usable in everyday clinical practice and in future research.

GLASS introduced two new concepts, the target arterial path (TAP) and estimated limb-based patency (LBP). The TAP is defined by the treating surgeon or interventionist based on appropriate angiographic image as the optimal arterial pathway to restore in-line (pulsatile) flow to the ankle and foot. LBP is defined as maintenance of in-line flow throughout the TAP, from groin to ankle. The complexity of disease traverse by the TAP is integrated in the GLASS. Femoropopliteal (FP) and infrapopliteal (IP) arterial segments are individually graded on scale of 0 to 4. Using a consensus-based matrix, these grades are combined to three overall GLASS stage (I-III) for the limb.

GLASS includes a simplified approach to the inflow (aortoiliac disease), a dichotomous stratification of severe calcification in the segment, and a simple modifier for pedal (inframalleolar) disease. GLASS stages (I-III) were defined on basis of expected technical success and anatomical durability for infrainguinal endovascular intervention and reflect the overall complexity of disease within the TAP. Inflow disease (aortoiliac and CFA) is considered separately. Furthermore, the inframalleolar disease is not considered within the primary assignment of GLASS staging. In conclusion, GLASS emphasizes on infrainguinal to infrapopliteal disease and is based on some assumptions.[36]

Table 2. Aorto-iliac (inflow) disease staging in GLASS.[36]

I	Stenosis of the common and/or external iliac artery, chronic total occlusion of either common or external iliac artery (not both), stenosis of the infrarenal aorta; any combination of these
II	Chronic total occlusion of the aorta; chronic total occlusion of common and external iliac arteries; severe diffuse disease and/or small-caliber (< 6mm) common and external iliac arteries; concomitant aneurysm disease; severe diffuse in-stent restenosis in the AI system

A, no significant CFA disease; B, significant CFA disease (> 50.0% stenosis)
AI, Aortoiliac; CFA, common femoral artery.
A simplified staging system for inflow (AI and CFA) disease is suggested. Hemodynamically significant disease (> 50.0% stenosis) of the CFA is considered a key modifier (A/B)

Table 3. Infra-malleolar/Pedal descriptor in GLASS.[36]

P0	Target artery crosses ankle into foot, with intact pedal arch
P1	Target artery crosses ankle into foot; absent or severely diseased pedal arch
P2	No target artery crossing ankle into foot

Table 4. Femoropopliteal (FP) and infrapopliteal (IP) disease grading in GLASS. [36]

	0	1	2	3	4
FP	• Mild or no significant (< 50.0%) disease	• Total length SFA disease < 1/3 (< 10 cm) • May include single focal CTO (< 5 cm) as long as not flush occlusion • Popliteal artery with mild or no significant disease	• Total length SFA disease 1/3 - 2/3 (10 – 20 cm) • May include CTO totaling < 1/3 (10 cm) but not flush occlusion • Focal popliteal artery stenosis < 2 cm, not involving trifurcation	• Total length SFA disease > 2/3 (> 20 cm) length • May include any flush occlusion < 20 cm or non-flush CTO 10 - 20 cm long • Short popliteal stenosis 2 – 5 cm, not involving trifurcation	• Total length SFA occlusion > 20 cm • Popliteal disease > 5 cm or extending into trifurcation • Any popliteal CTO
IP	• Mild or no significant disease in the primary target artery path	• Focal stenosis of tibial artery < 3 cm	• Stenosis involving 1/3 total vessel length • May include focal CTO (< 3 cm) • Not including TP trunk or tibial vessel origin	• Disease up to 2/3 vessel length • CTO up to 1/3 length (may include tibial vessel origin but not TP trunk)	• Diffuse stenosis > 2/3 total vessel length • CTO > 1/3 vessel length (may include vessel origin) • Any CTO of TP trunk if AT is not the target artery

Trifurcation is defined as the termination of popliteal artery at the confluence of the anterior tibial (AT) artery and tibioperoneal (TP) trunk. CFA, common femoral artery; CTO, chronic total occlusion; SFA, superficial femoral artery

Table 5. Assignment of GLASS stage.[36]

		Infrainguinal GLASS stage (I-III)				
FP grade	4	III	III	III	III	III
	3	II	II	II	III	III
	2	I	II	II	II	III
	1	I	I	II	II	III
	0	NA	I	I	II	III
		0	1	2	3	4
		IP grade				

NA, Not applicable.
After selection of the target arterial path (TAP), the segmental femoropopliteal (FP) and infrapopliteal (IP) grades are determined from high-quality angiographic images. Using the table, the combination of FP and IP grades is assigned to GLASS stages I to III, which correlate with technical complexity (low, intermediate, and high) of revascularization.

Three GLASS stages were defined based on the likelihood of immediate technical failure (ITF) and 1-year LBP after endovascular intervention of the selected TAP. GLASS stages demonstrate a gradient of infrainguinal disease complexity.[36]

Stage I: low-complexity disease: expected ITF < 10.0% and 1-year LBP >70.0%
Stage II: intermediate-complexity disease: expected ITF < 20.0% and 1-year LBP 50.0 – 70.0%
Stage III: high-complexity disease: expected ITF > 20.0% and 1-year LBP < 50.0%

Surgical technique

Endarterectomy

Endarterectomy is a direct removal of obstructive plaque from an arterial segment and it is the best applied for focal lesions in large-caliber vessels, particularly at arterial bifurcations. The advantages of endarterectomy for aorto-iliac lesion are no conduit needed and improving erectile dysfunction.[37]

After longitudinal arteriotomy was performed, the atherosclerotic plague was bluntly removed from the deep medial layer of the arterial wall. There is a study that showing a 10-years patency rate of endarterectomy of aortoiliac lesions was at 85.7%.[38] With the advent of modern endovascular technology and technique, technical success rates and 5 to 10-year patency rates of endovascular therapy are comparable but less invasive to endarterectomy. These have largely led to an abandonment of endarterectomy in aortoiliac location.[39]

Arterial bypass

Three factors must have been considered to achieve successful arterial bypass procedures.

- The inflow artery is an artery proximal to the occlusive lesions.
- The outflow artery is a target artery distal to the obstructive lesions.
- Conduit, a bridge or duct, is categorized into an autogenous graft and a synthetic graft. Although an autogenous graft is superior to synthetic in terms of lower patency and infection rates, for the aortoiliac lesion, the vein graft size is too small conduit supplying bilateral outflow arteries of both legs. Polytetrafluoroethelene (PTFE) or polyester woven graft are used for surgical bypass of aortoiliac regions without a difference in patency rates. [40, 41] One study showed more distal anastomosis stenose in patients with PTFE graft for aortobifemoral bypass.[42]

Aortobifemoral bypass

Aortobifemoral bypass is indicated in diffuse aortoiliac disease. Transperitoneal, retroperitoneal, and laparoscopic approaches can be considered. Transperitoneal approach advantages in anatomic exposure. Although retroperitoneal approach may cause more difficult to expose of right renal and right iliac arteries, it can keep the patients away from morbidity of intraperitoneal operations such as pain, pulmonary complication, wound complication, ileus, etc. and allows shorter length of hospital stay.[43, 44] Laparoscopic aortobifemoral bypass reduces postoperative complications, provides good cost-effectiveness, and comparable patency rate to open surgery but spends longer operative time.[45] Twenty to thirty cases had usually required for learning curve[46] and the conversion rate was about 13.0%.[43]

The transperitoneal approach starts with long midline incision from xiphoid to the pubis symphysis followed by two longitudinal or oblique incisions at both groins. After inflow and outflow arteries are exposed and controlled, a proximal anastomosis is created. End-to-end anastomosis provides good in-line flow, less turbulence, fewer pseudoaneurysm rates, and better patency rate.[42] An end-to-side anastomosis is compulsory for those with patent bilateral common iliac and internal iliac arteries but occlusion of bilateral external iliac arteries. Since the end-to-side configuration provides native flow to bilateral internal iliac arteries, contrary to the end-to-end fashion that requires retrograde blood flow through the patent of external iliac arteries for prevention of pelvic organ and buttock ischemia (Figure 4). The next step is tunneling the graft underneath both ureters and performing end-to-side anastomosis from graft to both common femoral arteries or profunda femoral arteries in case of diffuse occlusion of the superficial femoral arteries.[47] The recommended size of the bifurcated graft is 18 by 9 mm for men and 16 by 8 mm for women.

Five-year and ten-year primary patency rates of aortobifemoral bypass are 85.0 - 90.0% and 75.0 - 85.0% respectively. This configuration has the longest patency compared to other extra-anatomical bypass configurations. Early mortality rates are between 4.0 - 4.4%. Half of the mortalities are caused by cardiovascular events.[3, 48]

Axillofemoral bypass

An axillofemoral bypass is an alternative option, in case that aortobifemoral bypass is contraindicated such as active intraabdominal infection or high expected perioperative risk for transabdominal operation.[49, 50] Since 5-year patency rates of axillobifemoral and axillounifemoral bypass are lower than that of aortobifemoral bypass (71.0% and 51.0% vs. 85.0 - 90.0%, respectively).[3] Axillary inflow selection is considered from a SBP of both arms if the difference between each arm is more than 10 mmHg, the higher one is selected for an inflow artery. If there is minimal difference in SBP, the

selected inflow artery is ipsilateral to the target lower extremity. A proximal anastomosis is usually medial to pectoralis minor muscle at first part of the axillary artery to avoid overstretching of anastomosis when patients abduct the arm. The graft is placed in the subcutaneous layer along with the midaxillary line. The most common selected graft size is 8 mm in diameter or 6-mm diameter in patients with small vessel size. No difference in primary patency between ring-supported and unsupported graft[51] (Figure 5).

Femoro-femoral bypass

Femoro-femoral bypass is indicated in unilateral iliac occlusive disease or performs with an axillofemoral bypass. Regional or local anesthesia can be considered for the procedure. Five-year patency rate is 70.0 - 75.0%.[3]

Regarding operative techniques, the graft is placed in a prefascial subcutaneous suprapubic plane. For patient with extremely thick or thin abdominal wall or previous surgery or radiotherapy of suprapubic (hypogastric) region, the graft is placed in a preperitoneal plane. Downside of preperitoneal graft placement is higher risk of bowel or bladder injury. End-to-side anastomosis fashion is applied on both femoral arteries. Inverted C configuration is common. Recommended anastomosis length is about 3 times of the graft diameter. The more distal anastomosis may be warranted in obese patients to reduce the chance of graft kinking.

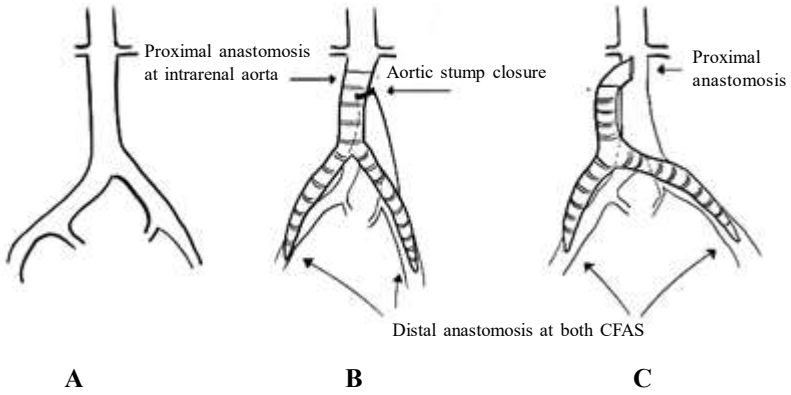


Figure 4. Aortobifemoral bypass; A: abdominal aorta, B: end-to-end proximal anastomosis, C: end-to-side proximal anastomosis.

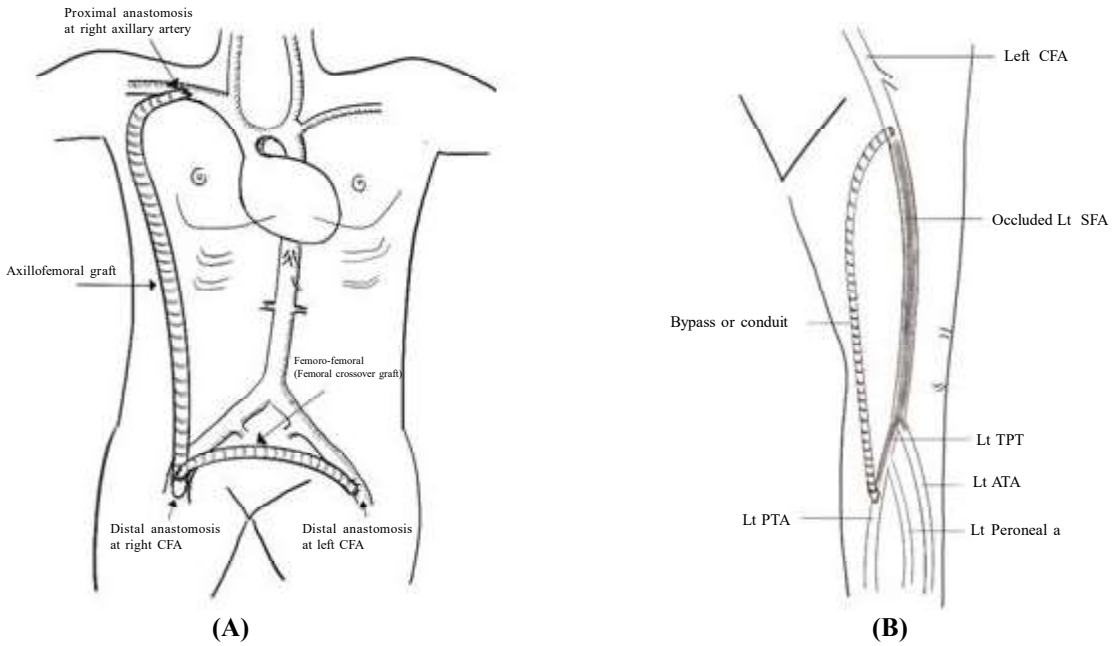


Figure 5. (A) axillofemoral bypass, (B) Infrainguinal bypass; Femoral to PTA bypass CFA, common femoral artery; SFA, superficial femoral artery; TPT, tibioperoneal trunk; ATA anterior tibial artery; PTA posterior tibial artery; peroneal a., peroneal artery.[3]

Infrainguinal (outflow) disease

Infrainguinal disease is generally defined as atherosclerosis extends down from the superficial femoral artery. Occlusion of outflow vessels is an indication for open surgical repair to fit patients with available autogenous vein graft.[3, 26, 27] Since endovascular treatment in long stenosis or occlusion lesions has a lower patency rate compared with surgical bypass, endovascular first concept can be considered for unfit patients. In the case of diffuse occlusive disease, inflow lesion is the priority for treatment.

Planning of surgical bypass requires consideration of 3 issues: inflow; outflow; and conduit. As previous mentioned, the selected inflow artery, proximal to the stenotic lesions, is either femoral artery or popliteal artery. Outflow artery should supply the arch of foot and length of bypass graft should be as short as possible.

Conduits are detours of blood flow from inflow to outflow artery. Many types of conduits have been used for bypass surgery.

Autogenous vein

Evidence showed longer primary patency and fewer complication rates in above-and below-knee femoropopliteal bypass by autogenous vein compared to prosthetic graft. [52] If available, the autogenous vein is the conduit of choice. The autogenous veins of choices are great saphenous vein, small saphenous vein, and arm vein[53], respectively.

Selection of a good autogenous vein graft based on preoperative evaluation; duplex venous ultrasonography can offer the vein sizing. Larger than 3mm in diameter is required and venosclerosis should be avoided.[54]

Prosthetic conduit

The advantage of prosthetic conduits is availability. The drawback is its shorter patency and a higher infection rate than autogenous vein graft. Prosthetic conduit is a second choice when autogenous vein graft is not available. Polyester graft has a higher 10-year patency rate than PTFE in the above-knee femoropopliteal bypass but not different in below the knee bypass. PTFE with vein cuff had risen the patency.[52, 55] Composite (spliced vein) graft, prosthetic with a vein graft, has similar patency, better secondary patency, more wound complication, and more reintervention rates compared to PTFE graft with vein cuff.[56]

Cryopreserved graft

Cryopreserves graft is a harvested cadaveric vessel preserved by cryopreservation. Some parts of the epithelial lining of blood vessels might be damaged resulting in delayed thrombosis and false aneurysm. Furthermore, the cost is expensive compared to the prosthetic graft and the patency rate is poor. The cryopreserved graft may be useful in infected prosthetic graft and an autogenous vein is unavailable.[57]

Human umbilical vein (HUV)

Human umbilical vein showed a higher patency rate than PTFE but late aneurysmal change can occur. So, HUV is rare in general practice.

Graft configuration

The two technique for autologous vein graft arrangement are reversed vein graft and in situ vein graft. The reversed vein bypass graft is performed by flipping vein graft, anastomosing distal site of the graft to inflow artery, then creating the distal anastomosis. The advantage is no requirement of valvulotomy, but the downside is a size discrepancy between vein graft and arterial inflow.

In situ vein bypass graft requires no fully dissection of vein graft from its bed but connection a proximal great saphenous vein to the femoral artery, then making distal anastomosis between the distal vein to outflow artery. In addition, this configuration requires valvulotomy. Evidence observed shows no difference in patency rates between the two configurations.[58, 59]

Complication

Systemic complications

Systemic complications in the surgical procedure of patients with PAD can be serious due to the patient's comorbidities. Major complications such as cardiovascular complications and stroke can be found at 4.7% and 1.7%, respectively.[60]

Local complications

Wound infection

Wound infection is an early complication and the most common cause of readmission. As for the lower extremities bypass, the wound infection rate is 4.8 – 17.0%. The risk factors includes hypertension, obesity, chronic kidney disease with long term dialysis, ABI less than 0.35, blood glucose higher than 180 mg/dl, operative time longer than 240 minutes,

blood transfusion more than 3 units, iodinated skin preparation, history of smoking, chronic obstructive pulmonary disease, congestive heart failure, and sepsis within 48 hours postoperatively.[61, 62]

Graft infection

Graft infection is a dreadful complication that can lead to devastating results included death or limb loss. In general, the treatment of infected graft should be graft removal with appropriate systemic antibiotic treatment. In the case of early infected autogenous vein graft that is not caused by *Pseudomonas aurugenosa* and no clinical of sepsis may be treated by local wound care and systemic antibiotics.[63, 64]

Graft thrombosis

Graft thrombosis can be classified into early (< 30 days) and late (> 30 days). Retained venous valves, anastomotic problems, inadequate local endarterectomy, clamp defects, or inadequate outflow can cause early graft thrombosis.[65] Thirty percent of late graft thrombosis that occurred in aortoiliac disease results from atherosclerosis progression at the anastomotic site. Other factors related to graft patency are the length of the bypass graft, the quality of the recipient artery, the extent of runoff to the foot, and the quality of conduit.[35]

False aneurysm

False aneurysm rates are around 1.0 - 5.0% in aortoiliac bypass. There are several causes such as infection, fabric degeneration, and following endarterectomy technique. Most of the time, false aneurysm at aortic anastomosis are discovered by surveillance imaging but pulsatile mass can be palpable at the common femoral anastomosis. The treatment of choice is debridement and open repair with interposition graft.[66]

Aortoenteric fistula

Aortoenteric fistula is a rare but lethal complication. This condition results from end-to-side anastomosis between aorta and graft eroding into the adjacent bowel (most common; third-part of duodenum), causing gastrointestinal bleeding that may be severe and lead to death. Operative correction is warranted. [67]

Postoperative follow-up

Routine surveillance should be performed at 4 to 6 weeks post-operative then every 6 months for the first year, then annually.[26] History taking, physical examinations, pulse examination at proximal artery, graft, outflow vessel, and ABI measurement are mandatory for surveillance. Symptoms and signs of limb ischemia or decrease ABI more than 0.15 indicates stenosis or occlusion. Surveillance imaging such as duplex ultrasonography (DUS) is more sensitive to detect graft thrombosis than ABI, especially in autogenous vein graft. DUS can estimate the degree of stenosis by calculating peak velocity ratio. Normal peak velocity ratio is less than 2.0. If the peak velocity ratio is more than 3.0, reintervention may be indicated.[68]

Conclusion

PAD results from atherosclerosis and is usually concomitant with several diseases or conditions such as hypertension, diabetes, hyperlipidemia, and smoking, etc. The physician should be suspicious and look for signs and symptoms of PAD in patients with risk factors. ABI is an easy screening and diagnostic tool. The goal of treatment is to increase the quality of life and prevent limb loss. Treatment consists of lifestyle modification, medical control of risk factors, and revascularization if indicated.

The keys to success in revascularization are patient selection, preoperative evaluation, planning, and meticulous surgical bypass. Post-operative long-term surveillance is mandated.

Acknowledgement

We would like thank Dr. Kritaya Kritayakirana, Dr. Suparerk Prichayudh, Dr. Apinan Uthaipaisarnwong, Dr. Punthita Aimsupanimitr, Dr. Sirinya Panya and Chulalongkorn Vascular and Trauma Team for all kind supports. And to my wife, Chawika Narueponjirakul, and my family, for their strong support of my academic endeavor.

References

1. Kullo IJ, Rooke TW. Clinical practice. Peripheral artery disease. N Engl J Med 2016;374:861-71.
2. Fowkes FG, Rudan D, Rudan I, Aboyans V, Denenberg JO, McDermott MM, et al. Comparison of global estimates of prevalence and risk factors for peripheral artery disease in 2000 and 2010: a systematic review and analysis. Lancet 2013;382:1329-40.

3. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, et al. Inter-society consensus for the management of peripheral arterial disease (TASC II). Eur J Vasc Endovasc Surg 2007;33 Suppl 1: S1-75.
4. Joosten MM, Pai JK, Bertoia ML, Rimm EB, Spiegelman D, Mittleman MA, et al. Associations between conventional cardiovascular risk factors and risk of peripheral artery disease in men. JAMA 2012;308: 1660-7.
5. Kallio M, Forsblom C, Groop PH, Groop L, Lepantalo M. Development of new peripheral arterial occlusive disease in patients with type 2 diabetes during a mean follow-up of 11 years. Diabetes Care 2003;26: 1241-5.
6. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, et al. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med 2004;141:421-31.
7. Meijer WT, Grobbee DE, Hunink MG, Hofman A, Hoes AW. Determinants of peripheral arterial disease in the elderly: the Rotterdam study. Arch Intern Med 2000;160:2934-8.
8. Newman AB, Siscovick DS, Manolio TA, Polak J, Fried LP, Borhani NO, et al. Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study. Cardiovascular Heart Study (CHS) Collaborative Research Group. Circulation 1993;88:837-45.
9. Murabito JM, D'Agostino RB, Silbershatz H, Wilson WF. Intermittent claudication. A risk profile from The Framingham Heart Study. Circulation 1997;96: 44-9.
10. Criqui MH, Vargas V, Denenberg JO, Ho E, Allison M, Langer RD, et al. Ethnicity and peripheral arterial disease: the San Diego Population Study. Circulation 2005;112:2703-7.
11. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. JAMA 2001; 285:2481-5.
12. Criqui MH, Aboyans V. Epidemiology of peripheral artery disease. Circ Res 2015;116:1509-26.
13. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Plasma concentration of C-reactive protein and risk of developing peripheral vascular disease. Circulation 1998;97:425-8.
14. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. Circulation 2004;110: 738-43.
15. Pradhan AD, Rifai N, Ridker PM. Soluble intercellular adhesion molecule-1, soluble vascular adhesion molecule-1, and the development of symptomatic peripheral arterial disease in men. Circulation 2002; 106:820-5.
16. Khandanpour N, Loke YK, Meyer FJ, Jennings B, Armon MP. Homocysteine and peripheral arterial disease: systematic review and meta-analysis. Eur J Vasc Endovasc Surg 2009;38:316-22.
17. Pande RL, Creager MA. Socioeconomic inequality and peripheral artery disease prevalence in US adults. Circ Cardiovasc Qual Outcomes 2014;7:532-9.
18. Vart P, Coresh J, Kwak L, Ballew SH, Heiss G, Matsushita K. Socioeconomic status and incidence of hospitalization with lower-extremity peripheral artery disease: Atherosclerosis risk in communities study. J Am Heart Assoc 2017;6. pii: e004995.
19. Bhatt DL, Steg PG, Ohman EM, Hirsch AT, Ikeda Y, Mas JL, et al. International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. JAMA 2006;295: 180-9.
20. Rutherford RB, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997;26:517-38.
21. Conte MS, Pomposelli FB, Clair DG, Geraghty PJ, McKinsey JF, Mills JL, et al. Society for Vascular Surgery practice guidelines for atherosclerotic occlusive disease of the lower extremities: management of asymptomatic disease and claudication. J Vasc Surg 2015;61(3 Suppl):2S-41S.
22. Diehm C, Allenberg JR, Pittrow D, Mahn M, Tepohl G, Haberl RL, et al. Mortality and vascular morbidity in older adults with asymptomatic versus symptomatic peripheral artery disease. Circulation 2009;120: 2053-61.
23. Leibson CL, Ransom JE, Olson W, Zimmerman BR, O'fallon WM, Palumbo PJ. Peripheral arterial disease, diabetes, and mortality. Diabetes Care 2004;27:2843-9.
24. Sigvant B, Lundin F, Wahlberg E. The risk of disease progression in peripheral arterial disease is higher than expected: A meta-analysis of mortality and disease progression in peripheral arterial disease. Eur J Vasc Endovasc Surg 2016;51:395-403.
25. Abu Dabrh AM, Steffen MW, Undavalli C, Asi N, Wang Z, Elamin MB, et al. The natural history of untreated severe or critical limb ischemia. J Vasc Surg 2015;62:1642-51.

26. Gerhard-Herman MD, Gornik HL, Barrett C, Barshes NR, Corriere MA, Drachman DE, et al. 2016 AHA/ACC Guideline on the management of patients with lower extremity peripheral artery disease: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 2017;135:e726-79.
27. Aboyans V, Ricco JB, Bartelink MEL, Bjorck M, Brodmann M, Cohnert T, et al. Editor's Choice - 2017 ESC Guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS). Eur J Vasc Endovasc Surg 2018;55:305-68.
28. Conte MS, Pomposelli FB. Society for Vascular Surgery Practice guidelines for atherosclerotic occlusive disease of the lower extremities management of asymptomatic disease and claudication. Introduction. J Vasc Surg 2015;61(3 Suppl):1S.
29. Mills JL, Sr., Conte MS, Armstrong DG, Pomposelli FB, Schanzer A, Sidawy AN, et al. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: risk stratification based on wound, ischemia, and foot infection (WIfI). J Vasc Surg 2014;59:220-34.e1-2.
30. Adam DJ, Beard JD, Cleveland T, Bell J, Bradbury AW, Forbes JF, et al. Bypass versus angioplasty in severe ischaemia of the leg (BASIL): multicentre, randomised controlled trial. Lancet 2005;366:1925-34.
31. Bradbury AW, Adam DJ, Bell J, Forbes JF, Fowkes FG, Gillespie I, et al. Bypass versus Angioplasty in Severe Ischaemia of the Leg (BASIL) trial: An intention-to-treat analysis of amputation-free and overall survival in patients randomized to a bypass surgery-first or a balloon angioplasty-first revascularization strategy. J Vasc Surg 2010;51(5 Suppl):5S-17S.
32. Moses DA, Johnston LE, Tracci MC, Robinson WP 3rd, Cherry KJ, Kern JA, et al. Estimating risk of adverse cardiac event after vascular surgery using currently available online calculators. J Vasc Surg 2018;67:272-8.
33. Sigterman TA, Bolt LJ, Krasznai AG, Snoeijs MG, Heijboer R, Schurink GH, et al. Loss of kidney function in patients with critical limb ischemia treated endovascularly or surgically. J Vasc Surg 2016;64: 362-8.
34. Jaff MR, White CJ, Hiatt WR, Fowkes GR, Dormandy J, Razavi M, et al. An update on methods for revascularization and expansion of the TASC lesion classification to include below-the-knee arteries: A supplement to the inter-society consensus for the management of peripheral arterial disease (TASC II). J Endovasc Ther 2015;22:663-77.
35. Conte MS. Critical appraisal of surgical revascularization for critical limb ischemia. J Vasc Surg 2013; 57(2 Suppl):8S-13S.
36. Conte MS, Bradbury AW, Kolh P, White JV, Dick F, Fitridge R, et al. Global vascular guidelines on the management of chronic limb-threatening ischemia. J Vasc Surg 2019;69:3S-125S e40.
37. Connolly JE, Price T. Aortoiliac endarterectomy: a lost art? Ann Vasc Surg 2006;20:56-62.
38. Inahara T. Evaluation of endarterectomy for aortoiliac and aortoiliofemoral occlusive disease. Arch Surg 1975;110:1458-64.
39. Vartanian SM, Conte MS. Surgical intervention for peripheral arterial disease. Circ Res 2015;116:1614-28.
40. Friedman SG, Lazzaro RS, Spier LN, Moccio C, Tortolani AJ. A prospective randomized comparison of Dacron and polytetrafluoroethylene aortic bifurcation grafts. Surgery 1995;117:7-10.
41. Roll S, Muller-Nordhorn J, Keil T, Scholz H, Eidt D, Greiner W, et al. Dacron vs. PTFE as bypass materials in peripheral vascular surgery—systematic review and meta-analysis. BMC Surg 2008;8:22.
42. Davidovic L, Vasic D, Maksimovic R, Kostic D, Markovic D, Markovic M. Aortobifemoral grafting: factors influencing long-term results. Vascular 2004; 12:171-8.
43. Kalko Y, Ugurlucan M, Basaran M, Nargileci E, Banach M, Alpagut U, et al. Comparison of transperitoneal and retroperitoneal approaches in abdominal aortic surgery. Acta Chir Belg 2008;108: 557-62.
44. Djedovic M, Rustempasic N, Djedovic S, Totic D, Solakovic S, Mujanovic E, et al. Comparison of transperitoneal and retroperitoneal approach in aorto iliac occlusive disease. Med Arch 2013;67:249-51.
45. Tiek J, Remy P, Sabbe T, D'hont C, Houthoofd S, Daenens K, et al. Laparoscopic versus open approach for aortobifemoral bypass for severe aorto-iliac occlusive disease—a multicentre randomised controlled trial. Eur J Vasc Endovasc Surg 2012;43: 711-5.
46. Fourneau I, Lerut P, Sabbe T, Houthoofd S, Daenens K, Nevelsteen A. The learning curve of totally laparoscopic aortobifemoral bypass for occlusive disease. How many cases and how safe? Eur J Vasc Endovasc Surg 2008;35:723-9.
47. Menard MT, Shah SK, Belkin M. Aortoiliac disease: direct reconstruction. In: Sidawy AN, Perler BA, editors. Rutherford's vascalar surgery and endovascular therapy. 9th ed. Vol. 2. Philadelphia, PA: Elsevier; 2014. p. 1397-415.
48. Hertzer NR, Bena JF, Karafa MT. A personal experience with direct reconstruction and extra-anatomic bypass

for aortoiliofemoral occlusive disease. J Vasc Surg 2007;45:527-35.
49. Nguyen KP, Perrone KH, Rahman A, Azarbal AF, Liem TK, Mitchell EL, et al. The role of axillofemoral bypass in current vascular surgery practice. Am J Surg 2016;211:968-71.
50. Dickas D, Verrel F, Kalff J, Koscielny A. Axillobifemoral bypasses: reappraisal of an extra-anatomic bypass by analysis of results and prognostic factors. World J Surg 2018;42:283-94.
51. Johnson WC, Lee KK. Comparative evaluation of externally supported Dacron and polytetrafluoroethylene prosthetic bypasses for femorofemoral and axillofemoral arterial reconstructions. Veterans Affairs Cooperative Study #141. J Vasc Surg 1999;30:1077-83.
52. Twine CP, McLain AD. Graft type for femoro-popliteal bypass surgery. Cochrane Database Syst Rev 2010;(5): CD001487.
53. Faries PL, Logerfo FW, Arora S, Pulling MC, Rohan DI, Akbari CM, et al. Arm vein conduit is superior to composite prosthetic-autogenous grafts in lower extremity revascularization. J Vasc Surg 2000;31: 1119-27.
54. Mills JL Sr. Infrainguinal disease: surgical treatment. In: Sidawy AN, Perler BA, editors. Rutherford's vascalar surgery and endovascular therapy. 9th ed. Vol. 2. Philadelphia, PA: Elsevier; 2014. p. 1438-62.
55. Rychlik IJ, Davey P, Murphy J, O'Donnell ME. A meta-analysis to compare Dacron versus polytetrafluroethylene grafts for above-knee femoropopliteal artery bypass. J Vasc Surg 2014;60:506-15.
56. Kreienberg PB, Darling RC 3rd, Chang BB, Champagne BJ, Paty PS, Roddy SP, et al. Early results of a prospective randomized trial of spliced vein versus polytetrafluoroethylene graft with a distal vein cuff for limb-threatening ischemia. J Vasc Surg 2002;35: 299-306.
57. Hartranft CA, Noland S, Kulwicki A, Holden CR, Hartranft T. Cryopreserved saphenous vein graft in infrainguinal bypass. J Vasc Surg 2014;60:1291-6.
58. Moody AP, Edwards PR, Harris PL. In situ versus reversed femoropopliteal vein grafts: long-term follow-up of a prospective, randomized trial. Br J Surg 1992;79:750-2.
59. Conte MS. Challenges of distal bypass surgery in patients with diabetes: patient selection, techniques, and outcomes. J Am Podiatr Med Assoc 2010;100: 429-38.
60. Conte MS, Bandyk DF, Clowes AW, Moneta GL, Seely L, Lorenz TJ, et al. Results of PREVENT III: a multicenter, randomized trial of edifoligide for the prevention of vein graft failure in lower extremity bypass surgery. J Vasc Surg 2006;43:742-51.
61. Gibson A, Tevis S, Kennedy G. Readmission after delayed diagnosis of surgical site infection: a focus on prevention using the American College of Surgeons National Surgical Quality Improvement Program. Am J Surg 2014;207:832-9.
62. Davis FM, Sutzko DC, Grey SF, Mansour MA, Jain KM, Nypaver TJ, et al. Predictors of surgical site infection after open lower extremity revascularization. J Vasc Surg 2017;65:1769-78.
63. Calligaro KD, Veith FJ, Schwartz ML, Dougherty MJ, DeLaurentis DA. Differences in early versus late extracavitary arterial graft infections. J Vasc Surg 1995;22:680-5.
64. Sriussadaporn S. The infected vascular graft. Chula Med J 1991;35:751-64.
65. Shoenfeld NA, O'Donnell TF, Bush HL Jr, Mackey WC, Callow AD. The management of early in situ saphenous vein bypass occlusions. Arch Surg 1987; 122:871-5.
66. Markovic DM, Davidovic LB, Kostic DM, Maksimovic ZL, Kuzmanovic IB, Koncar IB, et al. False anastomotic aneurysms. Vascular 2007;15:141-8.
67. Batt M, Jean-Baptiste E, O'Connor S, Saint-Lebes B, Feugier P, Patra P, et al. Early and late results of contemporary management of 37 secondary aortoenteric fistulae. Eur J Vasc Endovasc Surg 2011; 41:748-57.
68. Olojugba DH, McCarthy MJ, Naylor AR, Bell PR, London NJ. At what peak velocity ratio value should duplex-detected infrainguinal vein graft stenoses be revised? Eur J Vasc Endovasc Surg 1998;15:258-60.