Deep venous thrombosis and limb ischaemia

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Deep vein thrombosis

Predisposing factors for venous thromboembolism

Strong risk factors (odds ratio >10)
Fracture of lower limb
Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months)
Hip or knee replacement
Major trauma
Myocardial infarction (within previous 3 months)
Previous venous thromboembolism
Spinal cord injury
Moderate risk factors (odds ratio 2-9)
Arthroscopic knee surgery
Auto-immune diseases
Blood transfusion
Central venous lines
Chemotherapy
Congestive heart or respiratory failure
Erythropoiesis-stimulating agents
Hormone replacement therapy (depends on formulation)
In vitro fertilization

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European Heart Journal (2014):doi:10.1093/eurheartj/ehu283

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Predisposing factors for VTE (cont'd)

Infection (specifically pneumonia, urinary tract infection and HIV)
Inflammatory bowel disease
Cancer (highest risk in metastatic disease)
Oral contraceptive therapy
Paralytic stroke
Postpartum period
Superficial vein thrombosis
Thrombophilia
Weak risk factors (odds ratio <2)
Bed rest >3 days
Diabetes mellitus
Hypertension
Immobility due to sitting (e.g. prolonged car or air travel)
Increasing age
Laparoscopic surgery (e.g. cholecystectomy)
Obesity
Pregnancy
Varicose veins

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Inherited VE

Inherited thrombophilia

- Factor V Leiden mutation
- Prothrombin gene mutation
- Protein S deficiency
- Protein C deficiency
- Antithrombin (AT) deficiency
- Rare disorders
- Dysfibrinogenemia


SUPERDUPER HEROES
LOOK, A LETTER FROM MY DOCTOR... I HAVE TO WEAR TIGHTS TO PREVENT DVT WHEN FLYING AT 30,000 FEET. SO WHAT IS YOUR EXCUSE?
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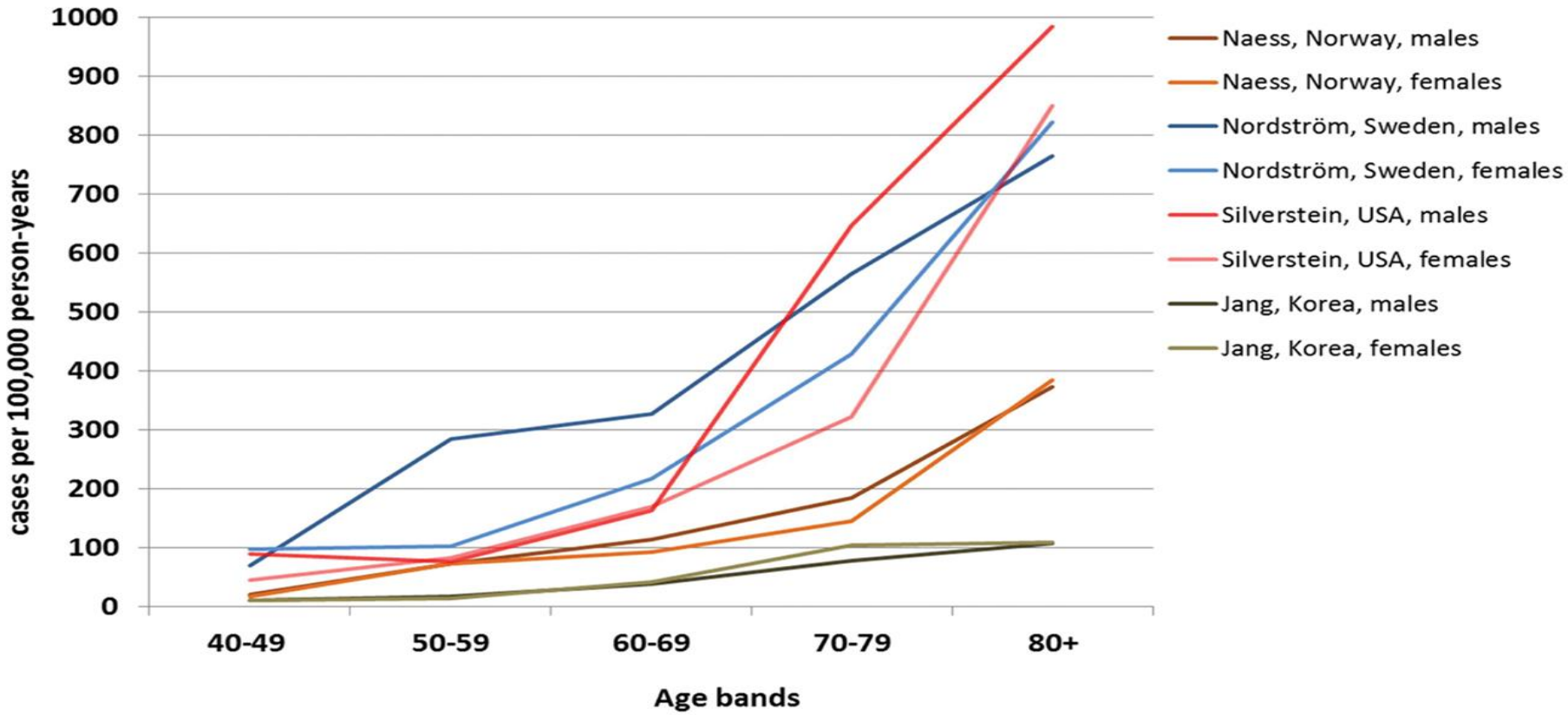
Figure 1 Venous thromboembolism incidence according to age group.



European Heart Journal, Volume 39, Issue 47, 14 December 2018, Pages 4208–4218, https://doi.org/10.1093/eurheartj/ehx003

Figure 2 Proposed deep vein thrombosis diagnostic and management algorithm. AC, anticoagulation; DOAC, direct oral ...

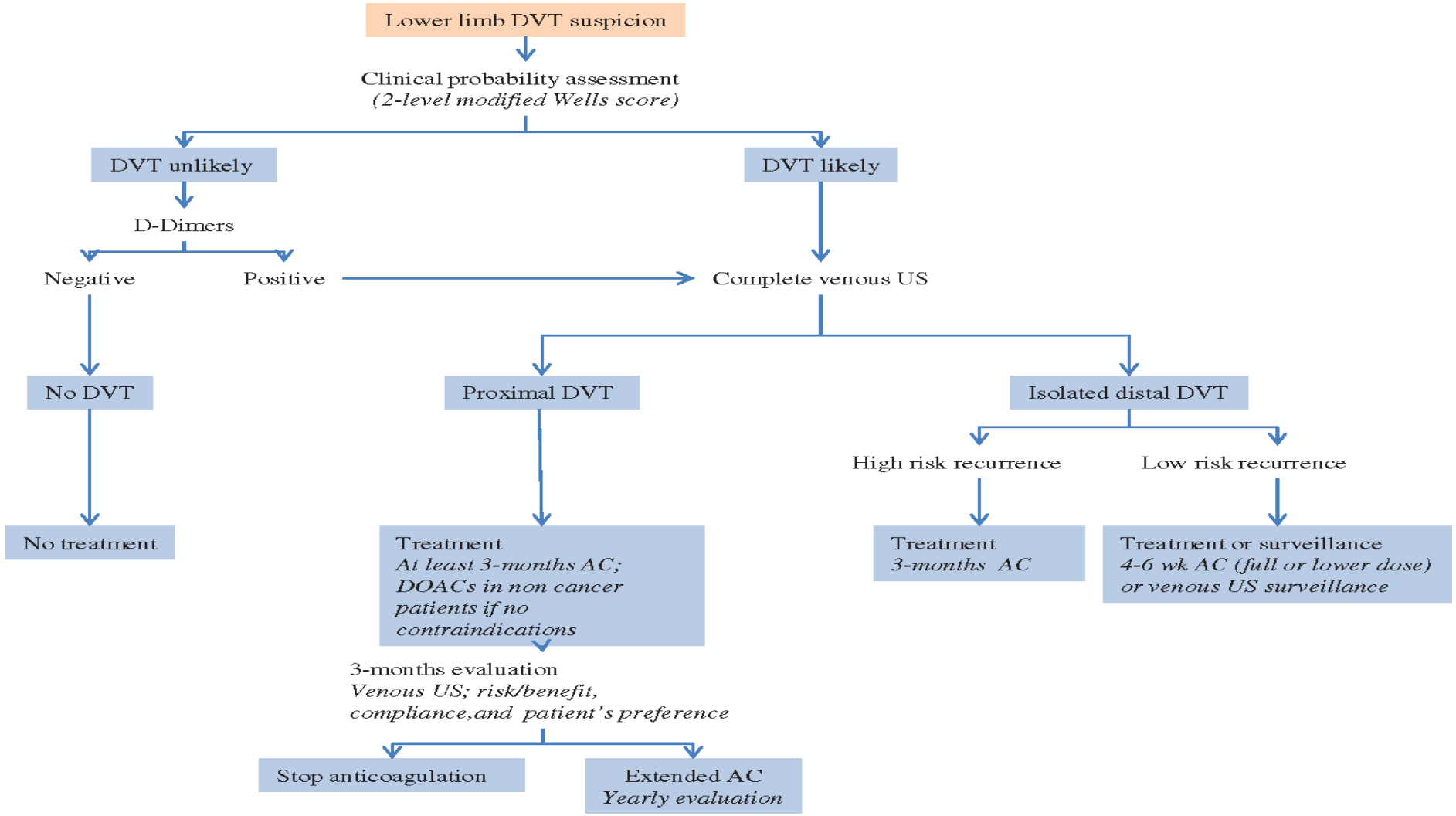


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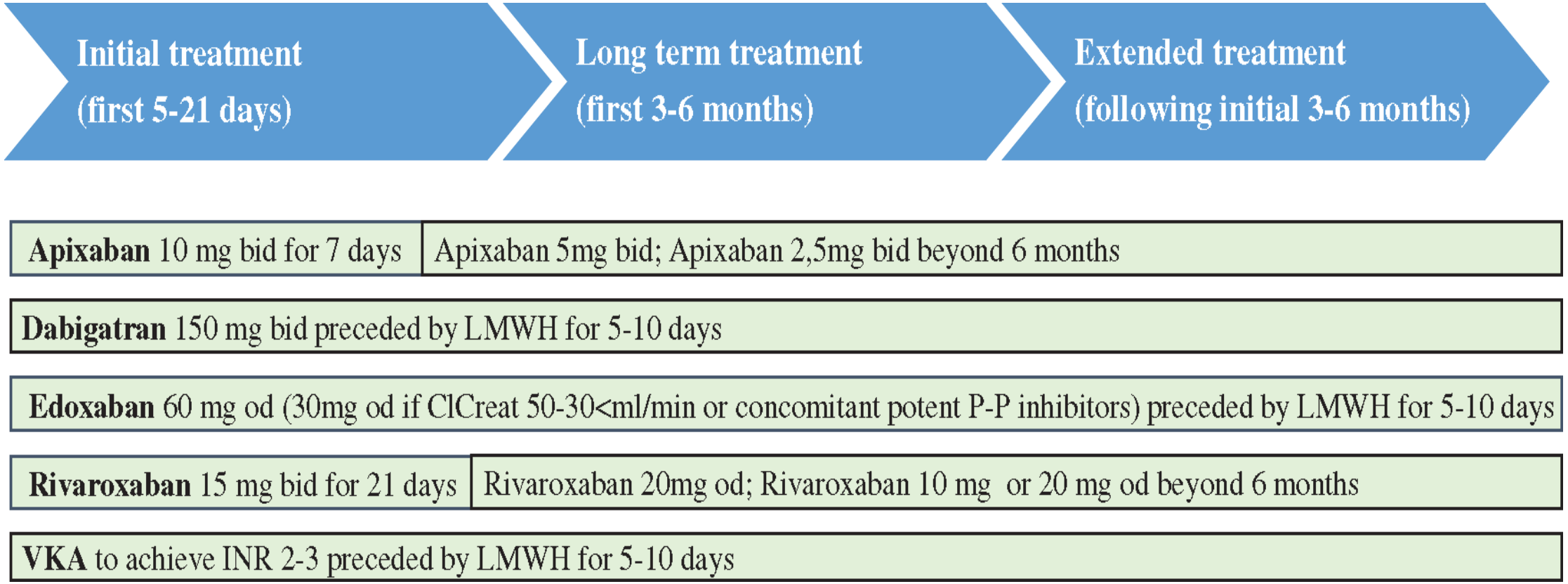
Table 2 The Wells score[12,13]

Clinical variable	Points
Active cancer (treatment ongoing or within previous 6 months or palliative)	+1
Paralysis, paresis or recent plaster immobilization of the lower extremities	+1
Recently bedridden for 3 days or more, or major surgery within the previous 12 weeks requiring general or regional anesthesia	+1
Localized tenderness along the distribution of the deep venous system	+1
Entire leg swelling	+1
Calf swelling at least 3 cm larger than that on the asymptomatic leg (measured 10 cm below the tibial tuberosity)	+1
Pitting edema confined to the symptomatic leg	+1
Collateral superficial veins (non varicose)	+1
Previously documented DVT	+1
Alternative diagnosis at least as likely as DVT	-2
Three-level Wells score	
Low	<1
Intermediate	1–2
High	>2
Two-level Wells score	
Unlikely	≤1
Likely	≥2

Figure 3 Deep vein thrombosis treatment phases. ClCreat: creatinine clearance; LMWH: low molecular weight heparin; P-P ...



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Consensus statement: initial and long-term management:

- Patients with proximal DVT should be anticoagulated for at least 3-months.
- Patients with isolated distal DVT at high-risk of recurrence should be anticoagulated, as for proximal DVT; for those at low risk of recurrence shorter treatment (4–6 weeks), even at lower anticoagulant doses, or ultrasound surveillance may be considered.
- In the absence of contraindications, DOACs should be preferred as first-line anticoagulant therapy in non-cancer patients with proximal DVT.
- Adjuvant CDT may be considered in selected patients with ilio-common femoral DVT, symptoms <14 days, and life expectancy >1 year if performed in experienced centres.
- Primary acute DVT stenting or mechanical thrombus removal alone are not recommended.
- Vena cava filters may be considered if anticoagulation is contraindicated, their use in addition to anticoagulation is not recommended.
- Compression therapy associated with early mobilization and walking exercise should be considered to relieve acute venous symptoms.

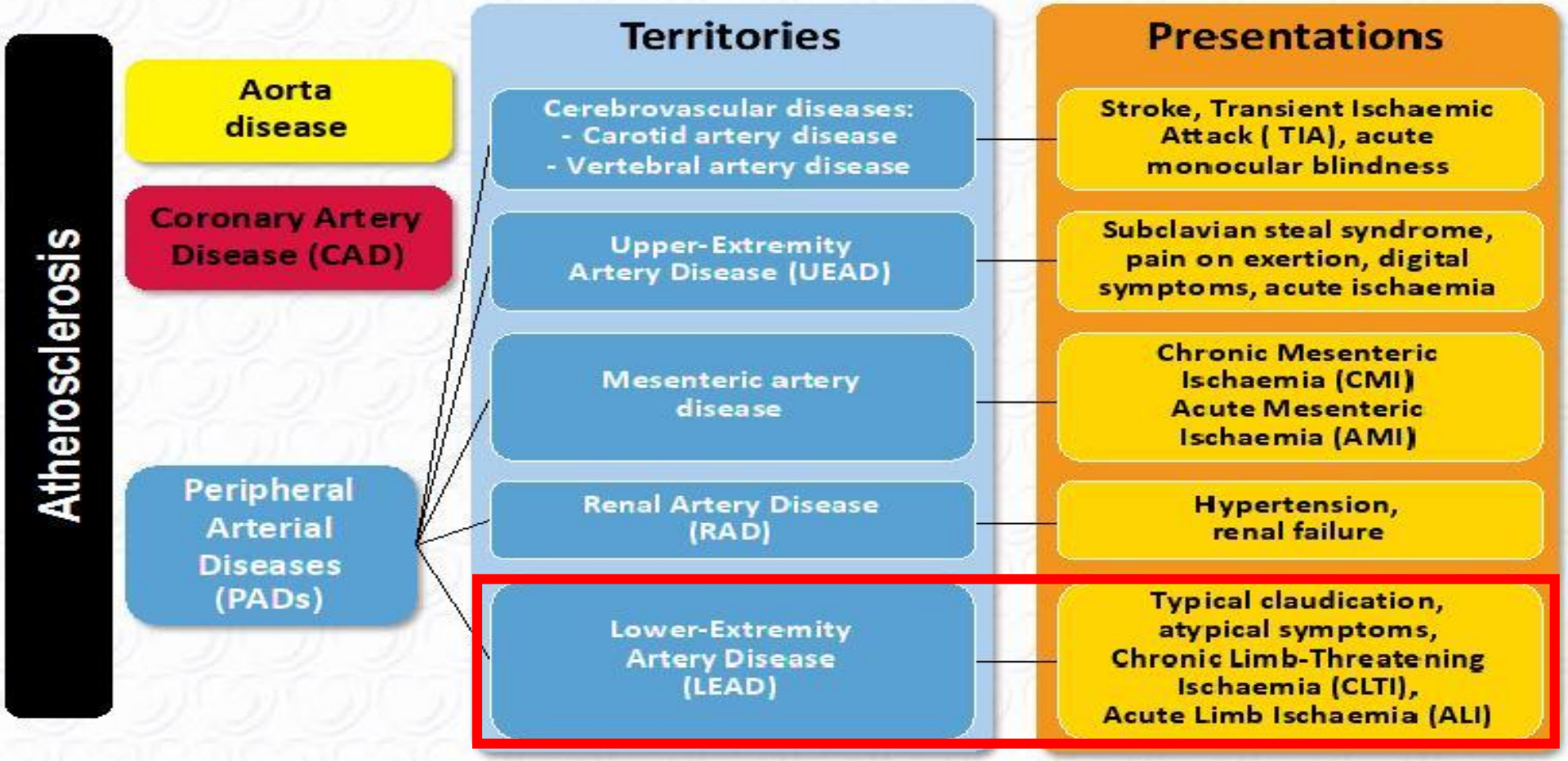
Table 4. Risk of recurrence after a first episode of unprovoked VTE

Risk factors for DVT recurrence		
Proximal DVT location	Male sex	Persistence of residual vein thrombosis at ultrasound
Obesity	Non-zero blood group	High D-dimer values
Old age	Early PTS development	Role of inherited thrombophilia is controversial

Clinical prediction rules assessing risk of recurrent VTE after first episode of unprovoked VTE[71]			
Score	**Vienna prediction model**	**DASH score**	**HERDOO-2**
Parameters	• D-dimer level at 3 weeks and 3, 9, 15, 24 months after stopping anticoagulation • Male sex • VTE location (Distal DVT, Proximal DVT, PE)	• Abnormal D-dimer 3–5 weeks after stopping anticoagulation • Male sex • Age<50 years • VTE not associated with oestrogen-progestatif therapy in women	• Abnormal D-dimer before stopping anticoagulation • Post thrombotic symptoms (hyperpigmentation, edema and redness) • Age ≥65 years • BMI ≥30
Validation study	Yes	Yes	Yes
Commentaries	Different nomograms are available to calculate risk of VTE recurrence at different time	Patients with low score (≤1) have an annual recurrence rate of 3.1%	It is applicable in women only. Women with low score (≤1) have an annual recurrence rate of 1.3%

Limb ischaemia

Presentations of Peripheral Arterial Diseases (PADs)



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Main points of medical history for assessment of peripheral arterial diseases

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Family history of CVD (coronary artery disease, cerebrovascular disease, aortic aneurysm,LEAD), and premature CVD (fatal or non-fatal CVD event or/and established diagnosis of CVD in first degree male relatives before 55 years or female relatives before 65 years).
Personal history of: • Hypertension • Diabetes • Dyslipidaemia • Smoking (present and/or past), passive smoking exposure • Chronic kidney disease • Sedentary life • Dietary habits • History of cancer radiation therapy • Psycho-social factors • Prior CVD
Transient or permanent neurological symptoms.
Arm exertion pain, particularly if associated with dizziness or vertigo.
Symptoms suggesting angina, dyspnoea.

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Main points of medical history for assessment of peripheral arterial diseases *(continued)*

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Abdominal pain, particularly if related to eating and associated with weight loss.
Walking impairment/claudication: • type: fatigue, aching, cramping, discomfort, burning, • location: buttock, thigh, calf, or foot, • timing: triggered by exercise, uphill rather than downhill, quickly relieved with rest; chronic, • distance.
Lower limb pain (including foot) at rest, and evolution at upright or recumbent position.
Poorly healing wounds of the extremities.
Physical activity assessment: • functional capacity and causes of impairment.
Erectile dysfunction.

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Physical examination for assessment of peripheral arterial diseases

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Auscultation and palpation of cervical and supraclavicular areas.
Careful inspection of upper extremities, including hands (i.e. colour, skin integrity).
Palpation of upper extremity pulses.
Blood pressure measurement of both arms and notation of inter-arm difference.
Auscultation at different levels including the flanks, peri-umbilical region, and groin.
Abdominal palpation, palpation of femoral, popliteal, dorsalis pedis, and posterior tibial artery pulses, temperature gradient assessment.

Physical examination for assessment of peripheral arterial diseases *(continued)*

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Careful inspection of lower limbs, including feet (i.e. colour, presence of any cutaneous lesion). Findings suggestive of lower extremity arterial disease, including calf hair loss and muscle atrophy, should be noted.
Peripheral neuropathy assessment in case of diabetes or LEAD: sensory loss (monofilament testing), ability to detect pain and light touch (sharp examination pin, cotton wool), vibration impairment (128 Hz tuning fork); deep tendon reflexes examination; sweating.

Laboratory testing in patients with peripheral arterial diseases

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Routine tests
Fasting plasma glucose.
Fasting serum lipid profile: • total cholesterol, • triglycerides, • high-density lipoprotein cholesterol, • low-density lipoprotein cholesterol.
Serum creatinine and creatinine clearance.
Urine analysis: urinary protein by dipstick test, microalbuminuria.
• Blood count. • Uric acid.

Laboratory testing in patients with peripheral arterial diseases *(continued)*

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Additional tests, based on findings from clinical history, physical examination and routine tests
Either glycated haemoglobin if fasting plasma glucose >5.6 mmol/L (101 mg/dL) or impaired glucose tolerance test when there is doubt.
Lipoprotein(a) if there is a family history of premature cardio-vascular disease.
Quantitative proteinuria if positive dipstick test.

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The Ankle-Brachial Index

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1. Who should have an ABI measurement in clinical practice?

- Patients with clinical suspicion for LEAD:
  - lower extremities pulse abolition and/or arterial bruit,
  - typical intermittent claudication or symptoms suggestive for LEAD,
  - non-healing lower extremity wound.
- Patients at risk for LEAD because of the following clinical conditions:
  - atherosclerotic diseases: CAD, any PADs,
  - other conditions: AAA, CKD, heart failure.
- Asymptomatic individuals clinically-free but at-risk for LEAD:
  - men and women aged >65 years,
  - men and women aged <65 years classified at high CV risk according the ESC Guidelines,
  - men and women aged >50 years with family history for LEAD.

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The Ankle-Brachial Index *(continued)*

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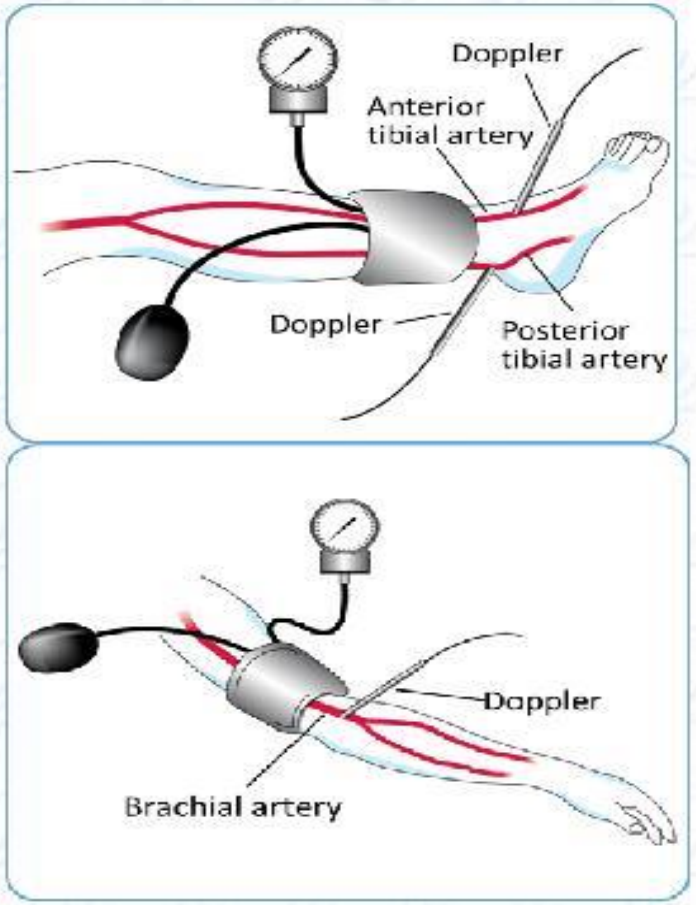
2. How to measure the ABI?

Supine position, cuff placed just above the ankle, avoid wounded zones. After a 5-10 minute rest, the SBP is measured by a Doppler probe (5-10 MHz) on the posterior and the anterior tibial (or dorsal pedis) arteries of each foot and on the brachial artery of each arm. Automated BP cuffs are mostly not valid for ankle pressure and may overestimate results in case of low ankle pressure. The ABI of each leg is calculated by dividing the highest ankle SBP by the highest arm SBP.



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The Ankle-Brachial Index *(continued)*

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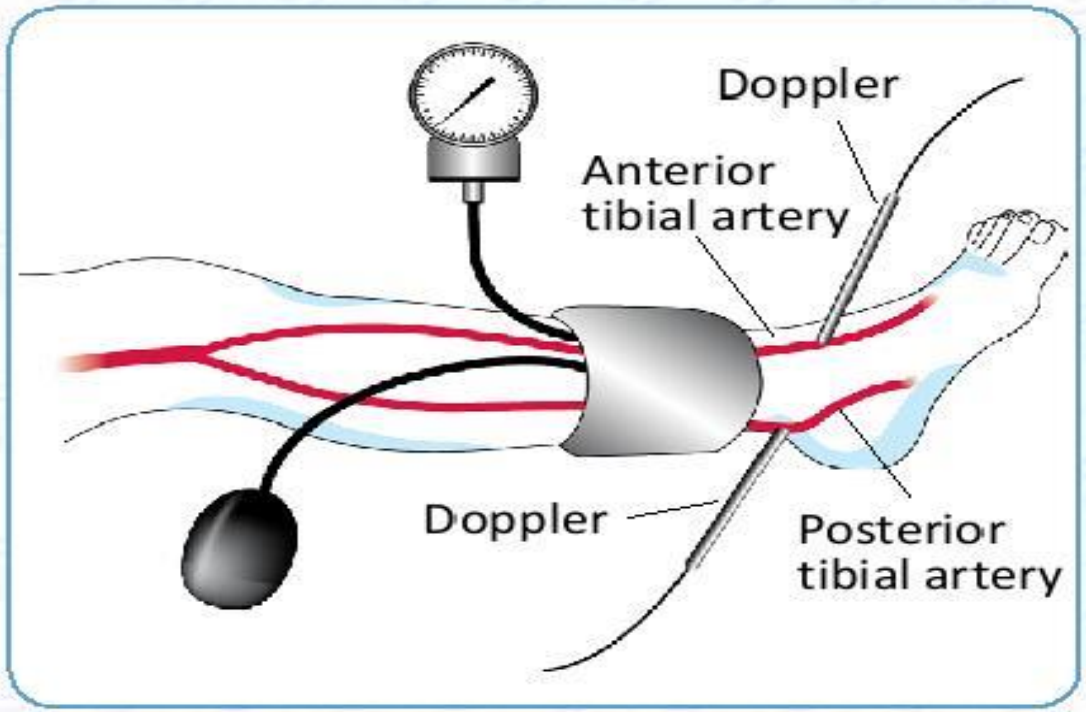

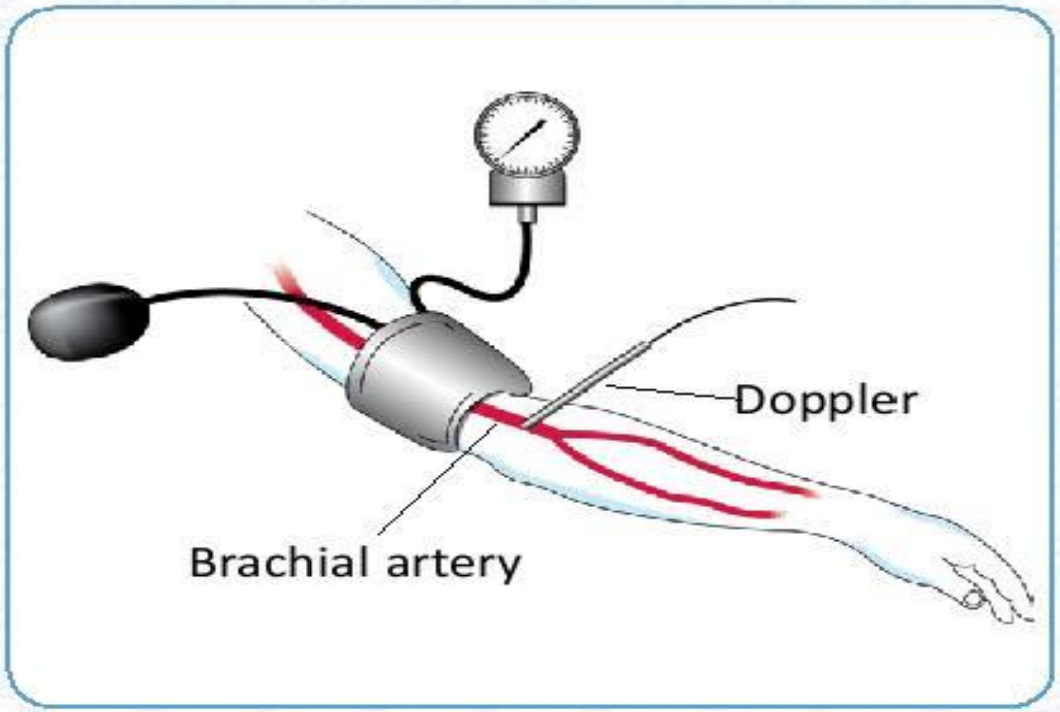

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The Ankle-Brachial Index *(continued)*

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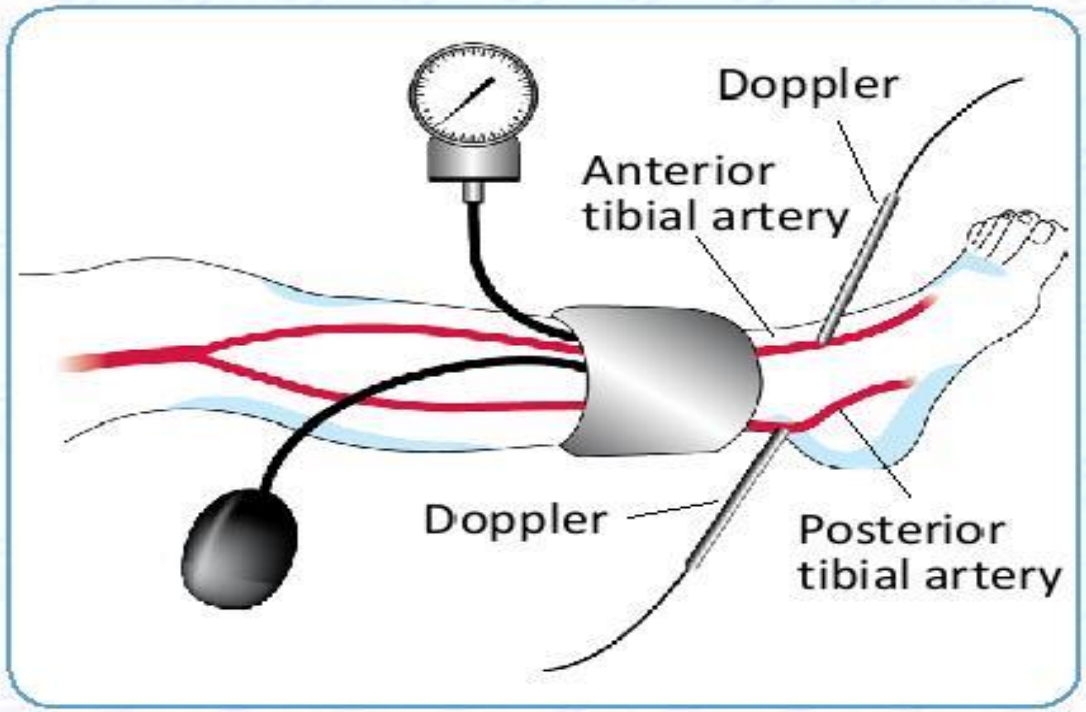

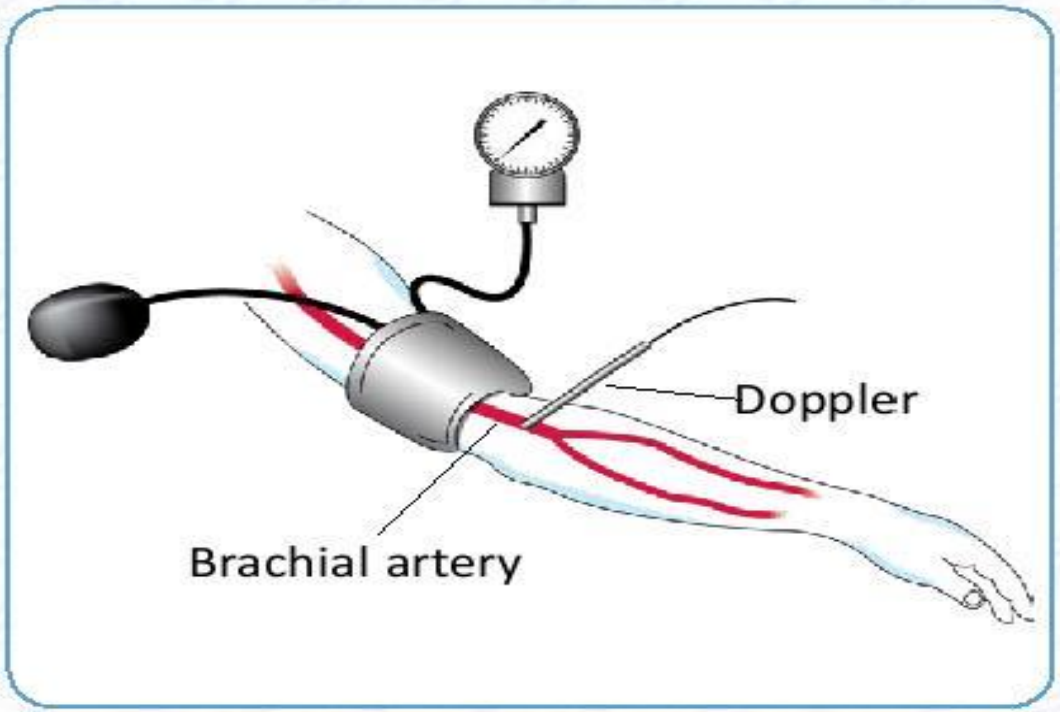

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The Ankle-Brachial Index *(continued)*

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3. How to interpret the ABI?

- For diagnosis of LEAD interpret each leg separately (one ABI per leg).
- For the CV risk stratification: take the lowest ABI between the two legs.
- Interpretation:

Imaging in patients with LEAD

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Recommendations	Class	Level
DUS is indicated as first-line imaging method to confirm LEAD lesions.	I	C
DUS and/or CTA and/or MRA are indicated for anatomical characterization of LEAD lesions and guidance for optimal revascularization strategy.	I	C
Data from an anatomical imaging test should always be analyzed in conjunction with symptoms and haemodynamic tests prior to treatment decision.	I	C
DUS screening for AAA should be considered.	IIa	C

Patients with peripheral arterial diseases: best medical therapy

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Recommendations	Class	Level
Smoking cessation is recommended in all patients with PADs.	I	B
Healthy diet and physical activity are recommended for all patients with PADs.	I	C
Statins are recommended in all patients with PADs.	I	A
In patients with PADs, it is recommended to reduce LDL-C to <1.8 mmol/L (70 mg/dL) or decrease it by ≥50% if baseline values are 1.8-3.5 mmol/L (70-135 mg/dL).	I	C

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Patients with peripheral arterial diseases: best medical therapy *(continued)*

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Recommendations	Class	Level
In diabetic patients with PADs, strict glycaemic control is recommended.	I	C
Antiplatelet therapy is recommended in patients with symptomatic PADs.	I	C
In patients with PADs and hypertension, it is recommended to control blood pressure at <140/90 mmHg.	I	A
ACEIs or ARBs should be considered as first line therapy in patients with PADs and hypertension.	IIa	B

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Antiplatelet therapy in patients with lower extremity artery disease

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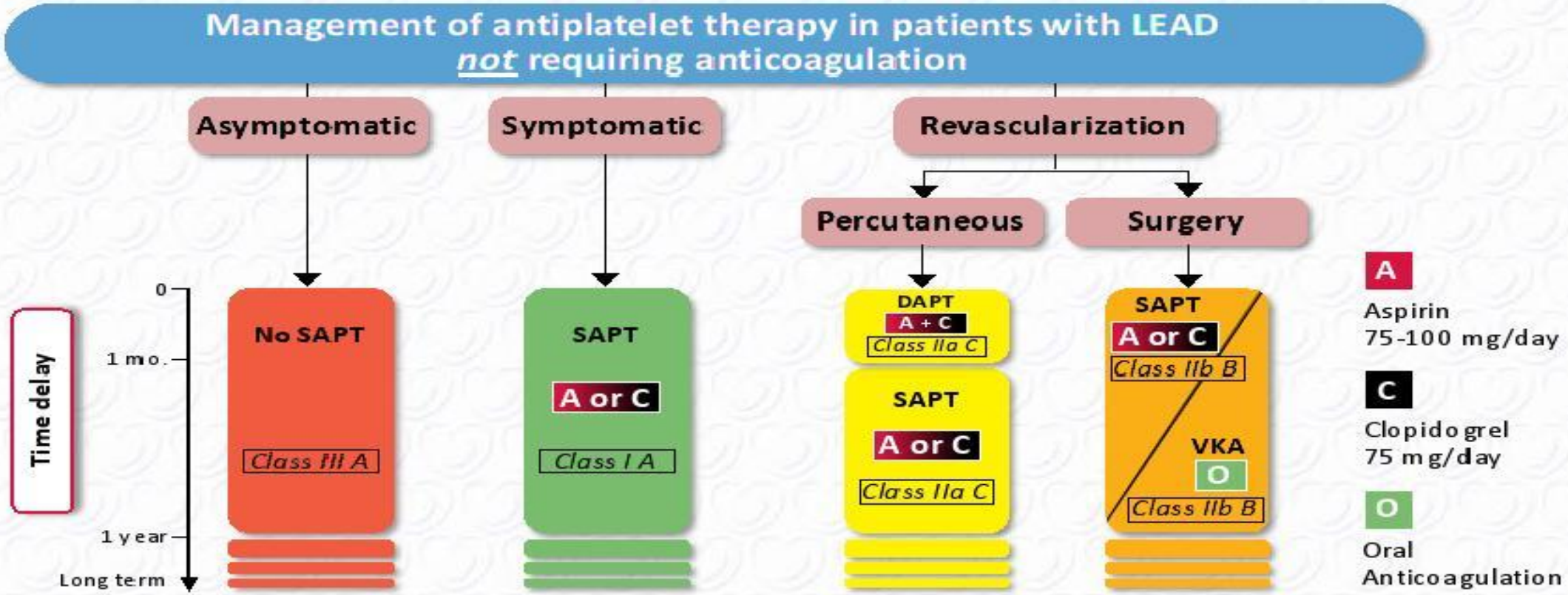

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Antithrombotic therapy in patients with LEAD requiring oral anticoagulation

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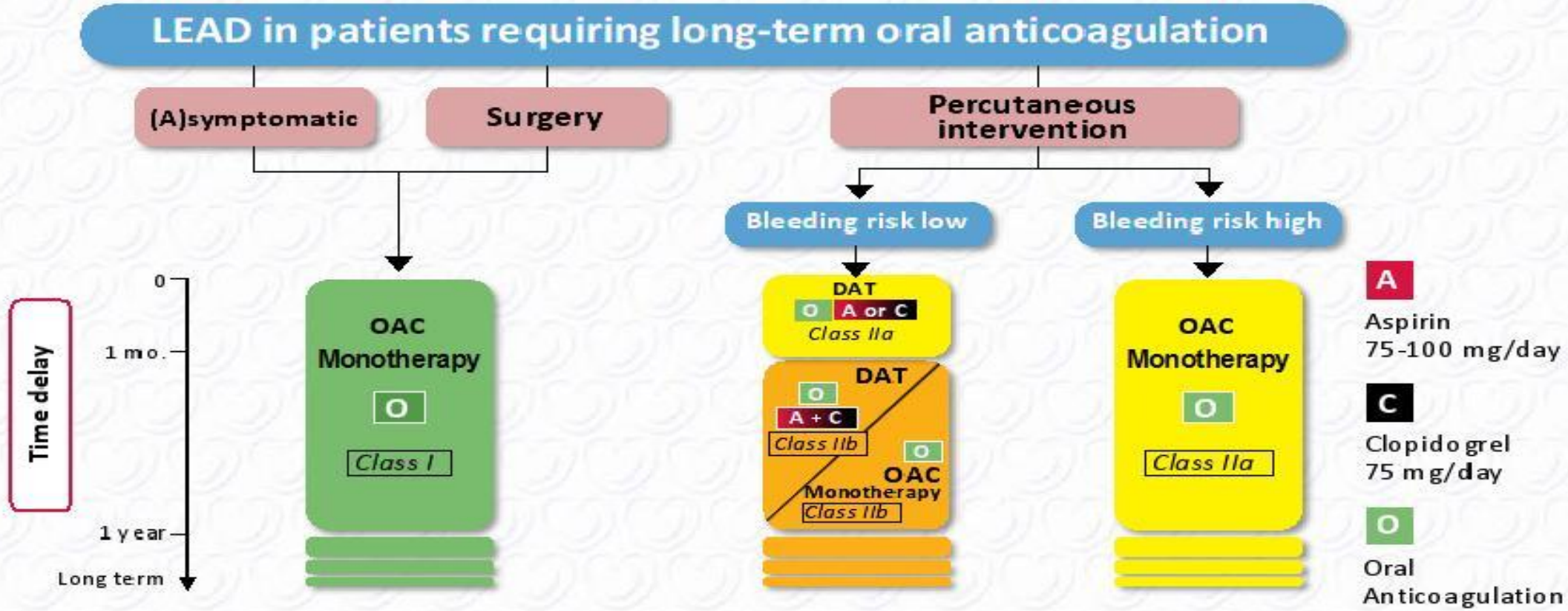

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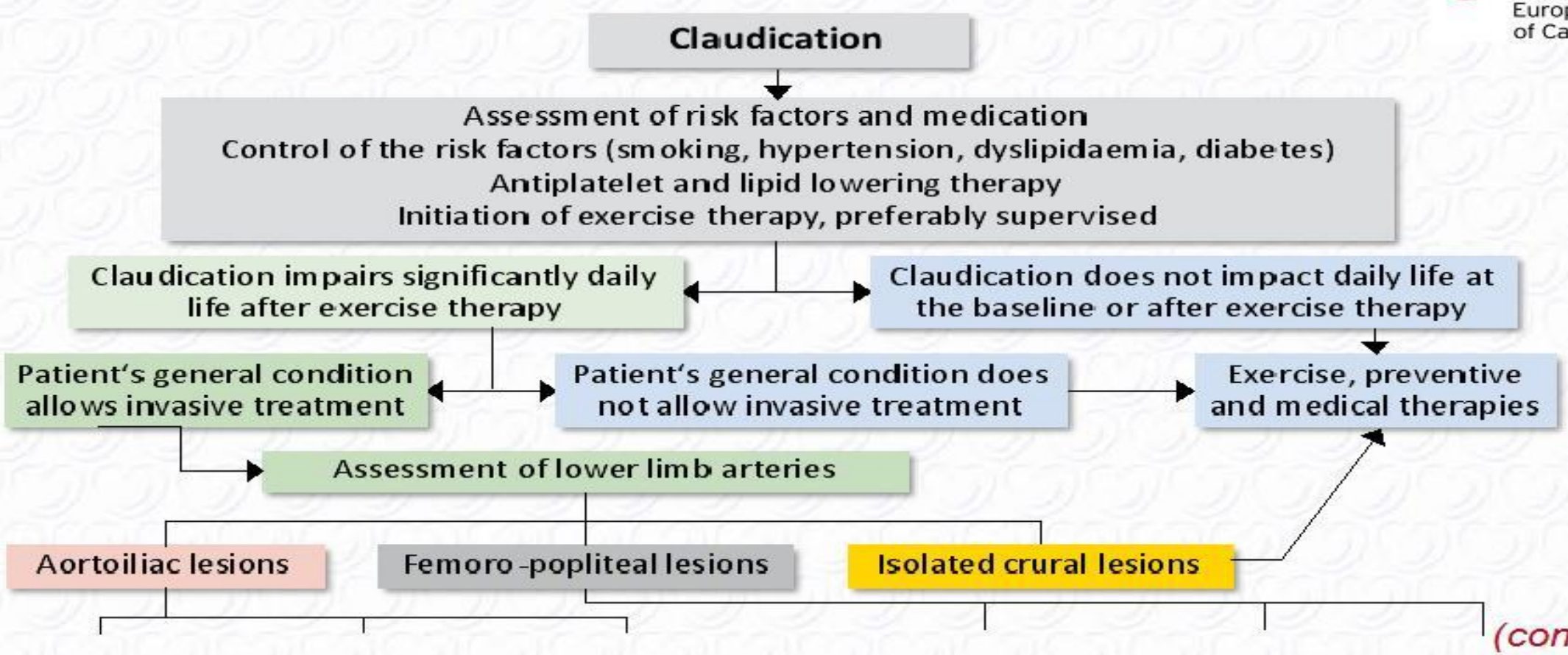
Management of patients with intermittent claudication



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Management of patients with intermittent claudication

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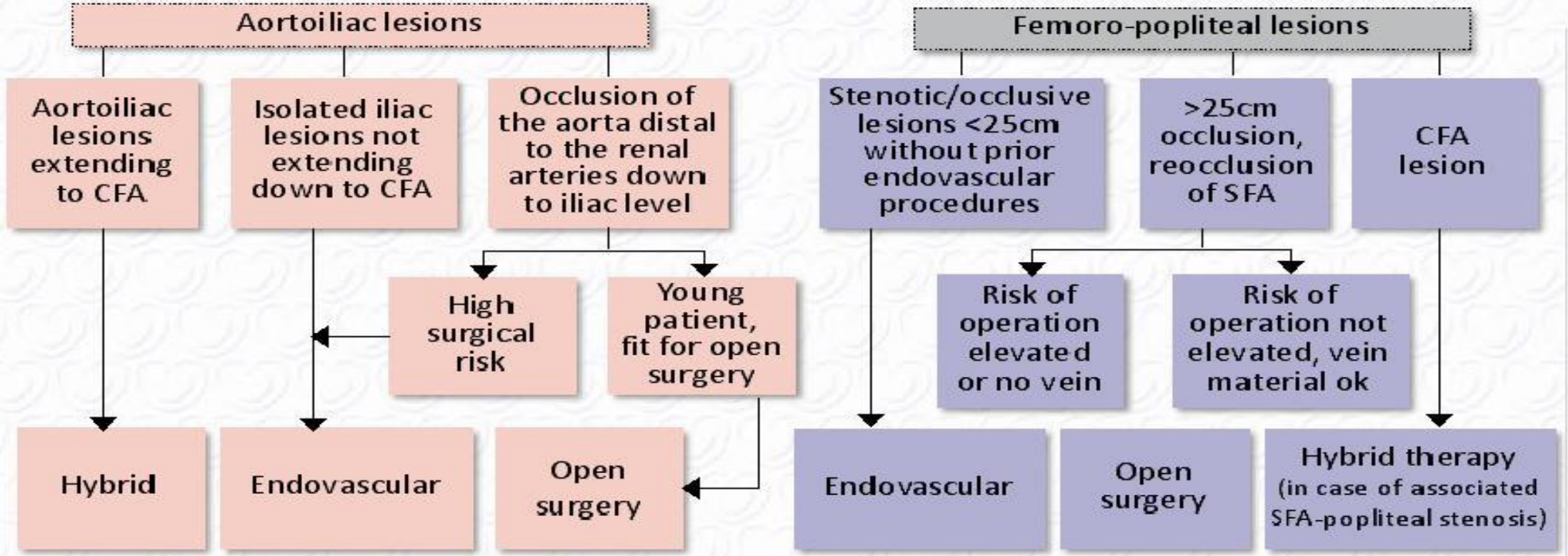

Post interventional exercise therapy and risk factor modification after ANY intervention

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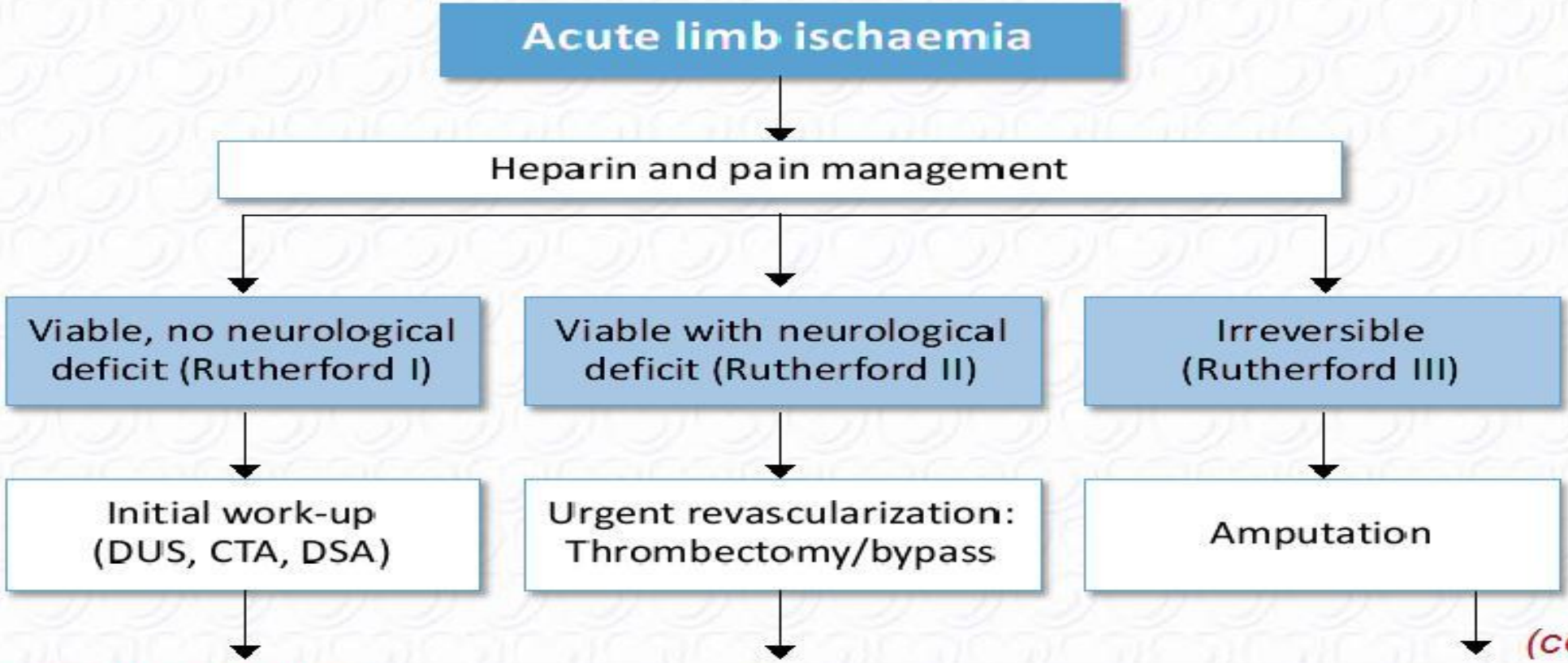
Management of acute limb ischaemia



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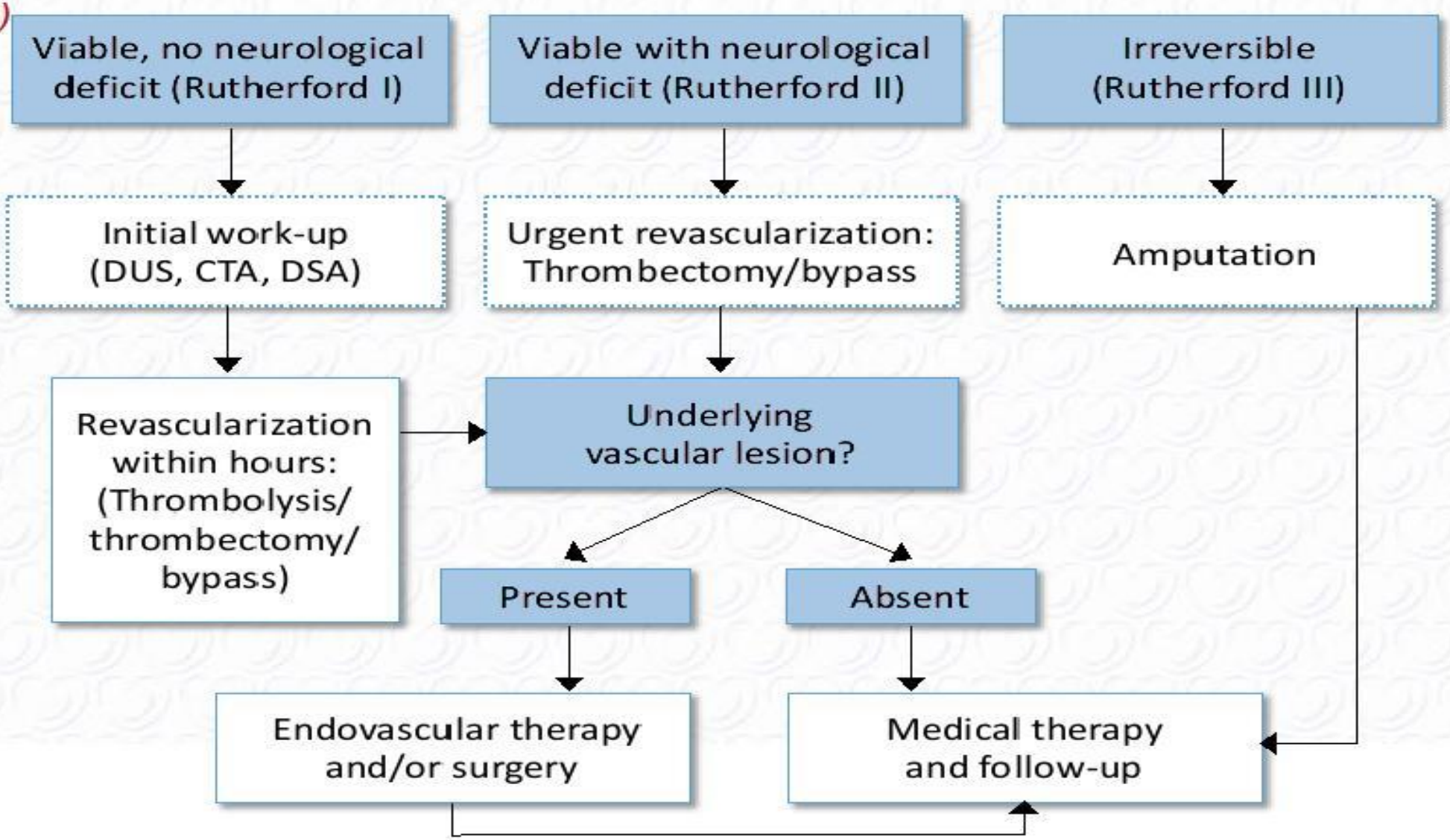

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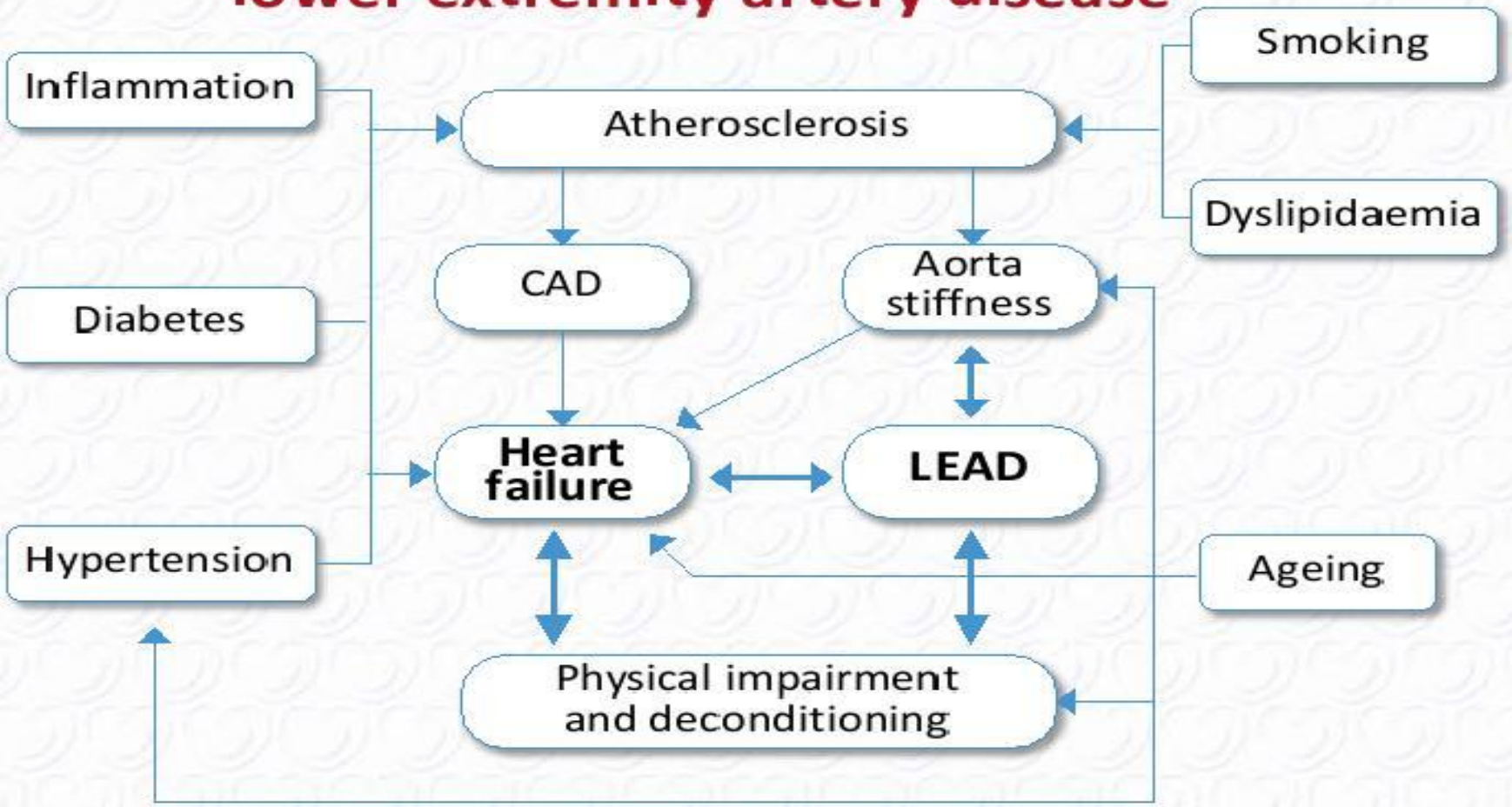
Interrelations between heart failure and lower extremity artery disease



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Thank you