

# Diseases of peripheral vessels

## Lower limb ischemia

Usually manifests by a pain during physical effort (walk, run) – intermittent claudications

Intermittent claudication distance – can be walked by the patient before stopping due to ischemic pain

In later stages steady pain – critical limb ischemia, skin defects, „marble“ skin, necrosis

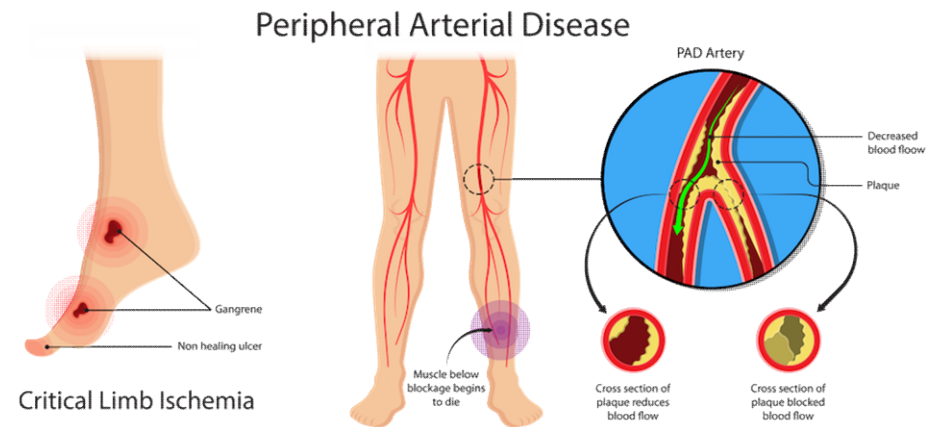


Smoking and badly compensated DM play important roles in the etiology

# Lower limb ischemia – Fontaine classification

- I) asymptomatic
- II) intermittent claudications
  - *Ila) above 200m*
  - *Ilb) below 200m*
- III) pain at rest
- IV) skin defects, gangrene

Hemodynamically significant stenosis – narrowing by approx. 50 %



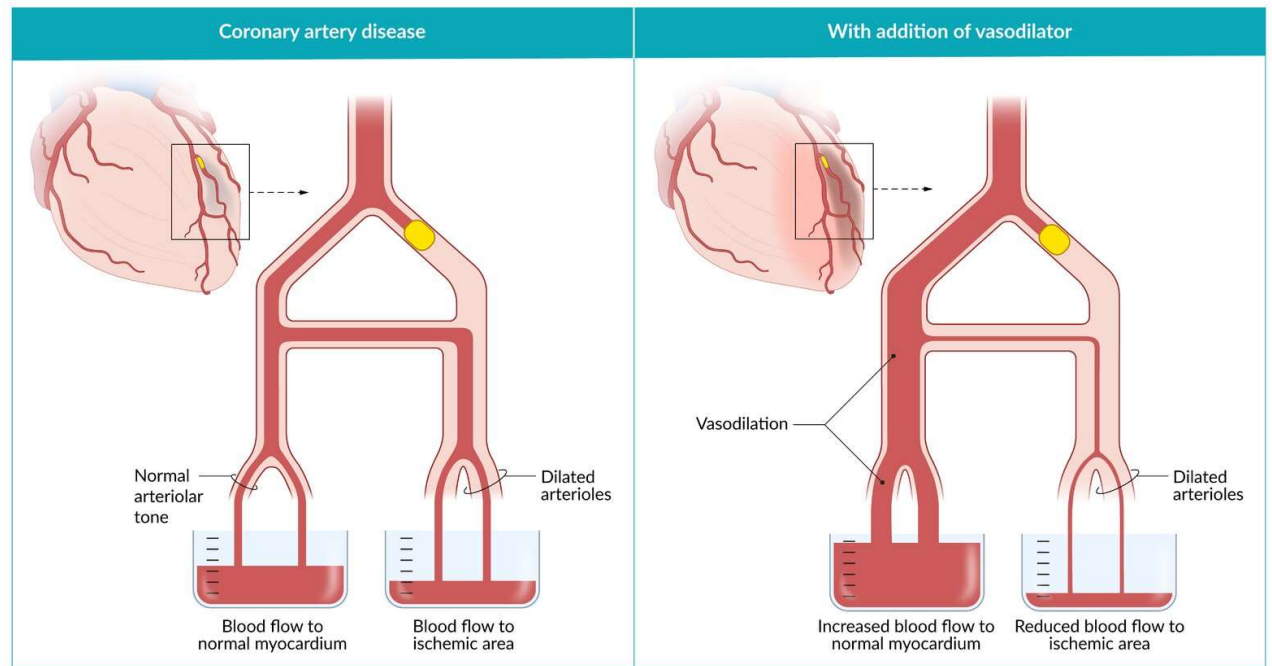
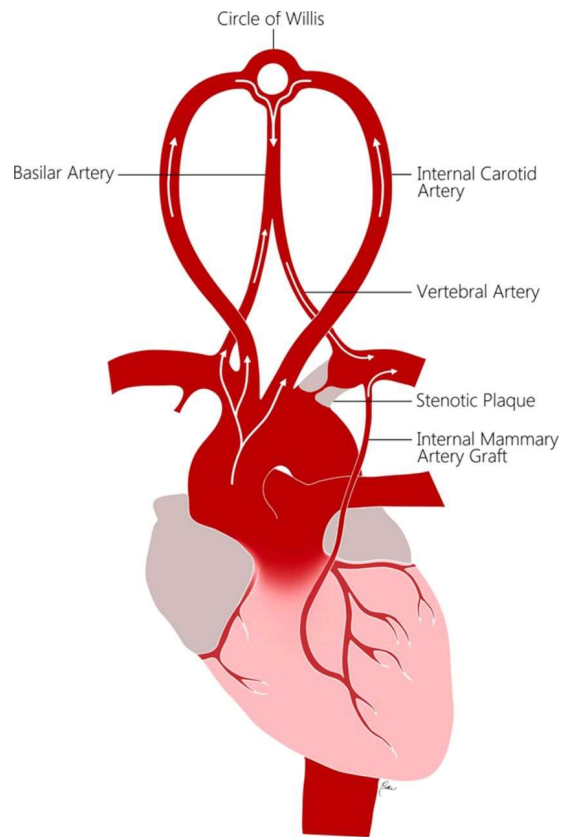
# Steal syndromes

- Occur in case when a collateral vessel bypass a stenosis (incl. artificial bypass)
- “Robin Hood” – vasodilation in ischemic area redirects the blood supply from healthy part of circulation (“the poor stealing from the rich”)
  - *subclavian steal syndrome – arm “steals” from the brain via vertebral artery → loss of consciousness*
- „Reversed Robin Hood“ – drug-induced vasodilation in healthy area redirects the blood supply from ischemic area (here, the vasodilatory mechanisms are already at maximum - “the rich stealing from the poor”)
  - *coronary steal syndrome – strong vasodilators may paradoxically worsen ischemia (e.g. combination of nitrates with sildenafil)*

# Steal syndromes - examples

Subclavian steal syndrome

Coronary steal syndrome



## Other atherosclerotic diseases

Renovascular hypertension (unilateral/bilateral stenosis – Goldblatt model)

Intestinal infarction, renal infarction, abdominal angina...

# Treatment of atherosclerosis

Treating risk factors (lifestyle intervention, antihypertensives, antidiabetics)

## Systemic

### 1) Treatment of lipid metabolism disorders

*Statins (block cholesterol synthesis)*

*Ezetimib (blocks cholesterol absorption)*

*PCSK9 inhibitors (upregulate LDL-R)*

*Fibrates (decrease VLDL production)*

*Gene therapy in monogenic dyslipidemia*

### 2) Treatment of inflammatory response

*IL-1 blockers*

# Treatment 2

## Local

PTA – percutaneous transluminal angioplasty

*POBA: plain old balloon angioplasty*

*BMS: bare metal stent*

*DES: drug-eluted stent*

covered by a **cytostatic** to

prevent neointimal hyperplasia  
and restenosis

*BVS: bio-vascular scaffold*

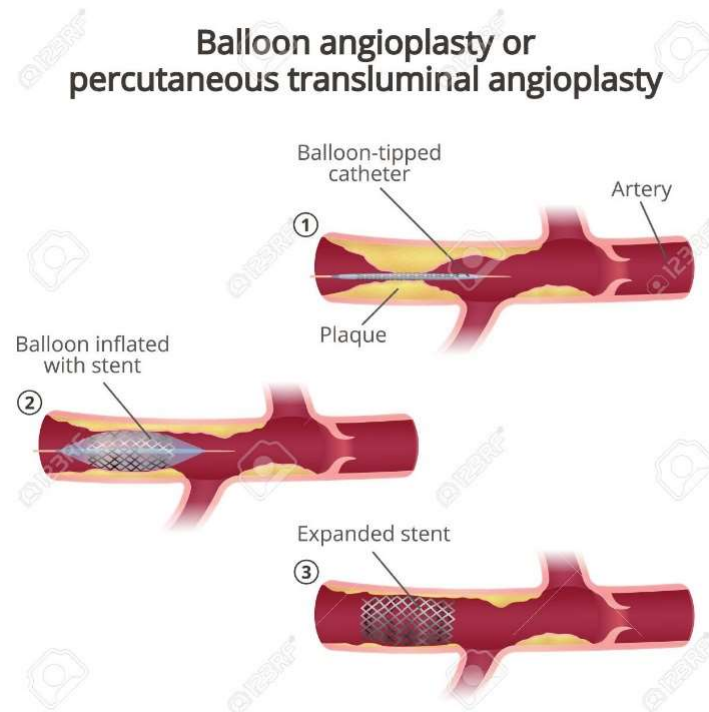
degradable, lower inflammatory  
response and risk of thrombosis

## Bypass

*Arterial*

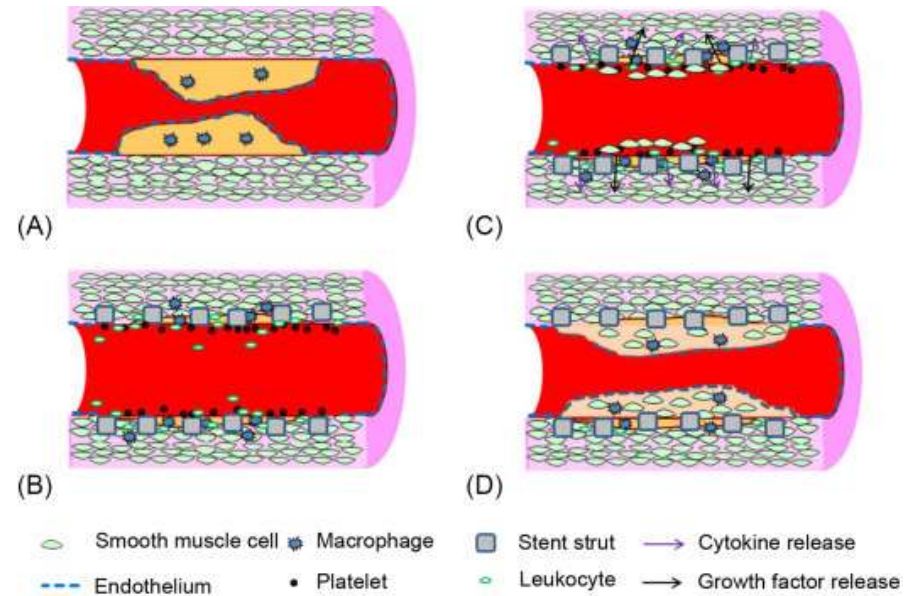
*Venous graft*

## Endarterectomy





# In-stent restenosis



Result of smooth muscle cells proliferation

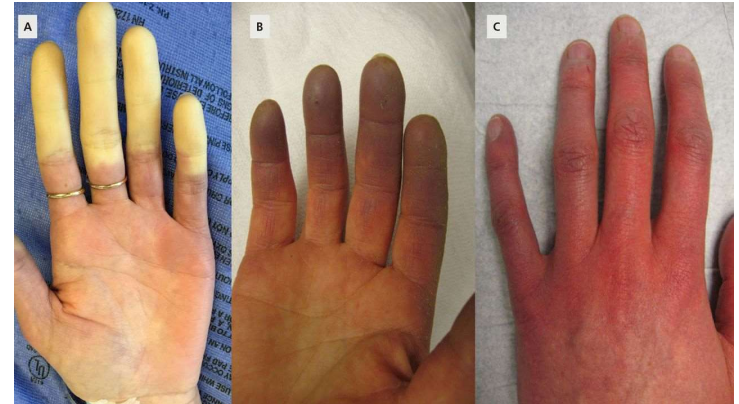
But: some degree of proliferation is necessary to cover the stent and stabilize the subendothelial space, otherwise the risk of thrombosis increases

↓ risk of restenosis in DES is accompanied by ↑ risk of thrombosis in early phase, local cytostatics are clinically efficient only in a range of years

# Vasospastic disorders

## Disorders of small arterioles

- spasms  $\leftrightarrow$  vasodilation
- $\uparrow$  sympathetic activity
- Raynaud phenomenon



- White: vasoconstriction, lack of blood, cold skin
- Blue:  $\uparrow$  deoxyHb in capillary vasodilation and hypoxia
- Red: blood flow restored, pain
- Can be provoked by stress or cold

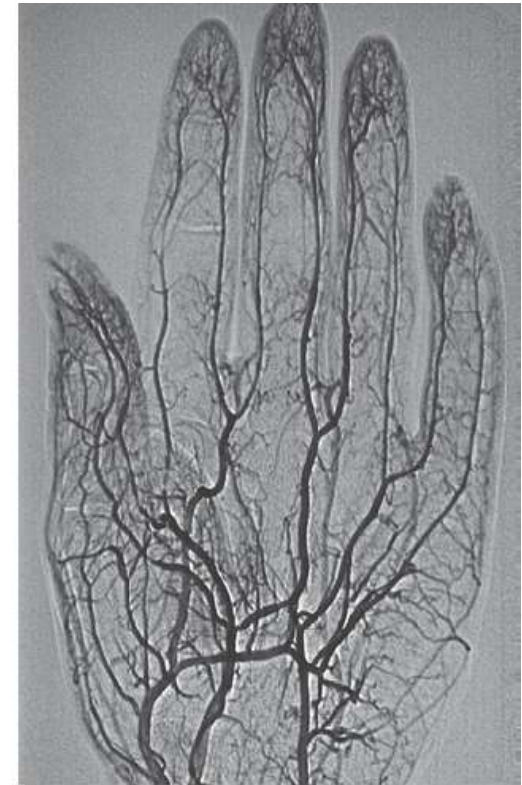
# Secondary vasospastic disorders

Result from other diseases

- Atherosclerosis
- Connective tissue diseases
- Vasculitis
- Frostbites
- Vibrations
- Treatment: reduction of cold and stress, vasodilators



a



b

# Vasculitis

- Inflammatory disorders based on immune pathology
  - Often immune complexes – IIIrd type in Gell and Coombs classification
- Affects both microcirculation and larger vessels
- Many vascular segments (× atherosclerosis)
- Primary × secondary (rheumatoid arthritis, SLE, Sjögren syndrome)
- Complications:
  - Vasospasms
  - Development of aneurysms
  - Microthrombi



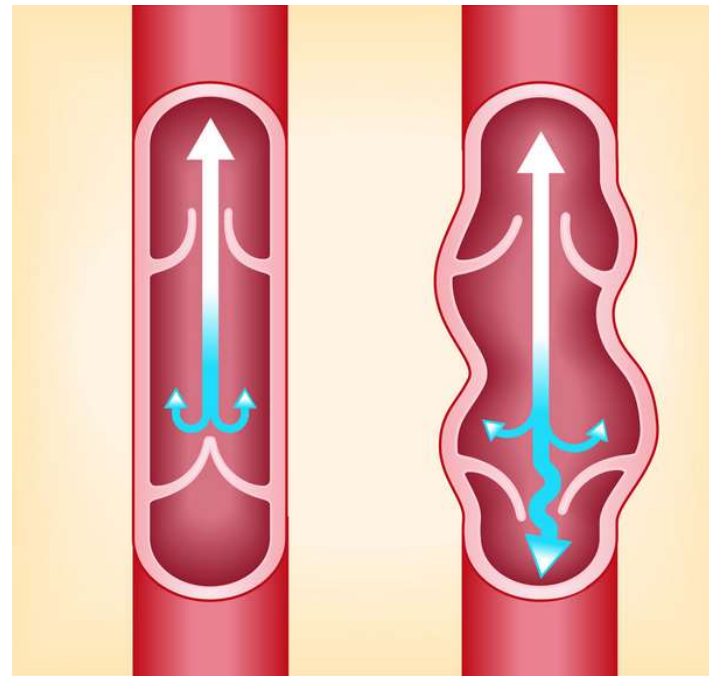
# Chronic venous insufficiency

↑hydrostatic pressure at the venous end of a capillary

Most often caused by venous valves insufficiency

Deep venous thrombosis – asymmetric oedema

Leg ulcers – most often of venous origin  
Increased filtration → increased capillary permeability → protein leak → „fibrin cuff“ → tissue ischemia → ulcer



# CVI classification

Widmer:

1st stage: oedema

2nd stage: stiff oedema with hyperpigmentation (hemosiderin – degradation product of ferritin)

3. stage: leg ulcer

CEAP (clinical-etiology-anatomy-pathophysiology) classification - detailed

