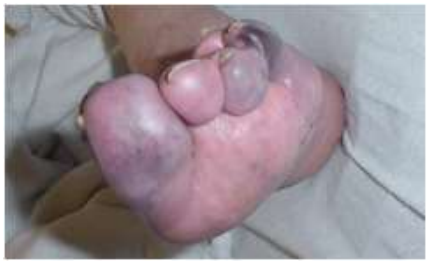
Diseases of peripheral vessels

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

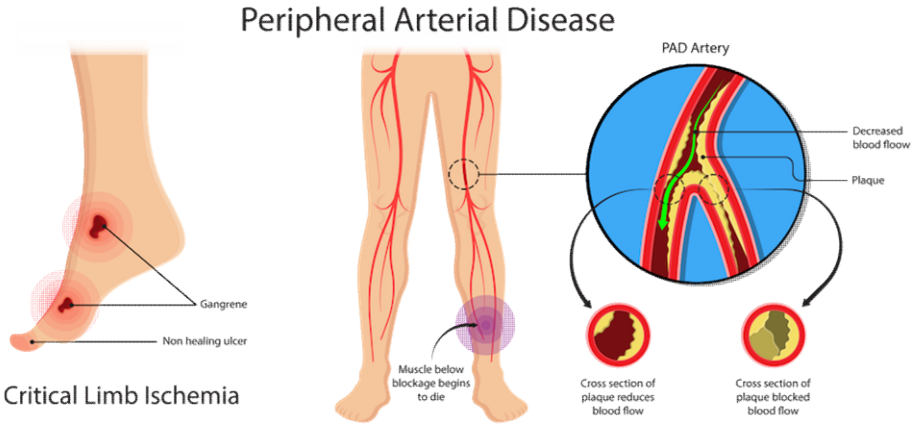


Smoking and badly compensated DM play important roles in the etiology

Lower limb ischemia – Fontaine classification

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

Hemodynamically significant stenosis – narrowing by approx. 50 %

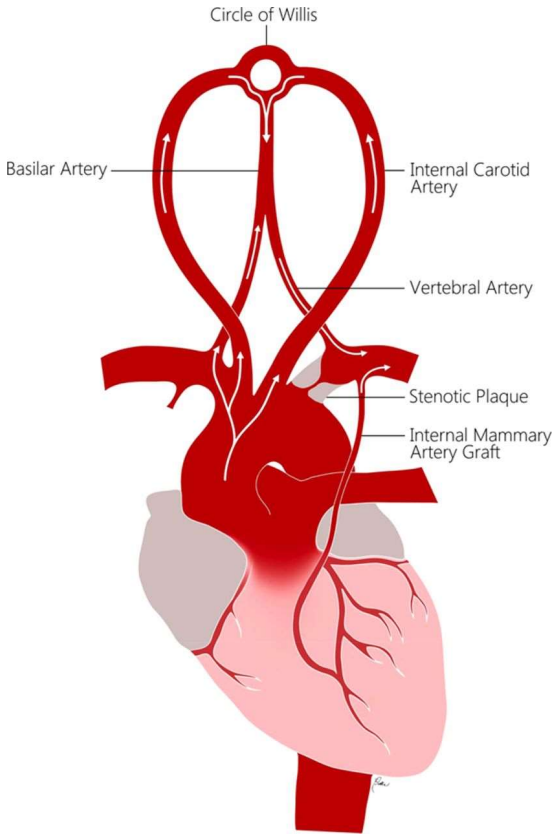


Steal syndromes

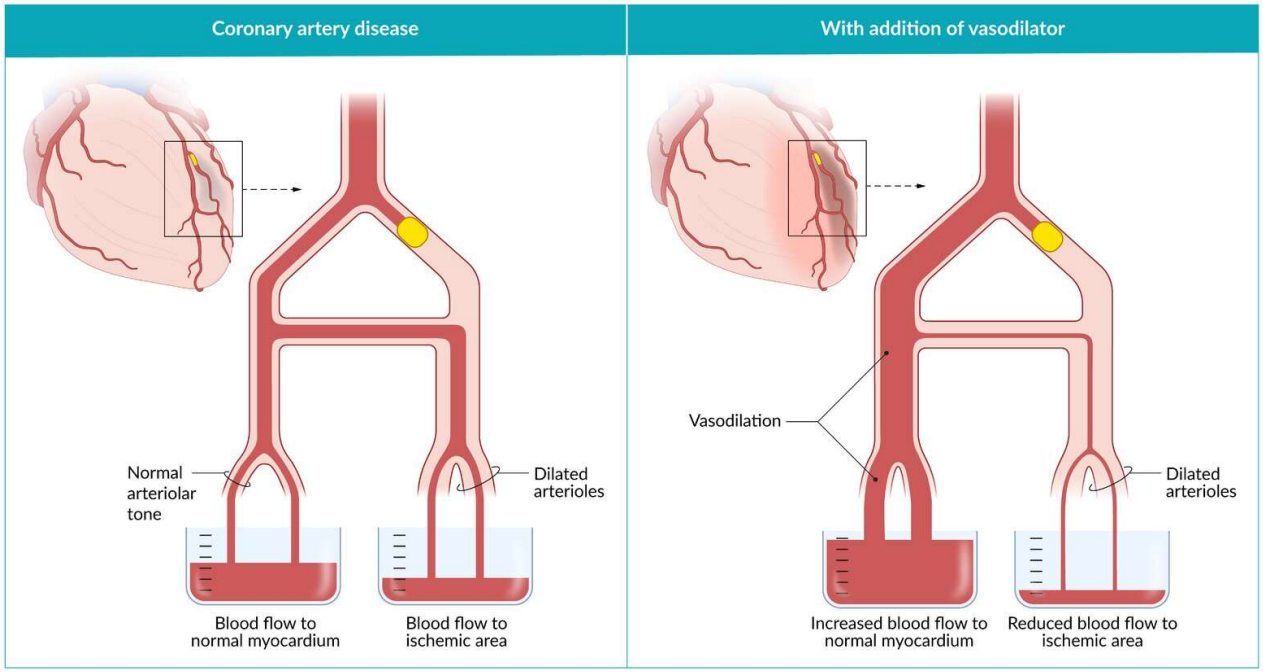
- Occur in case when a collateral vessel bypass a stenosis (incl. artificial bypass)
- "Robin Hood" – vasodilation in ischemic area redirects the blood supply from healthy part of circulation ("the poor stealing from the rich")
  - *subclavian steal syndrome – arm "steals" from the brain via vertebral 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 risc factors (lifestyle intervention, antihypertenzives, antidiabetics)

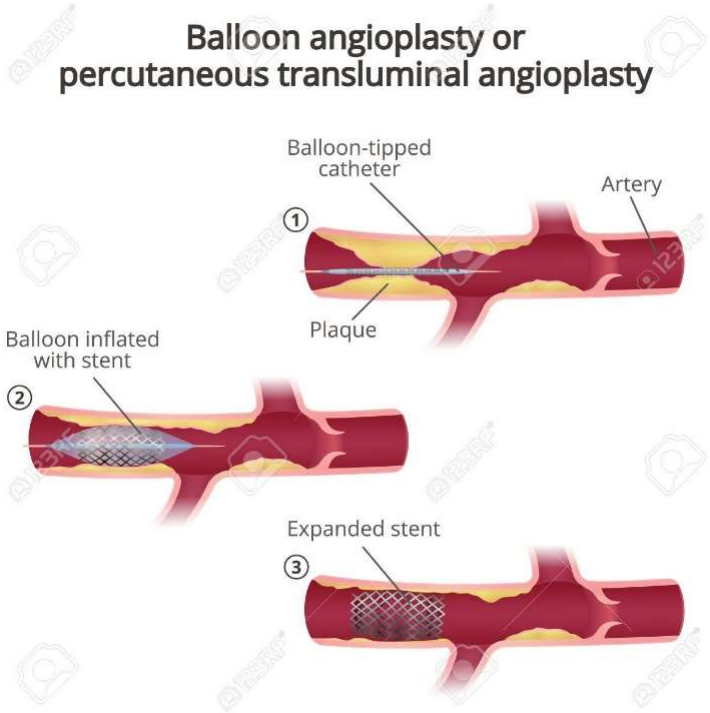
Systemic

1) Treatment of lipid metabolism disorders
   - *Statins (block cholesterol synhesis)*
   - *Ezetimib (blocks cholesterol absorbtion)*
   - *PCSK9 inhibitors (upregulate LDL-R)*
   - *Fibrates (decrease VLDL production)*
   - *Gene therapy in monogennic dyslipidemia*
2) Treatment of inflammatory response
   - *IL-1 blockers*

Treatment 2

Local

- PTA – percutaneous transluminal angioplasty
  - *POBA: plain old baloon 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 inflamatory
      - response and risk of thrombosis
- Bypass
  - *Arterial*
  - *Venous graft*
- Endarterectomy



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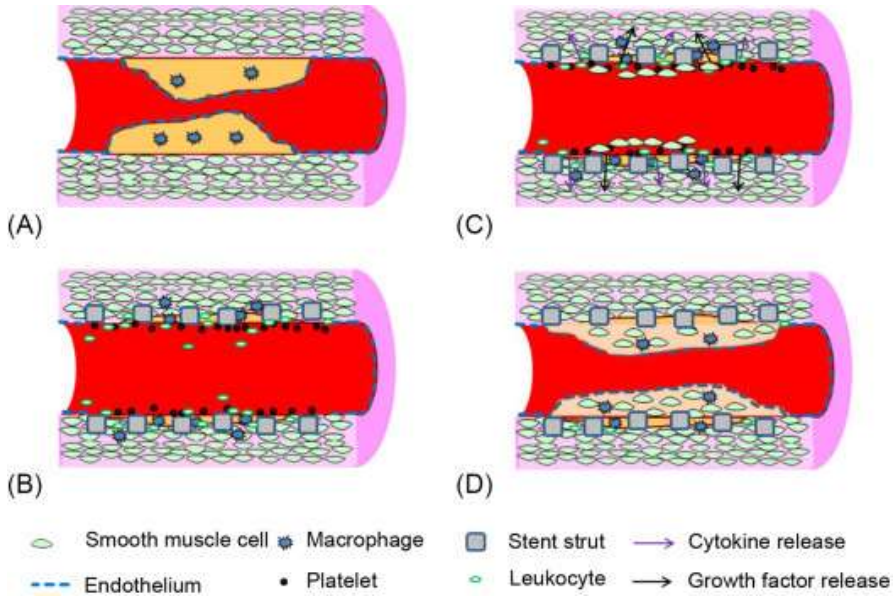
In-stent restenosis



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

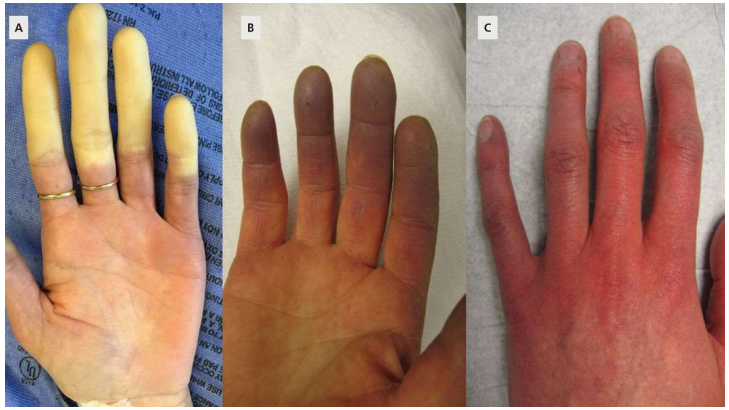
Vasospastic disorders

Disorders of small arterioles

•spasms ↔ vasodilation

•↑ sympathetic activity

•Raynaud phenomenon

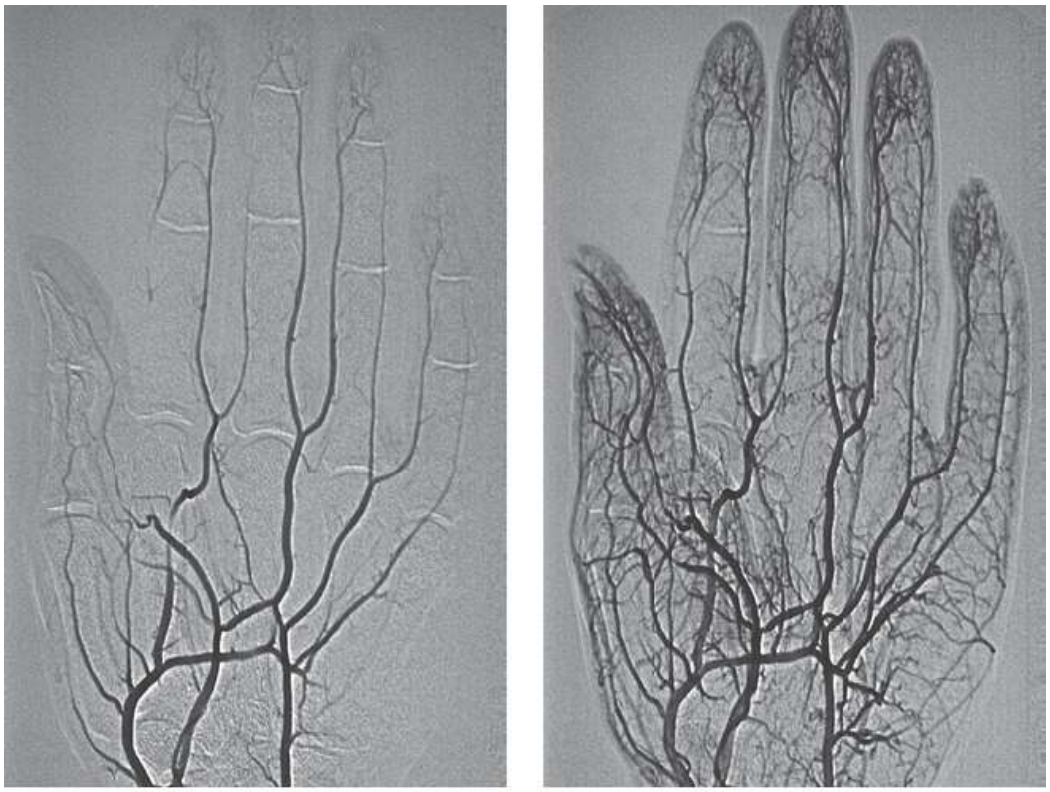


- White: vasoconstriction, lack of blood, cold skin
- Blue: ↑ deoxyHb in capillary vasodilation and hypoxia
- Red: blood flow restored, pain
- Can be provoked by stress or cold

Secondary vasospastic disorders

Result from other diseases

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



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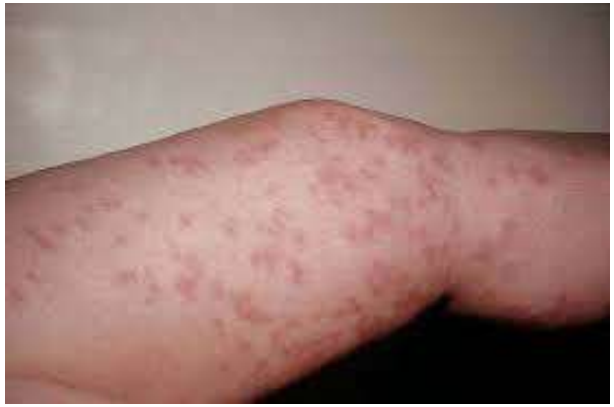
Vasculitis

- 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



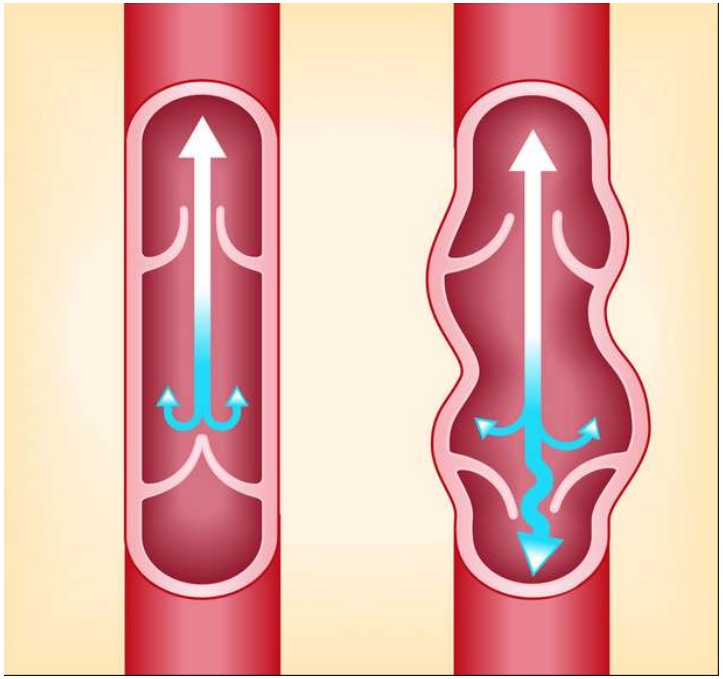
Chronic venous insufficiency

↑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



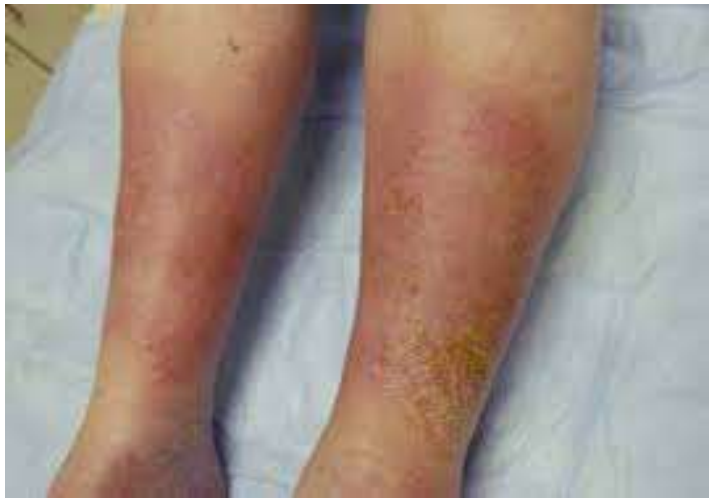
CVI classification

Widmer:

1st stage: oedema

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

3. stage: leg ulcer



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