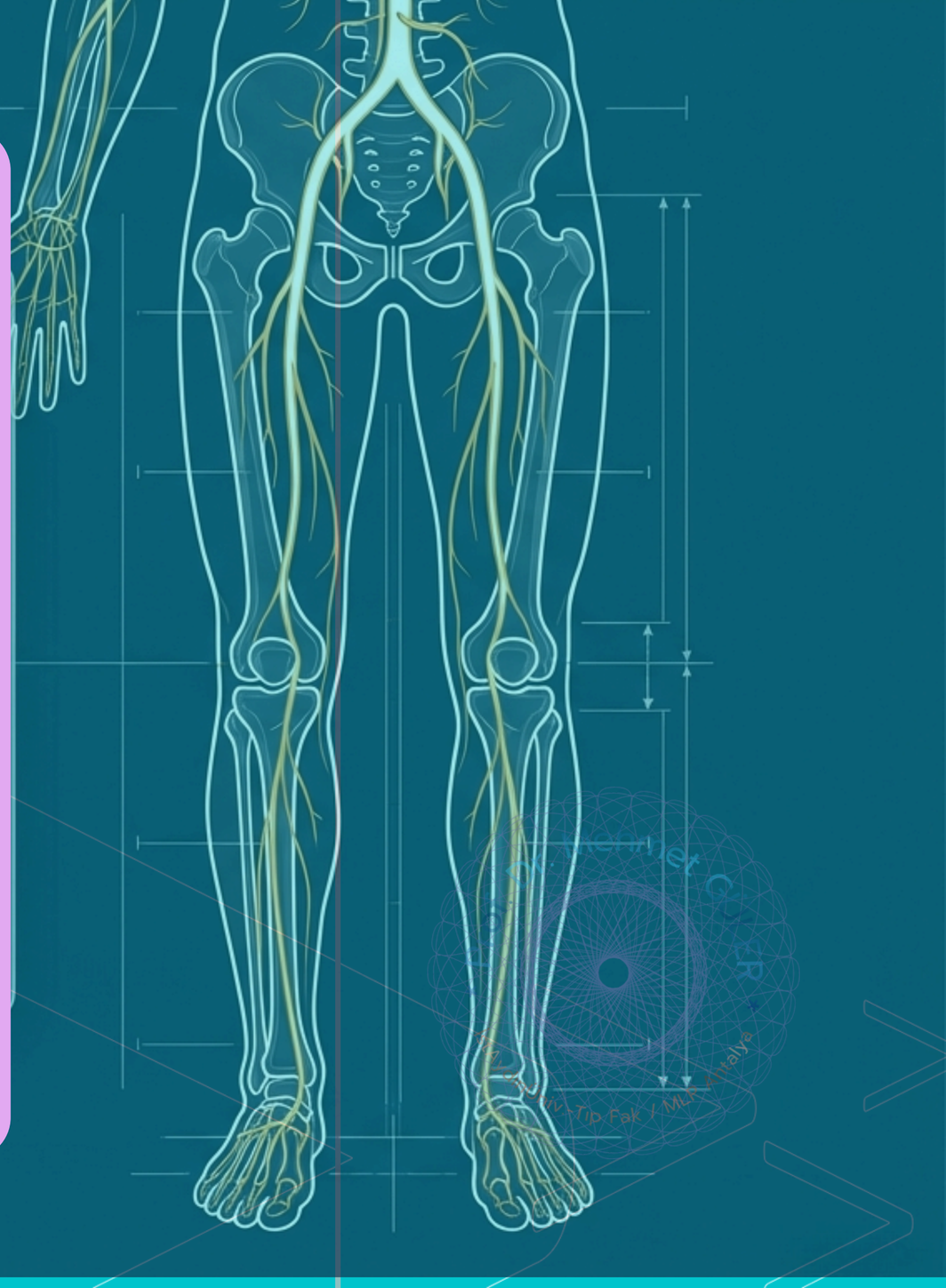
PERİFERİK VENÖZ HASTALIKLAR

Prof. Dr. Mehmet GÜLER
İAÜ Tıp Fakültesi



Ven Hastalıkları Hakkında ?
Ne Öğrenmeli, Ne Bilmeli?

Varis - Venöz Hipertansiyon

Kronik Venöz Yetmezlik

Bacak Ülseri arterden mi venden mi sinirden mi

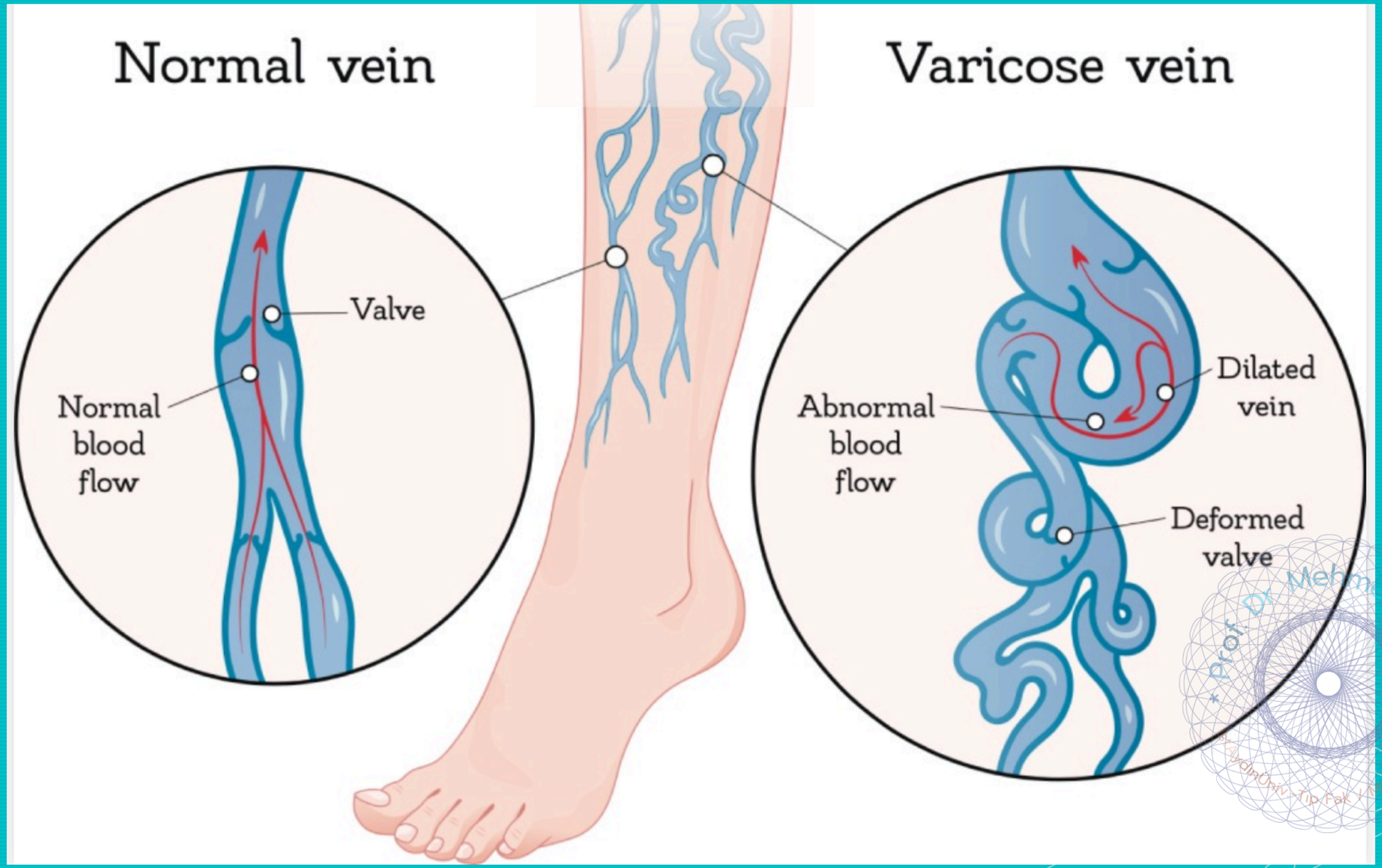
Derin Ven Trombozu Risk Faktörleri

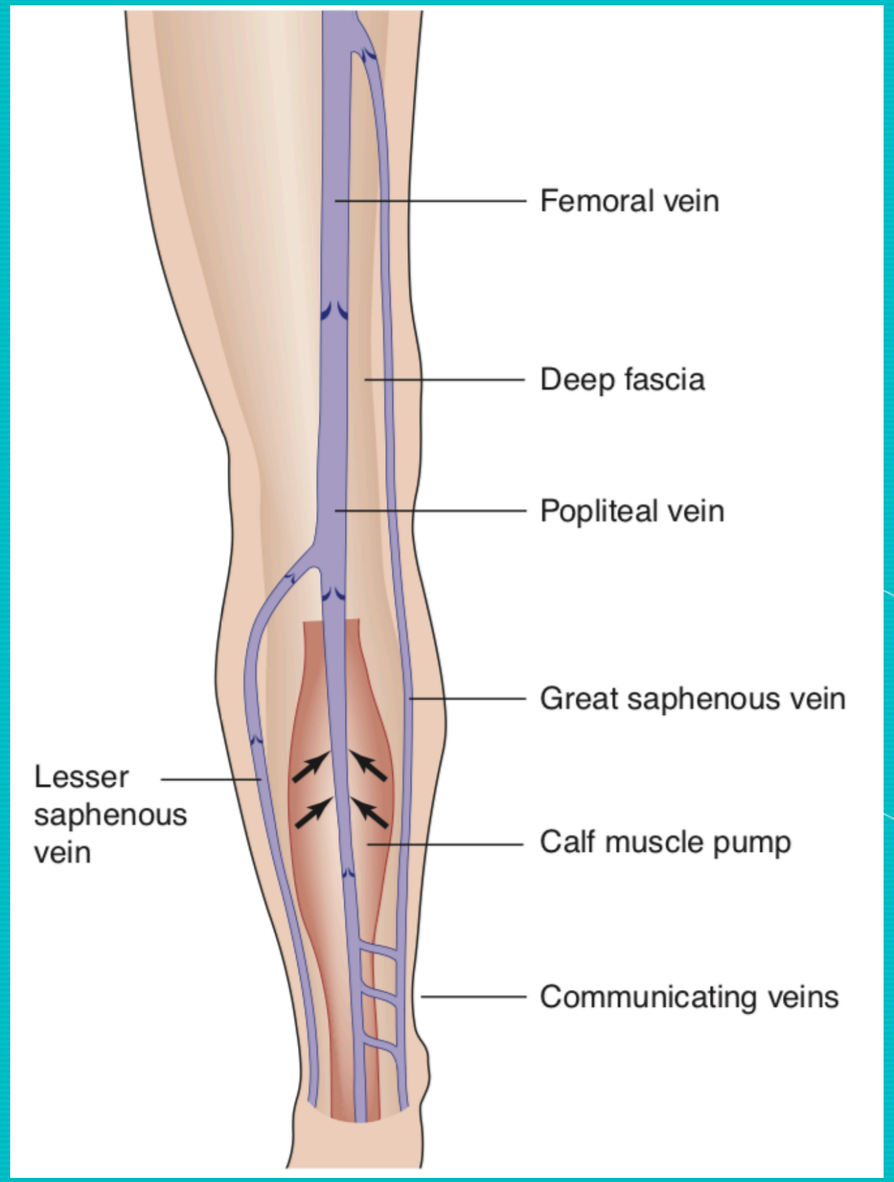
DVT Tanı Kriterleri ve Tedavisi

Antikoagülan tedavi endikasyonları - kontrendikasyonları

Pulmoner Emboli Tanısl

Garden&Parks 7. Ed. - Deakin 4. Ed. - Schwartz’s 12 Ed. - Sabiston 21 Ed. - Bailey&Love 27 Ed.


Normal vein
Valve
Normal blood flow
Varicose vein
Dilated vein
Abnormal blood flow
Deformed valve



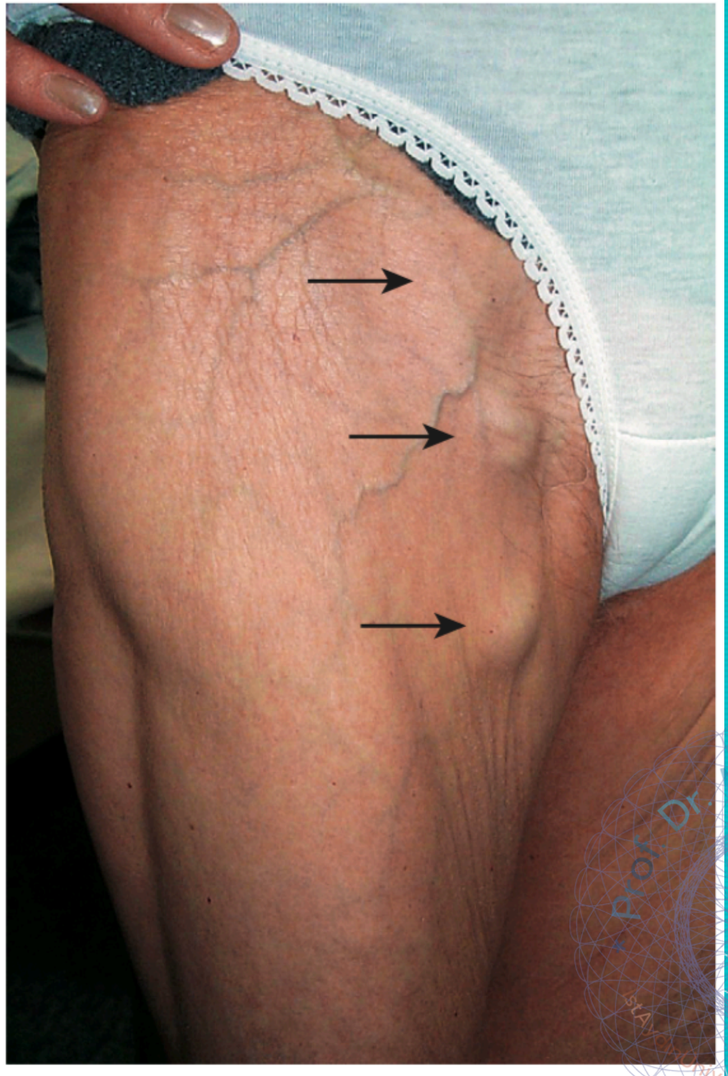


Fig. 21.29 Great saphenous varicose veins and saphena varix (arrow).

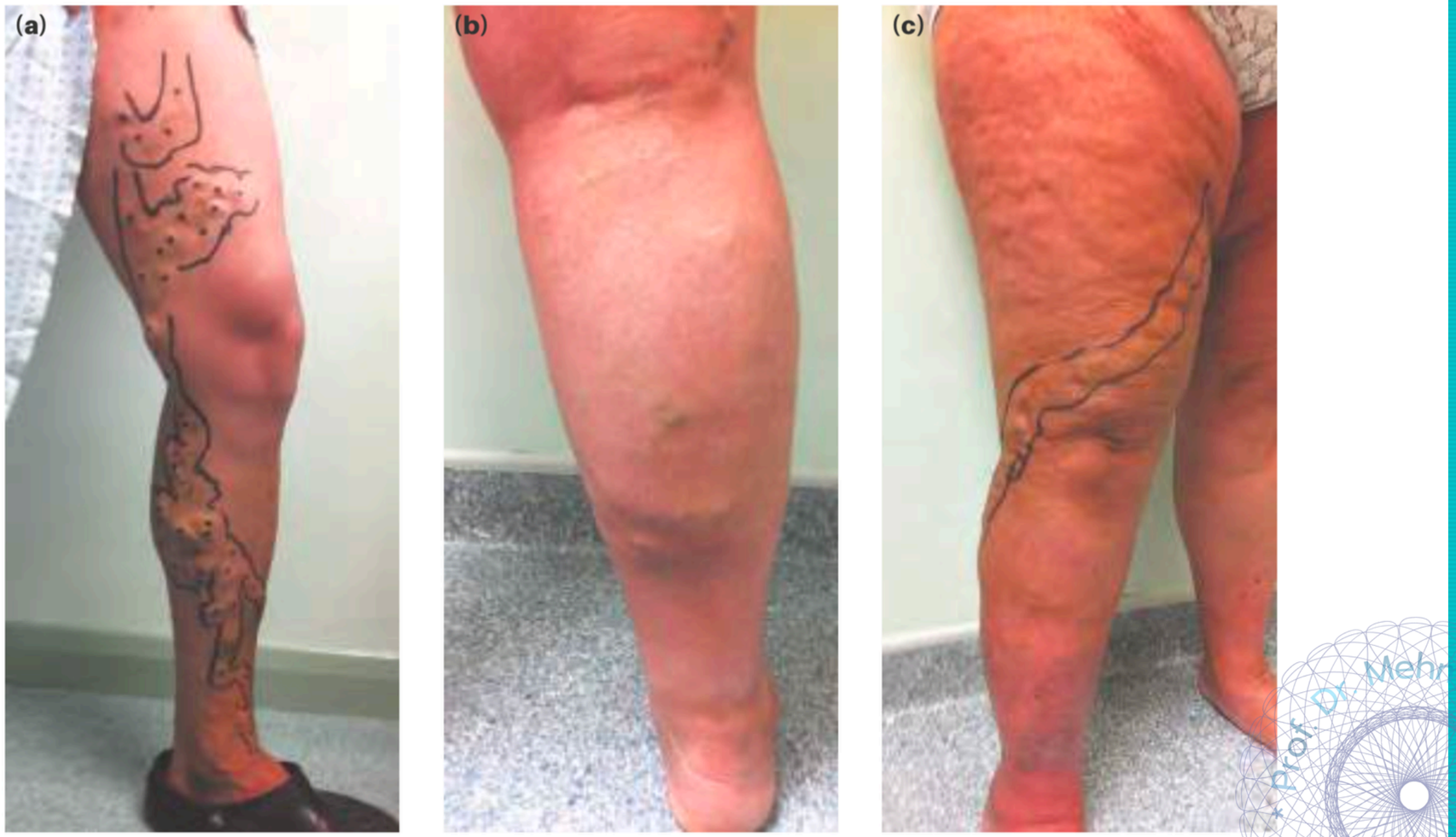
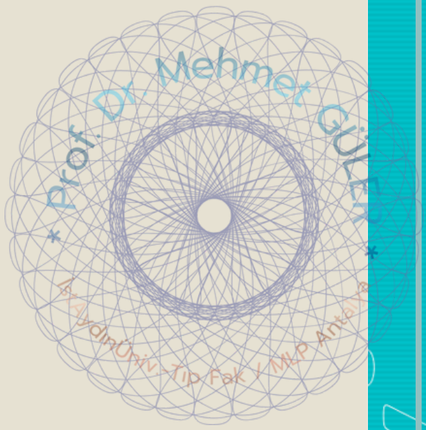


Figure 57.3 Varicose veins: **(a)** left leg varicose veins in the distribution of an incompetent great saphenous vein (marked for intervention); **(b)** right leg varicose veins in the distribution of the small saphenous system with a recent episode of phlebitis; **(c)** varicose veins in distribution of an isolated incompetent anterior accessory saphenous vein with associated gaiter area skin changes.

TABLE 57.1 Factors causing venous hypertension.

- Pressure gradient dysfunction:
  - Increased abdominal or thoracic pressure:
    - COPD
    - Pregnancy
    - Obesity
    - Large tumour
    - Constipation
  - Decreased calf muscle pump function:
    - Immobility
    - Ankle joint fusion
    - Paralysis
  - Venous structural deficit:
    - Valvular agenesis
    - Valvular incompetence
    - Venous dilatation
    - Venous tortuosity
    - Loss of vein wall compliance
    - Loss of venous tone
    - Arteriovenous fistula
  - Venous occlusion:
    - Agenesis
    - Thrombosis
    - Iatrogenic/trauma
  - Venous compression:
    - May–Thurner syndrome
    - Pelvic/abdominal tumour
    - Pelvic/abdominal radiotherapy



Clinical + Etiology + Anatomy + Pathophysiology or "C.E.A.P." is a comprehensive classification system for diagnosing venous disorders. "C" is for clinical severity rating and is most significant in physician-to-physician communication.

STAGE 1

Spider Veins

C1 refers to patients with telangiectasies (spider) and/or reticular veins.

D.O. Takeaway

Treatment of spider veins can help to slow or halt the progression of venous disease early and at its source.

STAGE 2

Varicose Veins

C2 describes patient with vein diameter that is larger than 3-4mm; the veins are pressurized enough to rise up off the skin.

D.O. Takeaway

If the varicose veins are tense and bulging when the patient stands, then become less so when the leg is elevated, then it is likely a superficial vein issue and easily treatable.

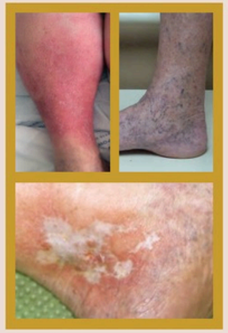
STAGE 3

Edema without Skin Changes

C3 is swelling related to venous disease, often below the knee.

D.O. Takeaway

Best visualized from the back than the front, diagnosis of C3 can be tricky. Only a valve study ultrasound can confirm edema is caused by venous disease, and sometimes the only symptom is swelling but no visible veins.



STAGE 4

Skin Discoloration

C4a, b refers to a variety of skin changes

a: corona phlebectasia (blue pigmentation and eczema)

b: lipodermatosclerosis (hardening of soft tissues) with hemosiderin staining, or atrophie blanche (whitish skin area that looks like scar tissue and can be mistaken for a healed ulcer)

D.O. Takeaway

C4 skin damage almost always occurs on the the lower leg at the "gaiter area." Although superficial venous reflux is usually the cause, this is also found in patients with more severe venous disease, such as post thrombotic syndrome (PTS).

STAGE 5

Skin Changes with Healed Ulceration

C5 is a healed skin ulcer. (One of the shortcomings of CEAP scale is once a C6, a patient can never do better than a C5.)

D.O. Takeaway

Although venous leg ulcers are the most common form of leg ulcer, there are other causes; if the area of healed ulcer is not surrounded by skin changes typical of C4, it may not be venous related. A healed venous leg ulcer is highly likely to recur if the underlying vein problem has not been treated.

STAGE 6

Skin Changes with Active Ulceration

C6 is an open active ulcer and the most severe category. (Despite advances in phlebology and prevention, C6 is still quite common.)

D.O. Takeaway

Venous leg ulcers can look markedly different in different people. The ulcer is an open sore with no skin covering the underlying tissue; it can appear pink with granulation tissue, or it can have yellow exudate. The surrounding skin is usually red as the body is using inflammation to try to heal the ulcer. Often, there is also brown skin around the ulcer.

C1 C2 C3 C4 C5 C6

CEAP Classification Of Venous Disorders

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Clinal Manifestation		Etiology		Anatomy		Pathophysiology	
C1	Telangiectasias Reticular vein	Ec	Congenital	As	Superficial Venous System	Po	obstruction
C2	Varicose vein Recurrent varicose vein **(C2r)**	Ep	Primary	AD	Deep venous System	Pr	Reflexes
C3	Edema	Es	Secondary	Ap	Perforators		
C4	Pigmentation **(C4a)** Lipodermatosclerosis **(C4b)** Corona Phlebectatica **(C4c)**						
C5	Healed Ulcer						
C6	Active venous Ulcer Recurrent Active venous Ulcer **(C6r)**						

Clinical Classification (C)	
C_0	No visible/palpable signs of venous disease
C_1	Telangiectasias or reticular veins
C_2	Varicose veins
C_3	Edema
C_{4a}	Pigmentation and/or eczema
C_{4b}	Lipodermatosclerosis and/or atrophy
C_5	Healed venous ulcer
C_6	Open venous ulcer
	Subscript
A	Asymptomatic
S	Symptomatic

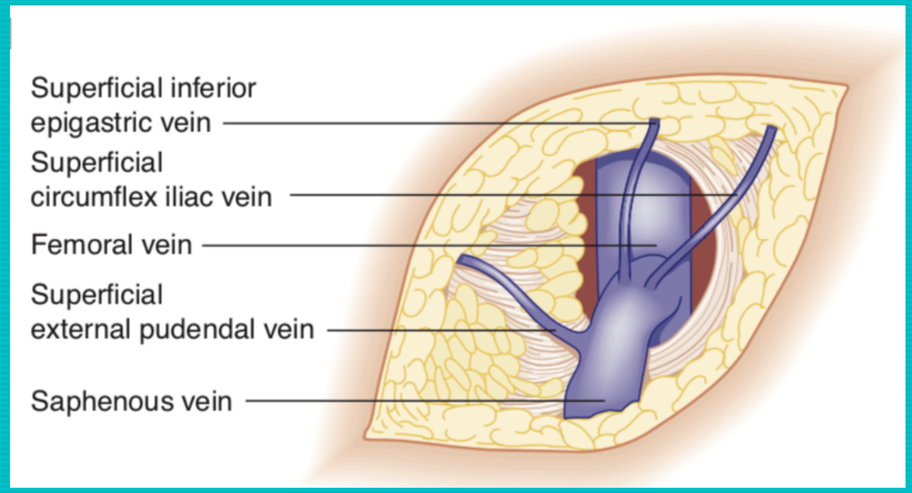
Etiologic Classification (E)	
E_c	Congenital
E_p	Primary
E_s	Secondary (postthrombotic)
E_n	No venous etiology identified
Anatomic Classification (A)	
A_s	Superficial veins
A_p	Perforator veins
A_d	Deep veins
A_n	No venous location identified
Pathophysiologic Classification (P)	
P_r	Reflux
P_o	Obstruction
$P_{r,o}$	Reflux and obstruction
P_n	No venous pathophysiology identifiable

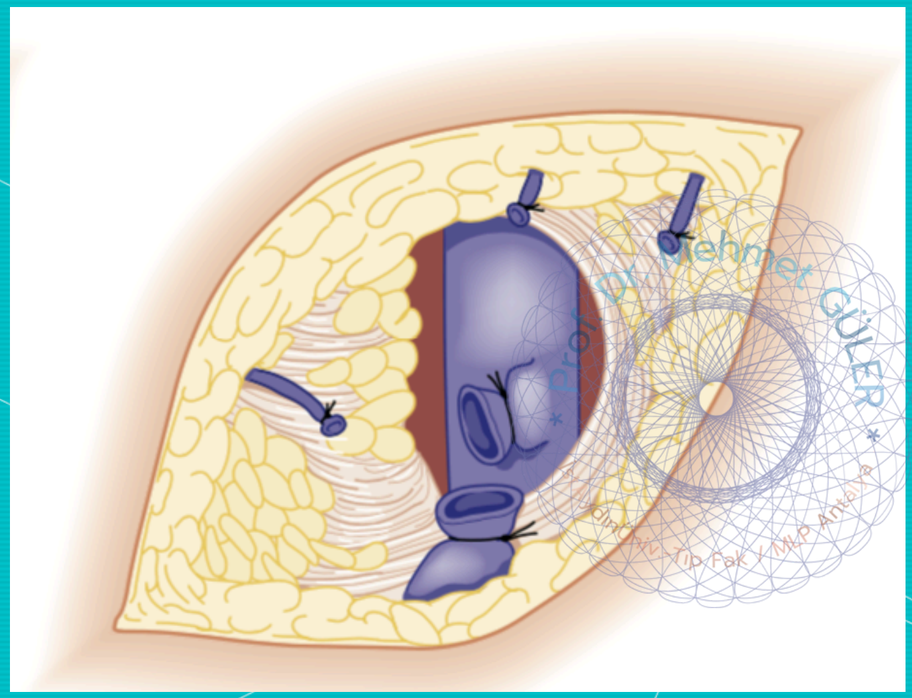
D


BD Plastipak

F

G


Superficial inferior
epigastric vein
Superficial
circumflex iliac vein
Femoral vein
Superficial
external pudendal vein
Saphenous vein


Prof. Dr. Mehmet GÜLER

"Uçaktan İndim, Bacağım Şişti"

- 35 yaşında kadın hasta,
- 10 saatlik bir kıtalararası **uçuş** sonrası
- Sol bacağında aniden başlayan **şişlik, ağrı ve kızarıklık** şikayetiyle acil servise başvuruyor.
- Bilinen bir hastalığı yok,
- Ancak 5 yıldır **oral kontraseptif**(OKS) kullanıyor.
- Fizik muayenede sol bacakta sağa göre belirgin **çap artışı**,
- Gode bırakan **ödem** ve
- Baldırda palpasyonla **hassasiyet** saptanıyor.
- Homan's belirtisi negatif.

Homan's belirtisi (ayağın dorsifleksiyonu ile baldırda ağrı) DVT için duyarlılığı ve özgüllüğü düşüktür (<%50), bu nedenle varlığı veya yokluğu tanıyı ekarte ettirmez.

Table 21.6 Differential diagnosis of the swollen limb

Non-Vascular or Lymphatic

General disease states
- Cardiac, renal and liver failure
- Hyperthyroidism (myxoedema)
- Allergic disorders
- Immobility and lower limb dependency

Local disease processes
- Ruptured Baker's cyst
- Myositis ossificans
- Bony or soft tissue tumours
- Arthritis
- Haemarthrosis
- Calf muscle haematoma
- Achilles tendon rupture
- Other trauma
- Reflex sympathetic dystrophy

Gigantism
- Rare; all tissues are uniformly enlarged

Drugs
- Steroids

Obesity
- Lipodystrophy, lipoidosis

Venous

Deep venous thrombosis
- The classic signs of pain and redness may be absent

Post-thrombotic syndrome
- Venous skin changes, secondary varicose veins on the leg and collateral veins on the lower abdominal wall
- Venous claudication may be present

Varicose veins
- Do not usually cause significant swelling

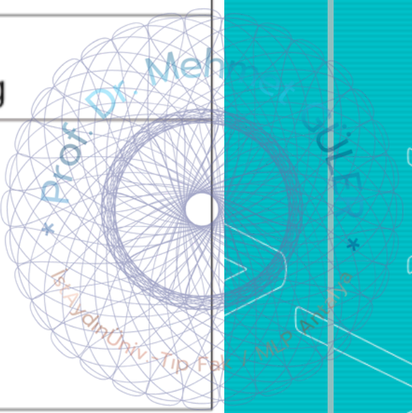
Venous malformations
- Most common is Klippel–Trenaunay syndrome
- Abnormal lateral venous complex, capillary naevus, hypo(a)plasia of deep veins and limb lengthening
- Lymphatic abnormalities often co-exist

External venous compression
- Pelvic or abdominal tumour including the gravid uterus
- Retroperitoneal fibrosis

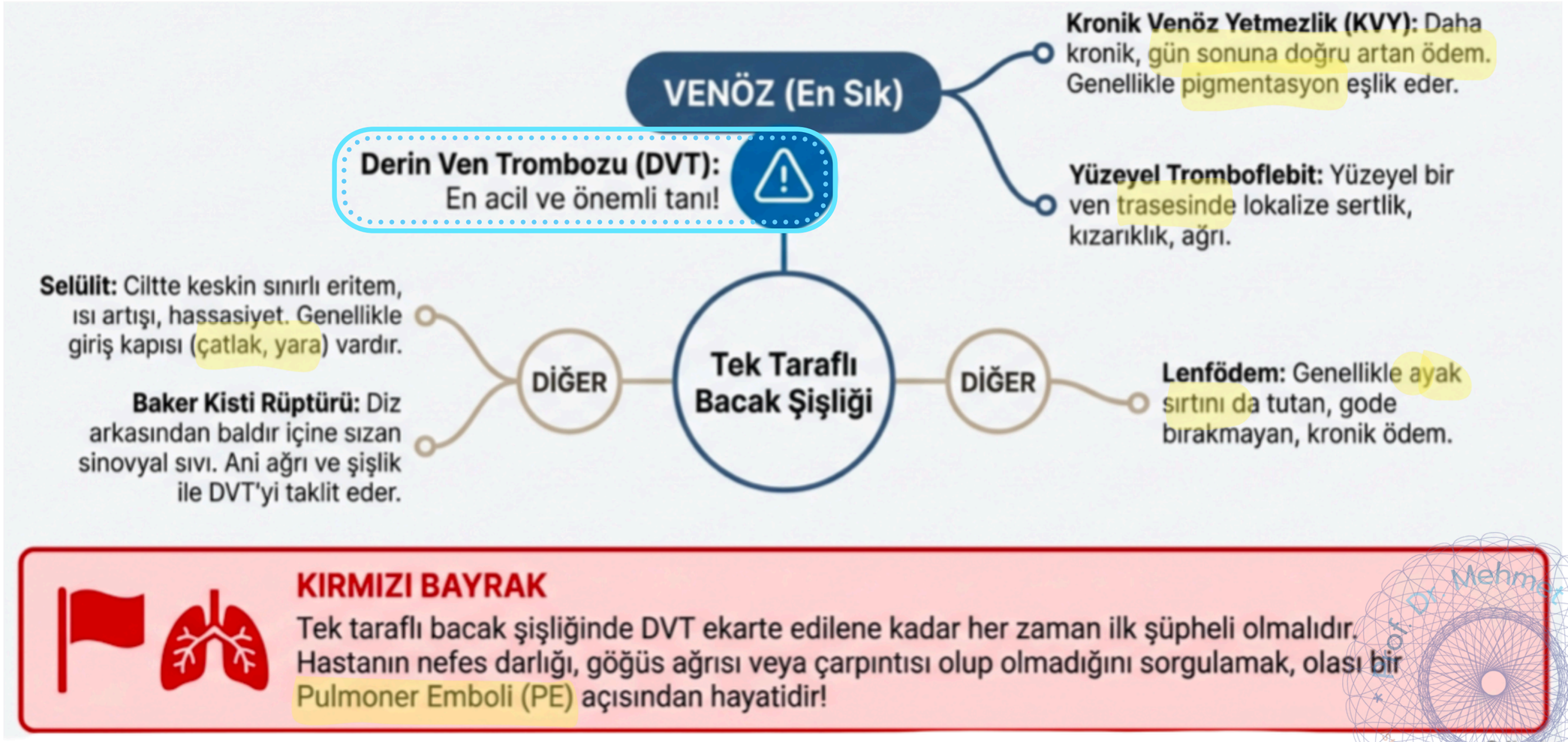
Arterial ischaemia-reperfusion
- Following lower limb revascularization for chronic and particularly acute ischaemia

Arteriovenous malformation
- May be associated with local or generalized swelling

Aneurysm
- Popliteal
- Femoral
- False aneurysm following (iatrogenic) trauma



Tek Taraflı Bacak Şişliği



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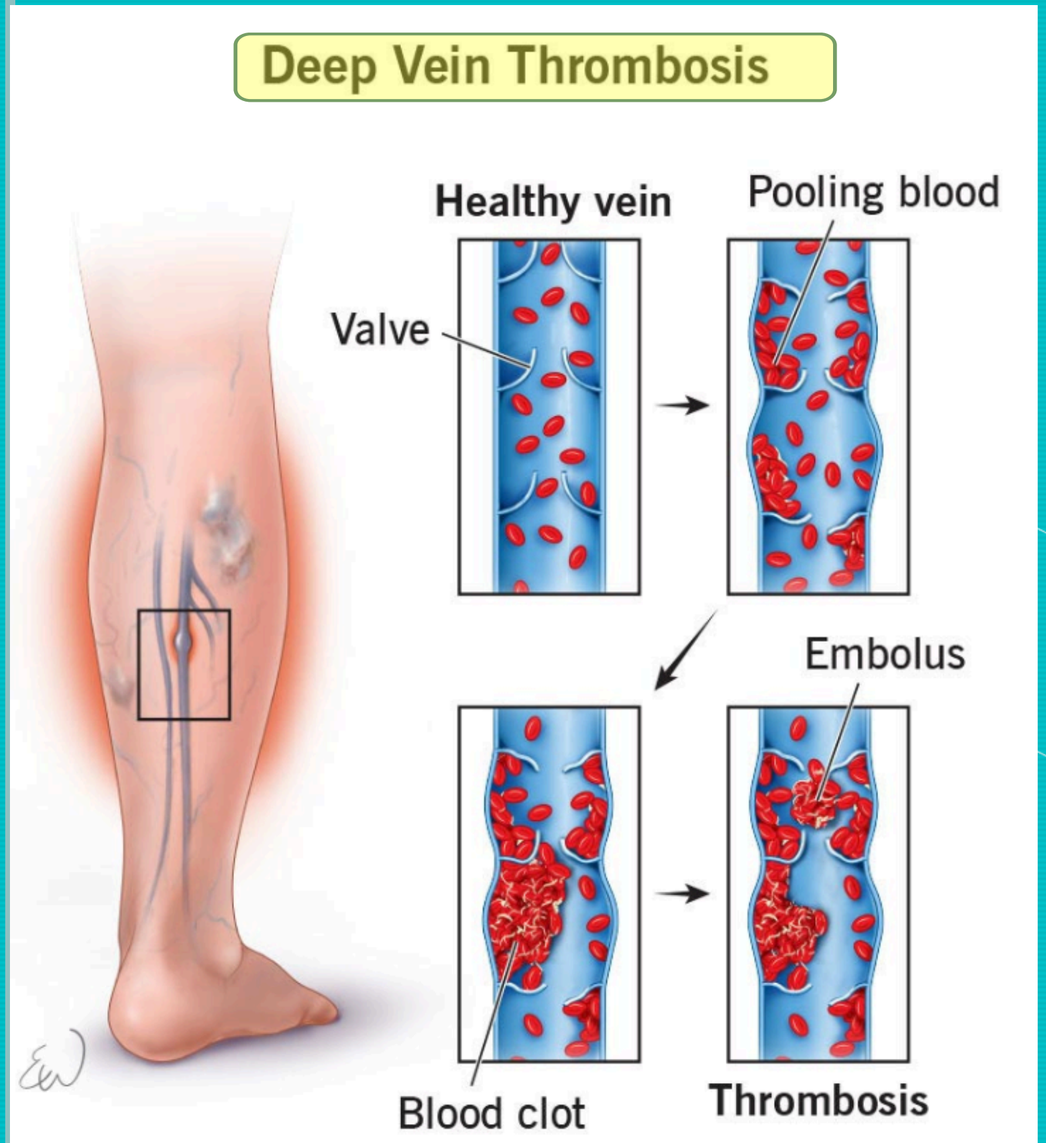
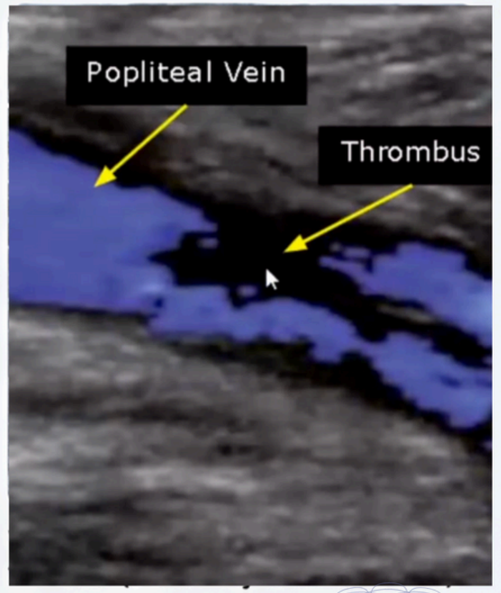


Derin Ven Trombozu (DVT) - Akut Pıhtılaşma

Neden Olur? Virchow Triadı:

1. **Staz** (İmmobilizasyon, uzun yolculuk)
2. **Endotel Hasarı** (Travma, cerrahi)
3. **Hiperkoagülabilite** (Malignite, OKS, genetik yatkınlık)



Nasıl Tanınır?

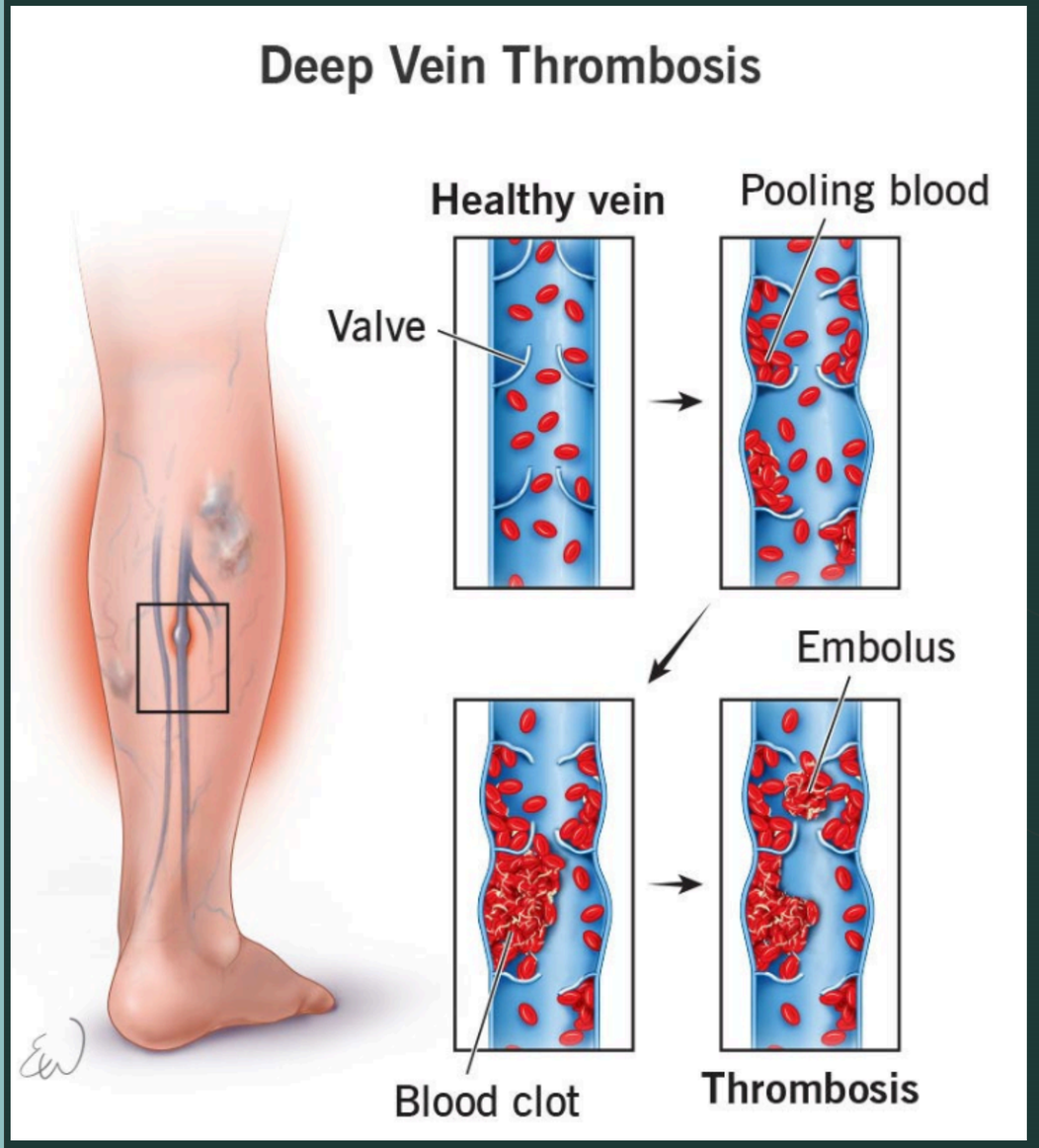
Altın Standart: Venöz Dupleks USG. Kompresyon manevrası ile venin kollabe olmaması tanı koydurucudur.

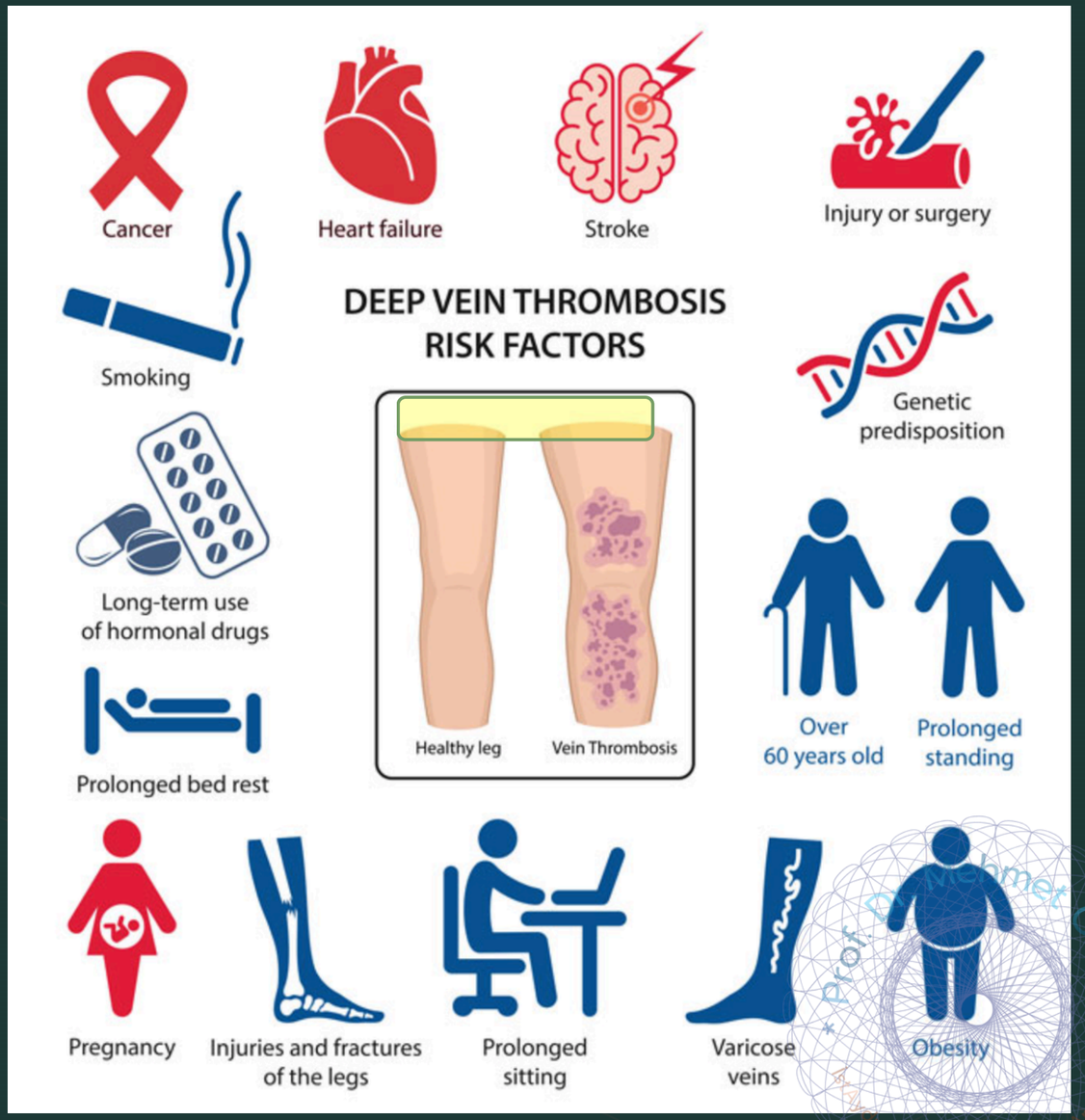
D-Dimer: Negatifliği DVT'yi büyük oranda ekarte eder, ancak pozitifliği spesifik değildir. Düşük klinik şüphede kullanılır.

Ne Yapılır?

Tedavinin Amacı: PE'yi önlemek, pıhtının büyümesini engellemek.

Tedavi: Akut dönemde heparin (genellikle Düşük Molekül Ağırlıklı Heparin - DMAH) ile başlanır, sonrasında oral antikoagülanlara (Warfarin veya Yeni Nesil Oral Antikoagülanlar - YNOAK) geçilir.


Deep Vein Thrombosis
Healthy vein
Pooling blood
Valve
Embolus
Blood clot
Thrombosis


Cancer
Heart failure
Stroke
Injury or surgery
Smoking
DEEP VEIN THROMBOSIS
RISK FACTORS
Genetic
predisposition
Long-term use
of hormonal drugs
Healthy leg
Vein Thrombosis
Over
60 years old
Prolonged
standing
Prolonged bed rest
Pregnancy
Injuries and fractures
of the legs
Prolonged
sitting
Varicose
veins
Obesity

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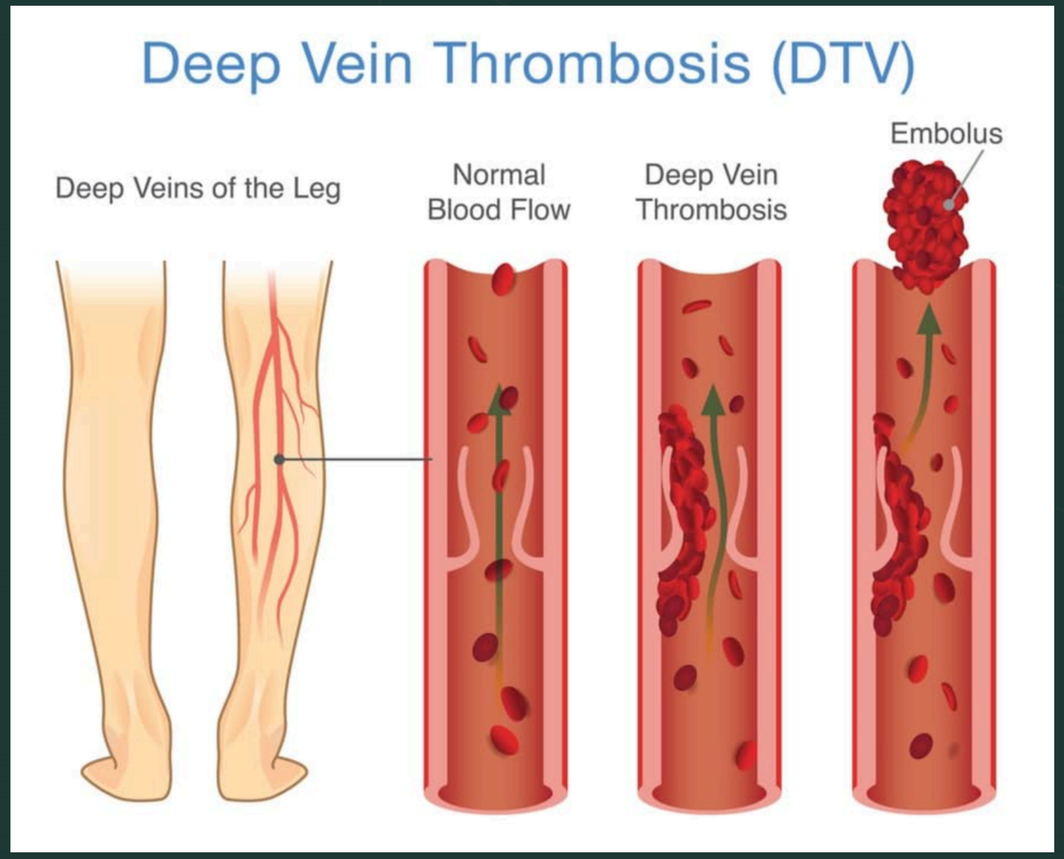
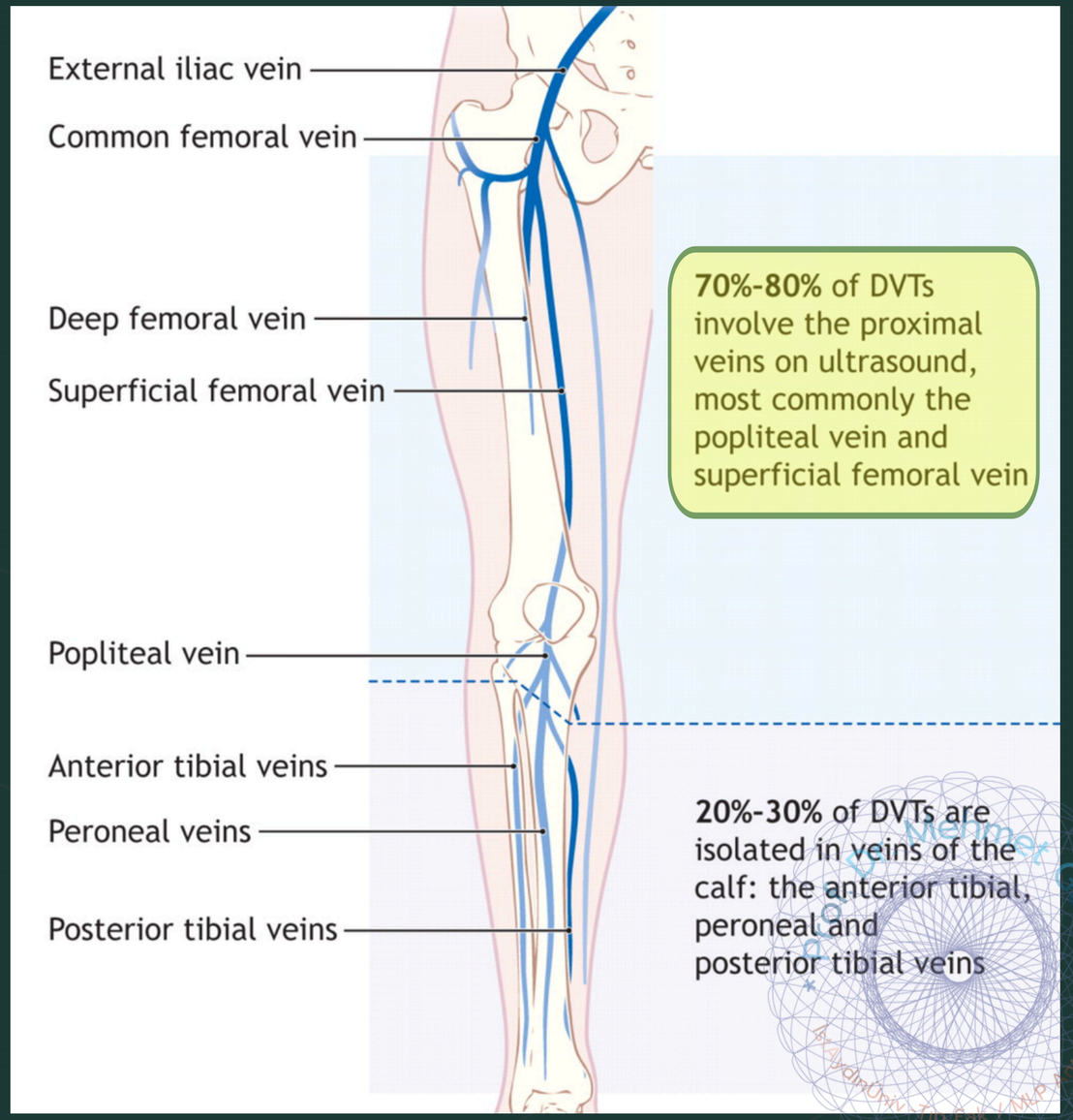

Deep Vein Thrombosis (DTV)
Deep Veins of the Leg
Normal Blood Flow
Deep Vein Thrombosis
Embolus


External iliac vein
Common femoral vein
Deep femoral vein
Superficial femoral vein
Popliteal vein
Anterior tibial veins
Peroneal veins
Posterior tibial veins
70%-80% of DVTs involve the proximal veins on ultrasound, most commonly the popliteal vein and superficial femoral vein
20%-30% of DVTs are isolated in veins of the calf: the anterior tibial, peroneal and posterior tibial veins

Risk factors for venous thromboembolism

Acquired

Advanced age

Hospitalization/immobilization

Hormone replacement therapy and oral contraceptive use

Pregnancy and puerperium

Prior venous thromboembolism

Malignancy

Major surgery

Obesity

Nephrotic syndrome

Trauma or spinal cord injury

Long-haul travel (>6 hours)

Varicose veins

Antiphospholipid antibody syndrome

Myeloproliferative disease

Polycythemia

Inherited

Factor V Leiden

Prothrombin 20210A

Antithrombin deficiency

Protein C deficiency

Protein S deficiency

Factor XI elevation

Dysfibrinogenemia

Mixed Etiology

Homocysteinemia

Factors VII, VIII, IX, XI elevation

Hyperfibrinogenemia

Activated protein C resistance without factor V Leiden

TABLE 57.2 Risk factors for venous thromboembolism.

Patient factors
Age
Obesity
Varicose veins
Immobility
Pregnancy
Puerperium
High-dose oestrogen therapy
Previous deep vein thrombosis or pulmonary embolism
Thrombophilia (see *Table 57.3*)
Disease or surgical procedure
Trauma or surgery, especially of pelvis, hip and lower limb
Malignancy, especially pelvic, and abdominal metastatic
Heart failure
Recent myocardial infarction
Paralysis of lower limb(s)
Infection
Inflammatory bowel disease
Nephrotic syndrome
Polycythaemia
Paraproteinaemia
Paroxysmal nocturnal haemoglobinuria antibody or lupus anticoagulant
Behçet's disease
Homocystinaemia

- prior thrombosis
- surgery
- advanced age
- restricted mobility
- cancer
- overweight

z

TABLE 57.3 Abnormalities of thrombosis and fibrinolysis (thrombophilia) that lead to an increased risk of venous thrombosis.

Congenital
Deficiency of antithrombin III, protein C or protein S
Antiphospholipid antibody or lupus anticoagulant
Factor V Leiden gene defect or activated protein C resistance
Dysfibrinogenaemias
Acquired
Antiphospholipid antibody or lupus anticoagulant

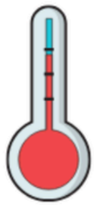
Physical Findings in Patients with Deep Vein Thrombosis

Clinical signs and symptoms are highly variable and unspecific
Diagnosing DVT by physical examination is inaccurate

The patient should be examined in the horizontal position with the knees slightly flexed. Examination should be systematic and include palpation over the posterior tibial veins and the peroneal, popliteal, superficial femoral, common femoral and iliac veins. Pain and tenderness located away from these regions suggests that these features are not due to venous thrombosis.

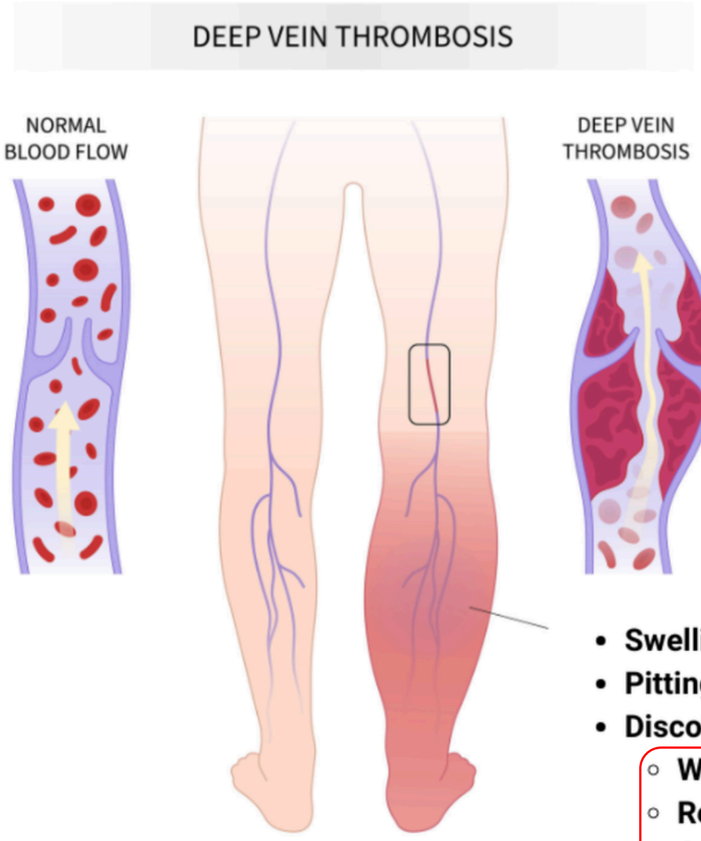
Michaeli sign

(low grade pyrexia without other causes)



Mahler sign

(increase in heart rate)



- **Swelling**
- **Pitting edema**
- **Discoloration**
  - **Whiteness**
  - **Redness**
  - **Cyanosis**
- **Leg tenderness**

Homan's sign

(triggering pain in the calf on the dorsiflexion of the foot)
(insensitive and non-specifi)

Collateral superficial veins

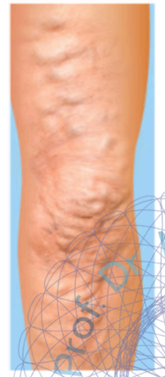


TABLE 57.6 Low-, medium- and high-risk patient groups for venous thromboembolism.

LOW
Minor surgery <30 minutes; any age; no risk factors
Major surgery >30 minutes; age <40; no other risk factors
Minor trauma or medical illness
MODERATE
Major surgery; age 40+ or other risk factors
Major medical illness: heart/lung disease, cancer, inflammatory bowel disease
Major trauma/burns
Minor surgery, trauma, medical illness in patient with previous DVT, PE or thrombophilia
HIGH
Major orthopaedic surgery or fracture of pelvis, hip, lower limb
Major abdominal/pelvic surgery for cancer
Major surgery, trauma, medical illness in patient with DVT, PE or thrombophilia
Lower limb paralysis (e.g. stroke, paraplegia)
Major lower limb amputation

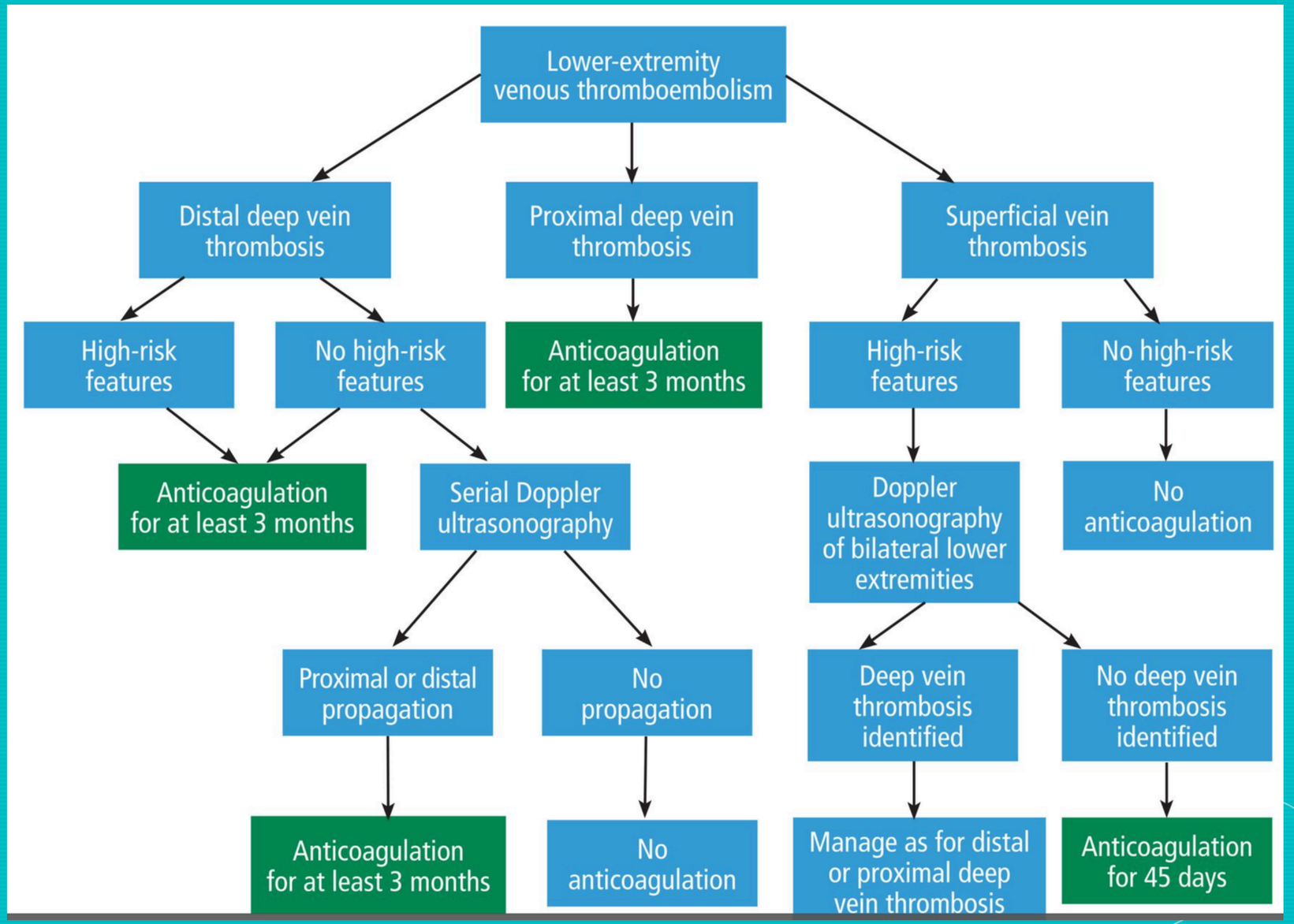
DVT, deep vein thrombosis; PE, pulmonary embolus.

DVT, deep vein thrombosis; PE, pulmonary embolus.

TABLE 57.4 Modified Wells criteria for predicting deep vein thrombosis (DVT).

Variable	Score
Lower limb trauma, surgery or immobilisation in a plaster cast	1
Bedridden for >3 days or surgery in last 4 weeks	1
Tenderness along the line of femoral or popliteal veins	1
Entire limb swollen	1
Calf >3 cm larger circumference than the other side	
10 cm below the tibial tuberosity	1
Pitting oedema	1
Dilated collateral superficial veins (not varicose veins)	1
Previous DVT	1
Malignancy (including treatment up to 6 months ago)	1
Intravenous drug abuse	3
Alternative diagnosis more likely than DVT	–2

Low probability (5%) of DVT (score –2 to 0), moderate probability (17%) of DVT (score 1–2), high probability (17–53%) of DVT (score >2).


Lower-extremity venous thromboembolism
Distal deep vein thrombosis
Proximal deep vein thrombosis
Superficial vein thrombosis
High-risk features
No high-risk features
Anticoagulation for at least 3 months
High-risk features
No high-risk features
Anticoagulation for at least 3 months
Serial Doppler ultrasonography
Doppler ultrasonography of bilateral lower extremities
No anticoagulation
Proximal or distal propagation
No propagation
Deep vein thrombosis identified
No deep vein thrombosis identified
Anticoagulation for at least 3 months
No anticoagulation
Manage as for distal or proximal deep vein thrombosis
Anticoagulation for 45 days

Contraindications to thrombolytic therapy

Absolute contraindications

Established cerebrovascular events (including transient ischemic attack) within last 2 months

Active bleeding diathesis

Recent (<10 days) gastrointestinal bleeding

Neurosurgery (intracranial or spinal) within last 3 months

Intracranial trauma within last 3 months

Intracranial malignancy or metastasis

Relative major contraindications

Cardiopulmonary resuscitation within last 10 days

Major nonvascular surgery or trauma within last 10 days

Uncontrolled hypertension (>180 mmHg systolic or >110 mmHg diastolic)

Puncture of noncompressible vessel

Intracranial tumor

Recent eye surgery

Minor contraindications

Hepatic failure, particularly with coagulopathy

Bacterial endocarditis

Pregnancy

Diabetic hemorrhagic retinopathy

Risk factors for major bleeding on anticoagulation

Age greater than 75

Recent major bleeding, ie, requiring transfusion of 2 or more units of blood; retroperitoneal, spinal, or intracranial bleeding

Severe liver dysfunction (baseline abnormal prothrombin time)

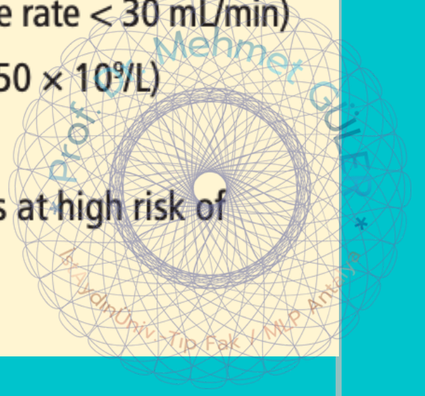
Severe renal impairment (creatinine clearance rate < 30 mL/min)

Severe thrombocytopenia (platelet count < 50 × 10^9/L)

Cancer

Acute hemorrhagic stroke or cerebral lesions at high risk of bleeding

Severe uncontrolled hypertension



Clinical suspicion of DVT

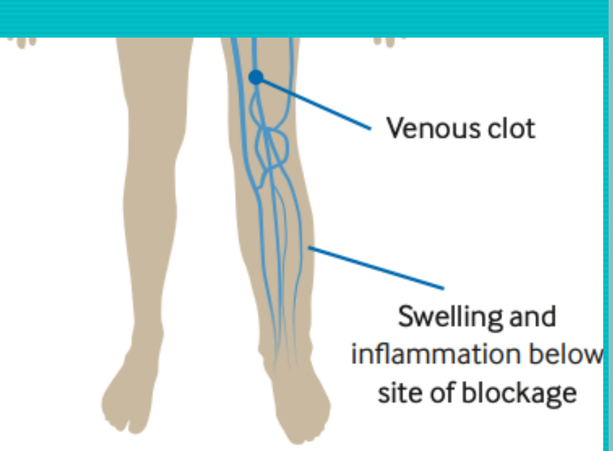
Common symptoms:

Swelling | Redness | Pain

Typically throbbing in nature, and comes on while walking or bearing weight

Patients may also be asymptomatic, having had investigation for other conditions such as pulmonary embolism or malignancy

Critical features or limb threatened



Modified Wells score (2003)

Active cancer treatment (ongoing, within 6 months, or palliative) +1	Paralysis, paresis, recent immobilisation of the lower limbs +1	Recently bedridden for more than 3 days, or major surgery within 4 weeks +1
Localised tenderness along distribution of deep venous system +1	Entire leg swelling +1	Calf swelling >3cm compared with asymptomatic leg +1
Pitting oedema (greater in symptomatic leg) +1	Collateral superficial veins (non-varicose) +1	Previous documented DVT +1

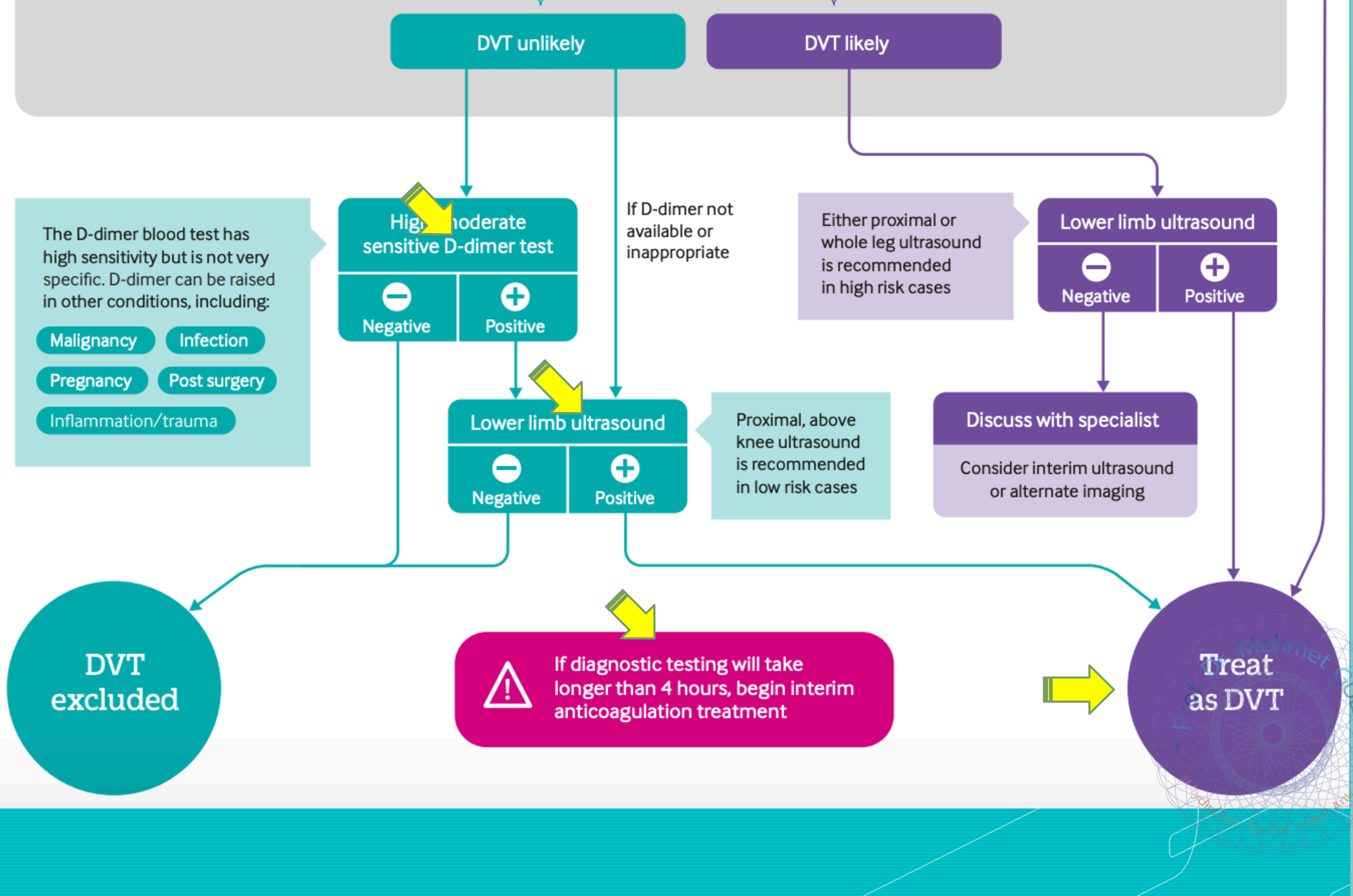
Alternative diagnosis as likely, or more likely than DVT −2

Total score:

−2	−1	0	1	2	3	4	5	6	7	8	9

DVT unlikely

DVT likely


DVT unlikely
DVT likely
The D-dimer blood test has high sensitivity but is not very specific. D-dimer can be raised in other conditions, including:
Malignancy
Infection
Pregnancy
Post surgery
Inflammation/trauma
High sensitive D-dimer test
Negative
Positive
If D-dimer not available or inappropriate
Either proximal or whole leg ultrasound is recommended in high risk cases
Lower limb ultrasound
Negative
Positive
Lower limb ultrasound
Negative
Positive
Proximal, above knee ultrasound is recommended in low risk cases
Discuss with specialist
Consider interim ultrasound or alternate imaging
DVT excluded
If diagnostic testing will take longer than 4 hours, begin interim anticoagulation treatment
Treat as DVT

z

Pulmonary Embolism

Most Common Presenting SYMPTOMS	Most Common Presenting SIGNS
Dyspnea at rest or with exertion (73%)	Tachypnea (54%)
Pleuritic pain (44%)	Calf or thigh swelling, erythema, tenderness, palpable cords (47%)
Cough (37%)	Tachycardia (24%)
Orthopnea (28%)	Rales (18%)
Calf or thigh pain or swelling or both (44%)	Decreased breath sounds (17%)
Wheezing (21%)	An accentuated pulmonic component of S2 (15%)
Hemoptysis (13%)	Jugular venous distension (14%)

TABLE 57.5 Modified Wells criteria for predicting pulmonary embolism (PE).

Variable	Score
Clinical signs and symptoms of deep vein thrombosis (DVT) (minimum of leg swelling and pain on palpation of deep veins)	3
Alternative diagnosis less likely than PE	3
Heart rate >100 bpm	1.5
Immobilisation >3 days or surgery within past 4 weeks	1.5
Previous DVT or PE	1.5
Haemoptysis	1
Malignancy (treatment or palliation within past 6 months)	1

A score of <4 means PE is unlikely (12.4%), >4 is suggestive of PE (37.1%).

Kronik Venöz Yetmezlik (KVY) - Kronik Kaçak

Neden Olur?

Genellikle geçirilmiş DVT sonrası kapakların hasar görmesi veya primer kapak yetmezliği sonucu kanın geriye kaçması (reflü). Bu durum venöz hipertansiyona yol açar.

Alt bacakta Kronik Venöz Yetmezlik bulguları: pigmentasyon, lipodermatoskleroz ve iyileşmiş ülser. (Görsel Kaynak: <IMAGE 1>)

Nasıl Tanınır?

Klinik Bulgular: Ayak bileğinde ödem, hiperpigmentasyon (hemosiderin birikimi), lipodermatoskleroz (ciltte sertleşme), "gaiter" bölgesinde (iç malleol üstü) venöz ülser.

Ne Yapılır?

Tedavinin Temeli: Kompresyon. Dereceli bası uygulayan varis çorapları veya elastik bandajlar venöz basıncı düşürür ve ödemi azaltır.

Figure 24-2. Characteristic hyperpigmentation of chronic venous insufficiency.

Figure 57.30 Post-thrombotic leg demonstrating features of eczema, pigmentation and mild lipodermatosclerosis.

Fig. 21.32 Chronic venous ulcer.

Figure 24-3. Venous ulceration located proximal to the medial malleolus.

Sadece SU ile yıka - Topikal antibiyotik KullanMA

Symptoms and signs of neuropathic ulcer versus ischemic ulcer

NEUROPATHIC ULCER	ISCHEMIC ULCER
Painless	Painful
Normal pulses	Absent pulses
Regular margins, typically punched-out appearance	Irregular margin
Often located on plantar surface of foot	Commonly located on toes, glabrous margins
Presence of calluses	Calluses absent or infrequent
Loss of sensation, reflexes, and vibration	Variable sensory findings
Increased in blood flow (arteriovenous shunting)	Decreased in blood flow
Dilated veins	Collapsed veins
Dry, warm foot	Cold foot
Bony deformities	No bony deformities
Red or hyperemic in appearance	Pale and cyanotic in appearance

Table 21.5 Differential diagnosis of leg ulceration

Clinical features	Arterial ulcer	Venous ulcer
Gender	Men > women	Women > men
Age	Usually presents > 60 years	Typically develops at 40–60 years but patient may not present for medical attention until much older; multiple recurrences are the norm
Risk factors	Smoking, diabetes, hyperlipidaemia and hypertension	Previous DVT, thrombophilia, varicose veins
Past medical history	Most have a clear history of peripheral, coronary and cerebrovascular disease	More than 20% have a clear history of DVT; many more have a history suggestive of occult DVT, i.e. leg swelling after childbirth, hip/knee replacement or long bone fracture
Symptoms	Severe pain is present unless there is (diabetic) neuropathy; pain may be relieved by dependency	About a third have pain, but it is not usually severe and may be relieved on elevation
Site	Normal and abnormal (diabetics) pressure areas (malleoli, heel, metatarsal heads, 5th metatarsal base)	Medial (70%), lateral (20%) or both malleoli and gaiter area
Edge	Regular, 'punched-out', indolent	Irregular, with neo-epithelium (whiter than mature skin)
Base	Deep, green (sloughy) or black (necrotic) with no granulation tissue; may involve tendon, bone and joint	Pink and granulating but may be covered in yellow-green slough
Surrounding skin	Features of severe limb ischaemia	Lipodermatosclerosis, varicose eczema, atrophe blanche
Veins	Empty, 'guttering' on elevation	Full, usually varicose
Swelling	Usually absent	Often present

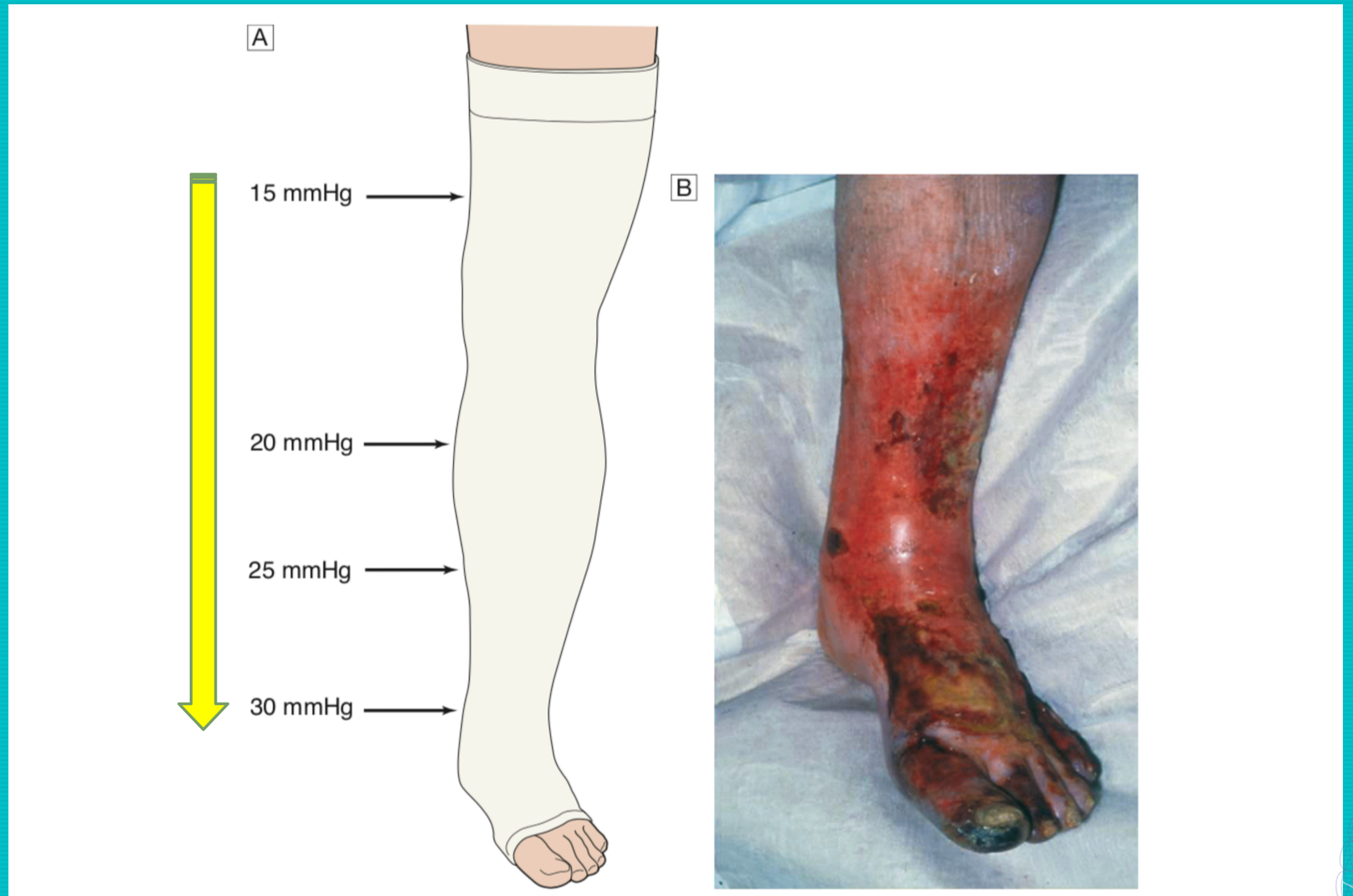


Fig. 21.33 Graduated elastic compression for venous ulcer. A Compression from the base of the toes to the tibial tuberosity usually suffices. B Extensive necrosis in a patient treated with compression for a venous ulcer in the presence of significant arterial disease. Above-knee amputation was required.

Venöz Bacak Ülseri

- Yaşam kalitesini oldukça bozar
- Ülserlerin iyileşmesi zor, tekrarlaması kolaydır
- Tedavisi pahalıdır
- Tedavinin temeli; kompresyon ve venöz hipertansiyonun azaltılması

Summary box 57.2

Venous leg ulcer

- Is associated with a profound impairment in quality of life
- Ulcers are not infrequently difficult to heal and prone to recurrence
- The treatment of these chronic wounds is associated with high costs to healthcare systems and patients
- The mainstay of treatment is the reduction in venous hypertension, with compression and increasingly superficial venous ablation

FIG. 65.7 Venous stasis ulcer.

FIG. 65.8 Healed venous stasis ulcer.

Lymphoedema.

LENFÖDEM
Elevasyon
Kompresyon

Fig. 21.35 Elephantiasis of the lower limb. Note that this oedema is nonpitting suggestive of chronic lymphoedema.

ELEFANTİYAZİS
Wuchereria Bancrofti
Albendozole-Ivermectin
Dietylcarbamazine

Venöz Hastalık

Yetişkin nüfusta - Variköz ven %50+,
Kronik Venöz Yetmezliğe (KVY) bağlı cilt değişiklikleri %10
Yaşam boyu kronik venöz ülserasyon riski %1

KVY'de temel tedavi – Kompresyon
Arteriyel dolaşım yeterli olmalı (Nabızlar(+), ABİ>0,8 olmalı)

Tedavi Planı için – Duplex Ultrasonografi
Yüzeyel/Derin, Reflü/Obstrüksiyon

Postop DVT - Sıklıkla ASEMPTOMATİK
Tanı için Duplex USG, Venografi
PE hastalarının çoğunda bacak bulgusu YOK
Tanı için BT Pulmoner Anjiografi

Tüm Hastane Hastaları İçin - TROMBOEMBOLİK RİSK DEĞERLENDİRMESİ şart,
Gerekiyorsa PROFLAKSİ.
Halen hastanelerde potansiyel olarak
önlenebilir ölümlerin en sık nedeni PULMONER EMBOLİ

Venous disease

- Varicose veins are present in over 50% of the adult population; approximately 10% have skin changes of chronic venous insufficiency; and the life time risk of chronic venous ulceration is about 1%
- Compression therapy is the mainstay of treatment for chronic venous insufficiency, but should never be implemented unless the arterial status of the leg is known to be satisfactory (palpable pedal pulses and/or an ABPI > 0.8)
- If patients with lower limb venous disease are being considered for surgical or endovenous treatment, they should undergo duplex ultrasonography to delineate the pattern of superficial and deep venous reflux and obstruction
- DVT in postoperative patients is often asymptomatic. Most postoperative patients developing (fatal) PE have normal legs on clinical examination. Duplex ultrasound or venography should be used to confirm the diagnosis of DVT; V/Q scan or CT pulmonary angiography can be used to diagnose PE
- All hospital patients, medical and surgical, should have their thromboembolic risk assessed and receive prophylaxis accordingly. PE remains the most common cause of potentially preventable death in hospital

Periferik Damar Hastalığı - Özetin Özeti

1 

BACAK AĞRISINI CİDDİYE AL.

Klodikasyo, sistemik aterosklerozun bir belirtisidir. Hastanın kardiyak riskini de yönet. Şüphedeysen mutlaka **ABİ ölç**.

2 

TEK TARAFLI BACAK ŞİŞLİĞİ = DVT (Aksi İspatlanana Kadar).

Pulmoner emboli riskini asla unutma. Acil **Dupleks USG** iste.

3 

PULSATİL KARIN KİTLESİ = AAA

Tesadüfen saptanan anevrizmayı mutlaka bir uzmana **yönlendir**. Rüptür triadı (ağrı, hipotansiyon, kitle) ölümcül bir acildir.

4 

GİA (TIA) = İNME UYARISIDIR.

'Geçti' diye düşünme. GİA geçiren hastayı acil olarak değerlendir ve **Karotis Dupleks USG** planla. Zaman beyindir.

5 

DİYABETİK AYAK = İSKEMİ + ENFEKSİYON.

Nöropatik bir ülserin altında yatan Periferik Arter Hastalığını (PAH) gözden kaçırma. Bu ikili, amputasyonun en sık nedenidir. Mutlaka vasküler durumunu değerlendir.