عوارض سکته مغزی و مدیریت آن

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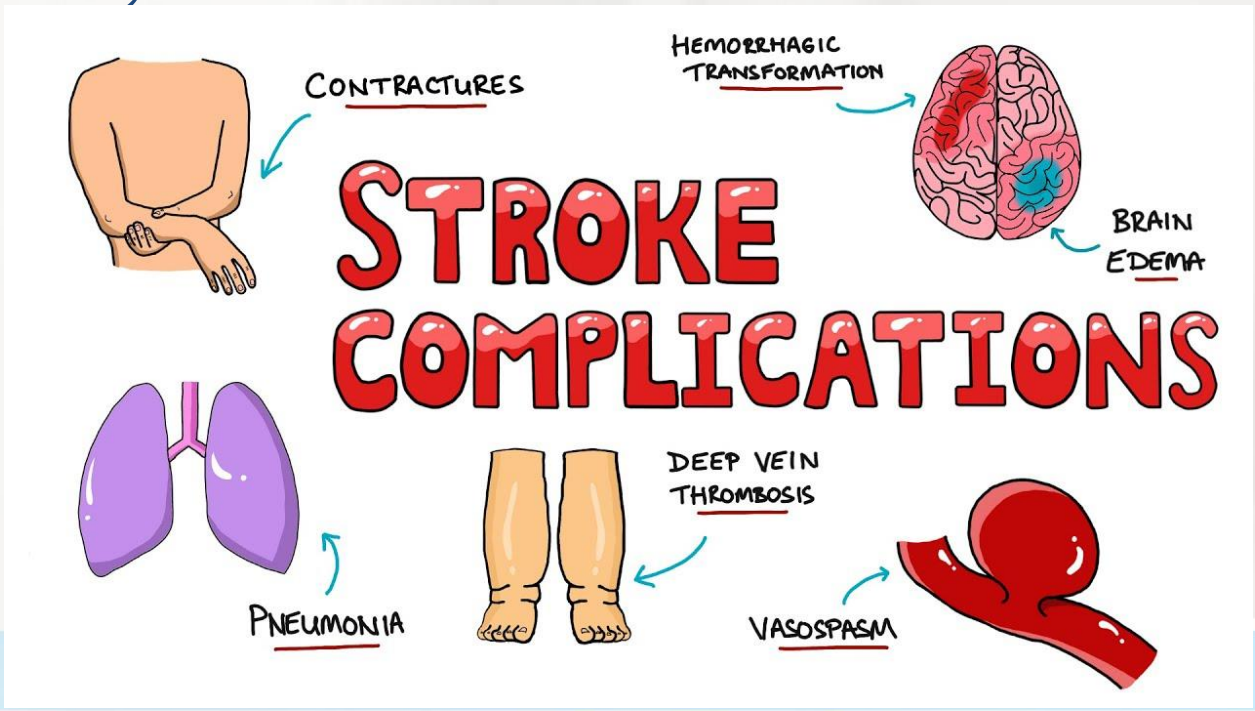
- You may experience one or more of these common complications after your stroke.

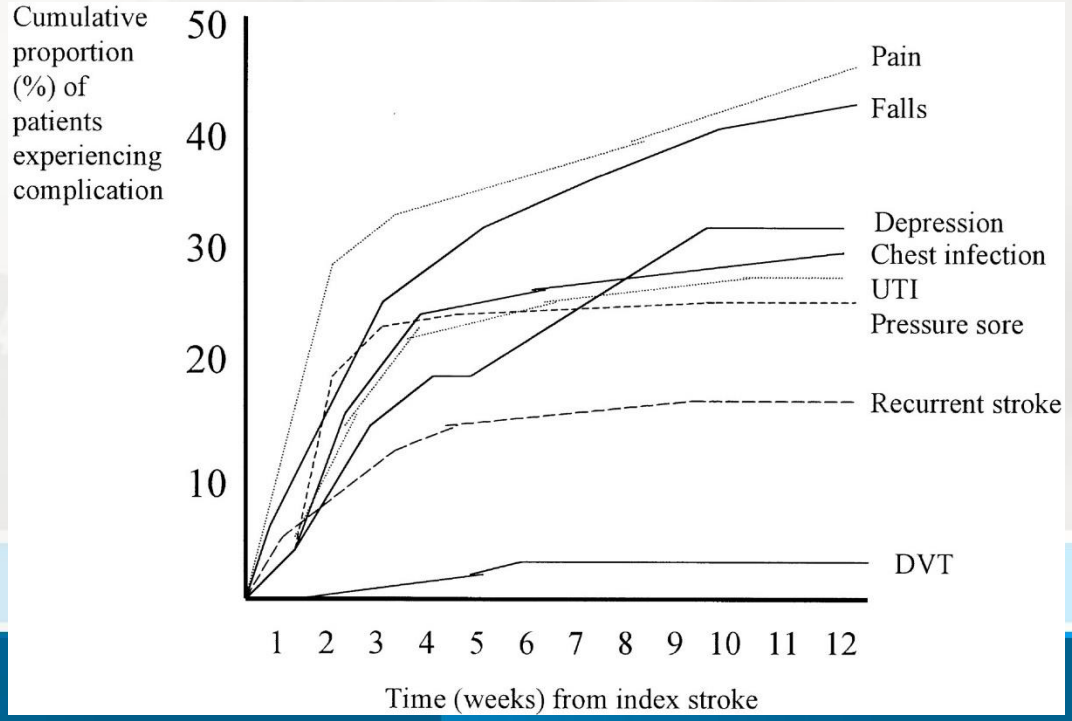


Stroke complications

- Blood clots or deep vein thrombosis (DVT)
- Depression and other mood changes.
- Pneumonia / UTI
- Involuntary muscle tightening or spasticity.
- Chronic headaches
- Bed sores
- Fall

Prevalence of post stroke complications

- Complications during hospital admission were recorded in **(85%) of stroke** patients



Neurological

- Recurrent stroke (9% of patients),
- Epileptic seizure (3%);
- Brain Edema

Infections

- Urinary tract infection (24%),
- Chest infection (22%),
- Others (19%);

Mobility related

- Falls (25%),
- Falls with serious injury (5%),
- Pressure sores (21%);
- Thromboembolism—deep venous thrombosis (2%),
- Pulmonary embolism (1%);

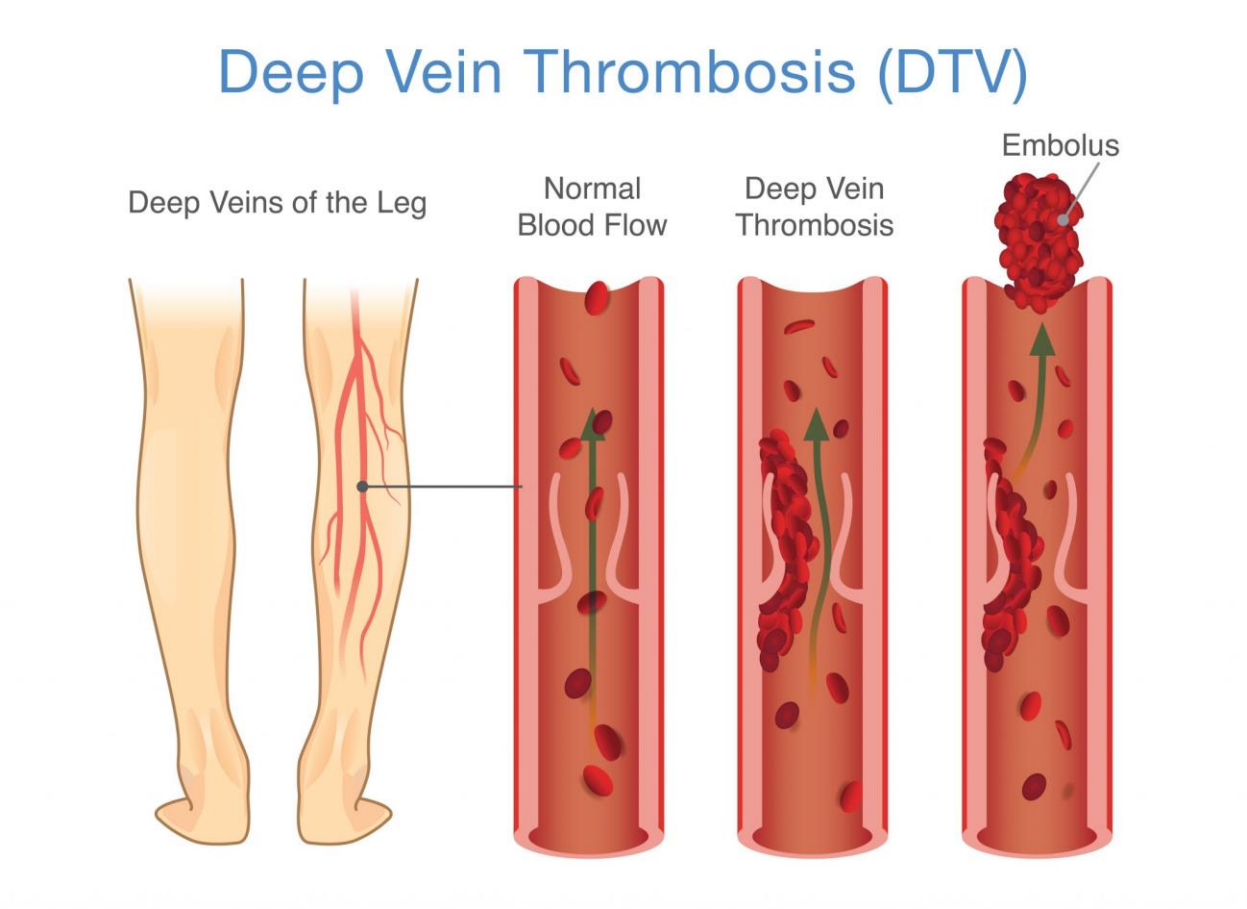
Pain

- Shoulder pain (9%),
- other pain (34%);

Psychological

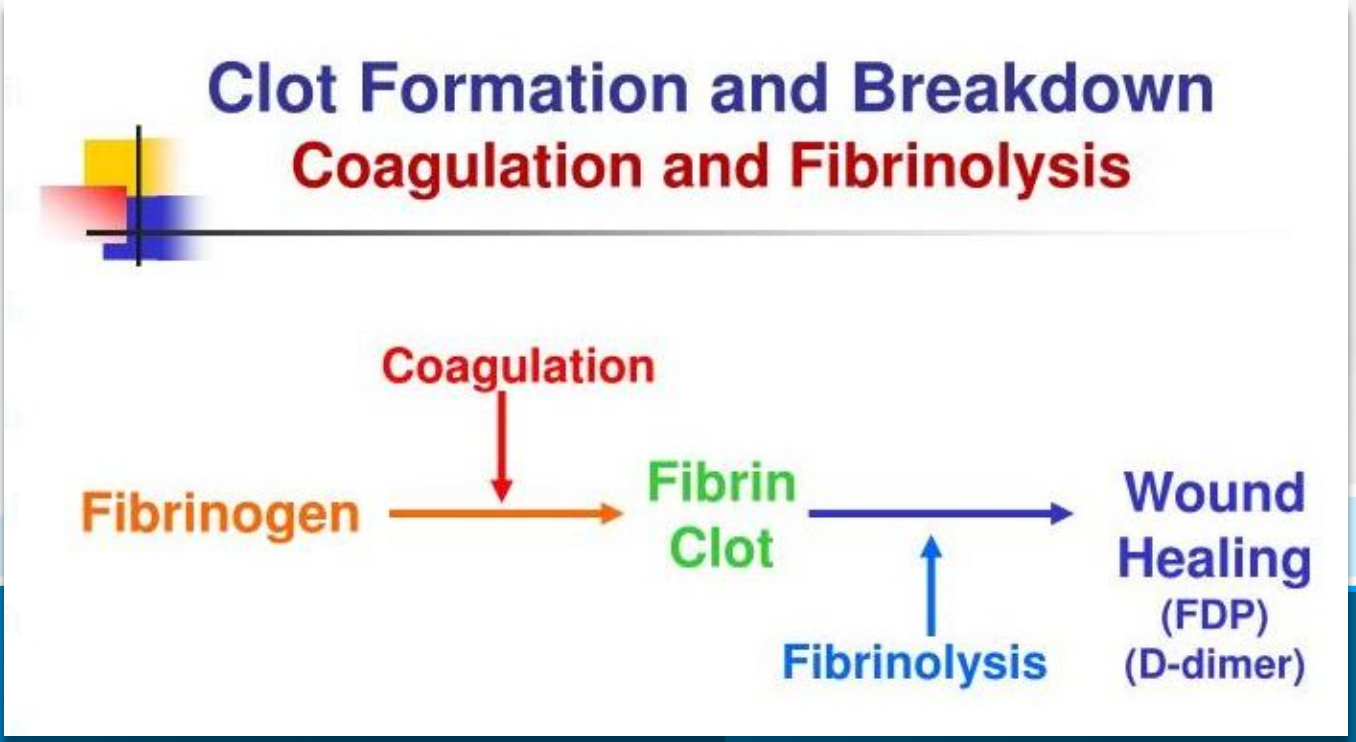
- Depression (16%),
- Anxiety (14%),
- Emotionalism (12%),
- Confusion (56%)

DVT


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

- Venous thrombi may originate in **superficial or deep** veins anywhere in the body, most commonly developing in the leg veins.
- The **deep and intramuscular calf veins** are frequent sites of thrombus initiation

- When the balance of the **fibrin generation** overwhelms the **fibrinolytic system**, thrombus extension into the proximal leg veins may occur



DIAGNOSIS

TABLE 106-1 MODEL FOR DETERMINING CLINICAL SUSPICION OF DEEP VEIN THROMBOSIS

VARIABLES	POINTS[a]
Active cancer (treatment ongoing or within previous 6 mo or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden ≥ 3 d or major surgery within the past 4 wk	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Affected calf 3 cm greater than asymptomatic calf (measured 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Dilated superficial veins (nonvaricose)	1
Previous deep vein thrombosis (DVT)/pulmonary embolism	1
Alternative diagnosis is at least as likely as that of deep vein thrombosis	–2
Total points	

[a]Total score < 2 indicates DVT unlikely; score ≥ 2 indicates DVT likely.

Laboratory testing

- **D-Dimer** is a cross-linked fibrin degradation product generated when mature thrombus is cleaved by plasmin.

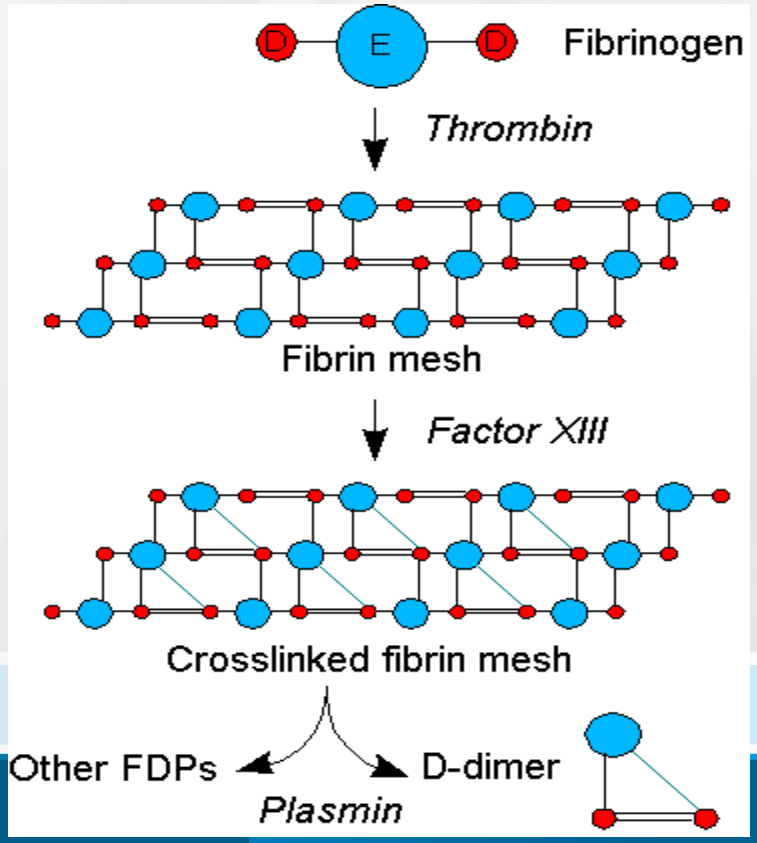
- Levels of D-dimer are typically elevated in the presence of acute DVT

Elevated D-dimer levels including

- Advanced age,
- Inflammation,
- Infection,
- Malignancy,
- Recent surgery

D Dimer

- If the D-dimer result is negative, then DVT essentially ruled out,



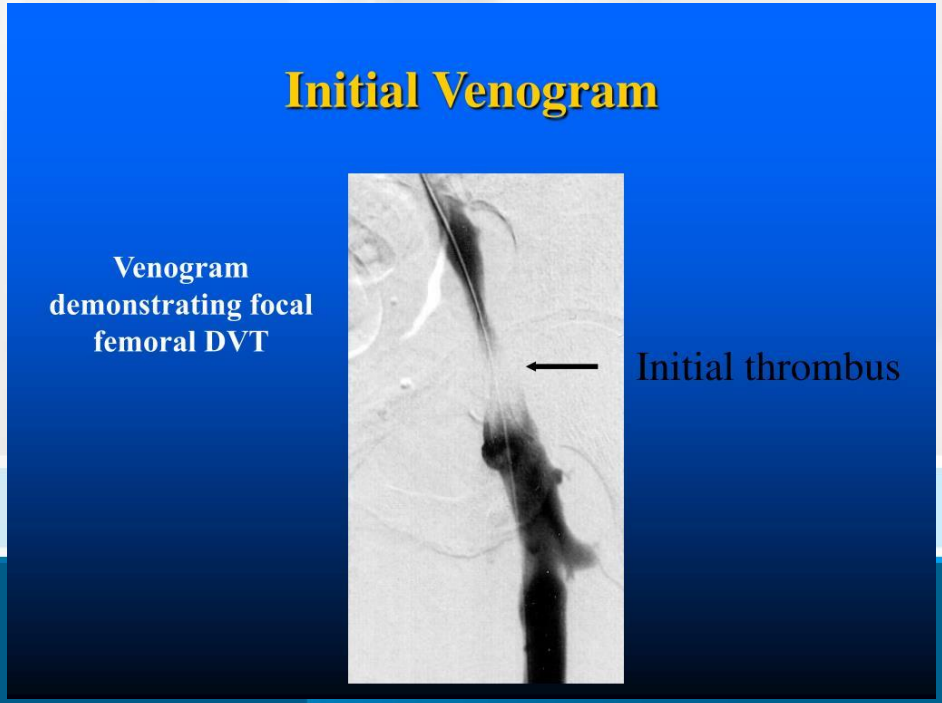
Contrast venography

- Contrast venography is the gold standard for DVT diagnosis,

- But has largely been replaced by noninvasive compression ultrasonography.

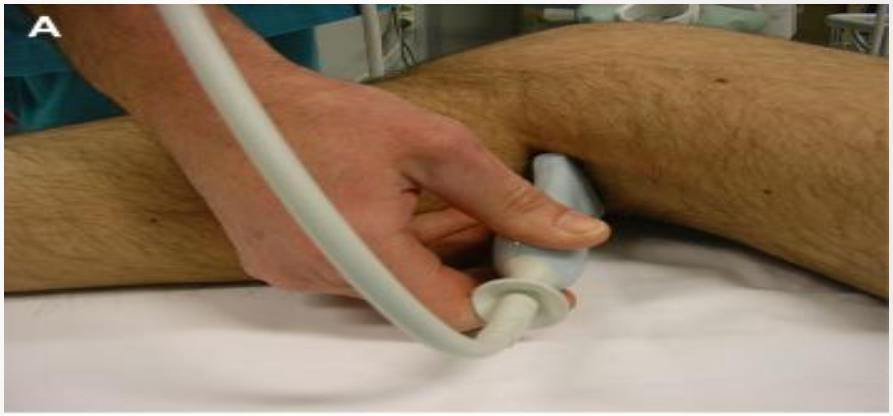
Contrast Venography

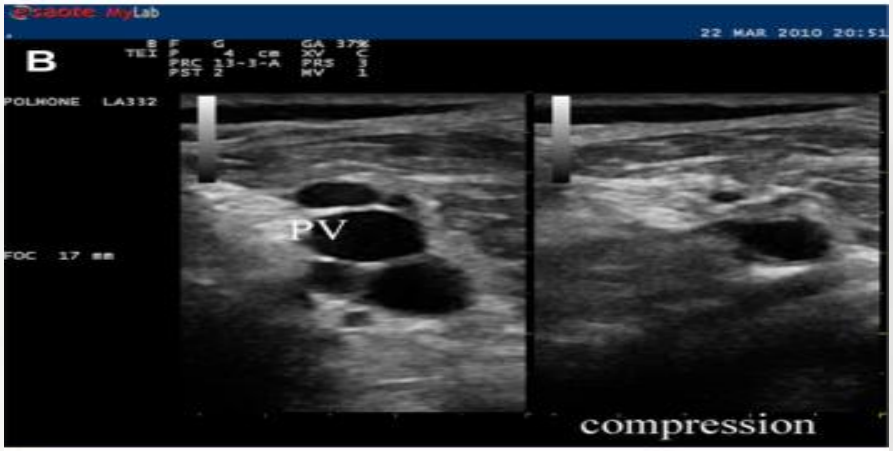
- The role of venography is generally limited to clarify discrepancies between high clinical probability and nondiagnostic ultrasonography

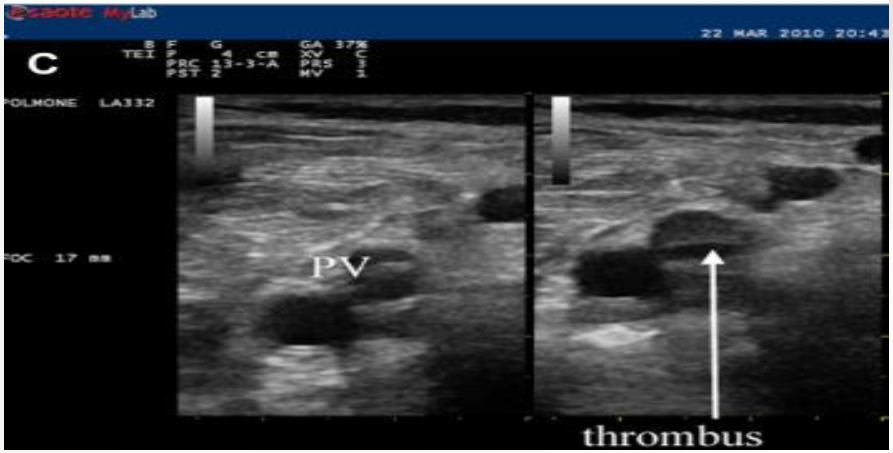


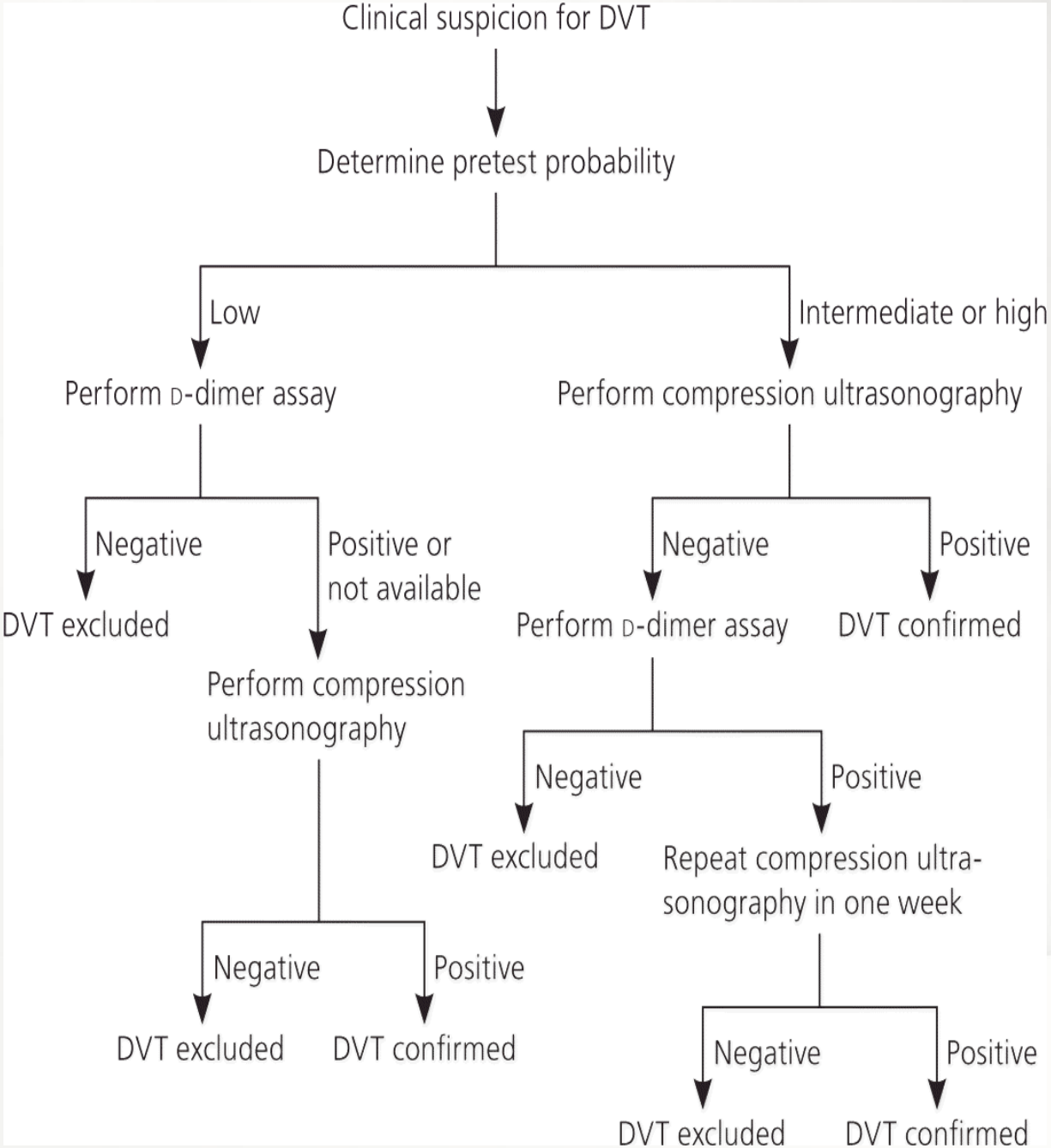
Compression Ultrasonography

- Whole-leg compression ultrasound involves imaging
- the entire venous network **every 1 to 2 cm** along its course with external pressure applied through the ultrasound transducer to visualize **complete vessel collapse.**


A


B
PV
compression


C
PV
thrombus


Clinical suspicion for DVT
Determine pretest probability
Low
Intermediate or high
Perform D-dimer assay
Perform compression ultrasonography
Negative
Positive or not available
Negative
Positive
DVT excluded
Perform D-dimer assay
DVT confirmed
Perform compression ultrasonography
Negative
Positive
DVT excluded
Repeat compression ultra-sonography in one week
Negative
Positive
Negative
Positive
DVT excluded
DVT confirmed
Negative
Positive
DVT excluded
DVT confirmed

Pulmonary Embolism

- Presenting signs and symptoms of acute PE are nonspecific and often insufficient for diagnosis.
- Dyspnea,
- Tachypnea,
- Tachycardia
- Chest pain.

- Symptoms of DVT (leg pain and swelling) are present in more than 40% of patients with acute PE

- Simultaneous DVT is identified in up to 60% of patients diagnosed with a PE.

TABLE 106-2 MODEL FOR DETERMINING A CLINICAL SUSPICION OF PULMONARY EMBOLISM

VARIABLES	POINTS[a]
Clinical signs and symptoms of deep vein thrombosis (minimum of leg swelling and pain with palpation of the deep veins)	3.0
An alternative diagnosis is less likely than pulmonary embolism (PE)	3.0
Heart rate > 100 beats/min	1.5
Immobilization or surgery in the previous 4 wk	1.5
Previous deep vein thrombosis/pulmonary embolism	1.5
Hemoptysis	1.0
Malignancy (treatment ongoing or within previous 6 mo or palliative)	1.0
Total points	

[a]Total score ≤ 4 indicates PE unlikely; score > 4 indicates PE likely.

Laboratory testing

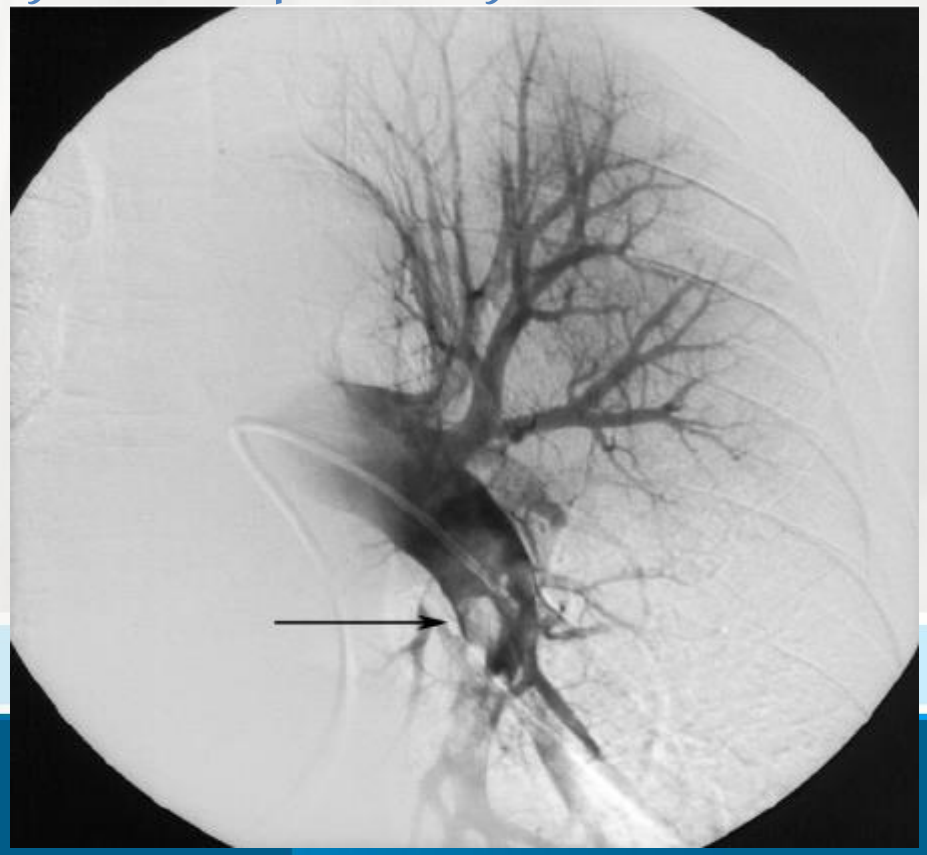
- Similar to its role in DVT evaluation, a negative D-dimer result combined with unlikely pretest probability can safely exclude the diagnosis of PE.

Chest Radiography

- The diagnostic utility of chest radiography in suspected PE is primarily to exclude other causes of dyspnea and hypoxemia (ie, pneumothorax or pneumonia), as most chest radiographs are normal with an acute PE.

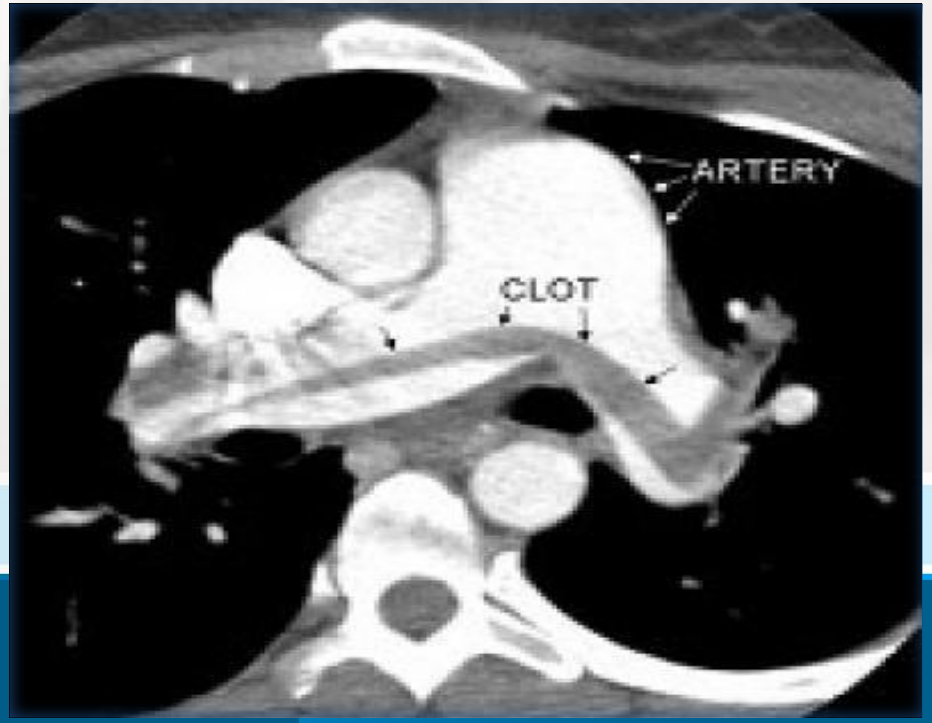
Pulmonary Angiography

- Pulmonary angiography was the reference standard for diagnosis of PE but has largely been replaced by noninvasive imaging



Computed Tomographic Pulmonary Angiography

- CTPA have made it the primary imaging modality for suspected PE, except when contraindicated (severe renal insufficiency contrast allergy).

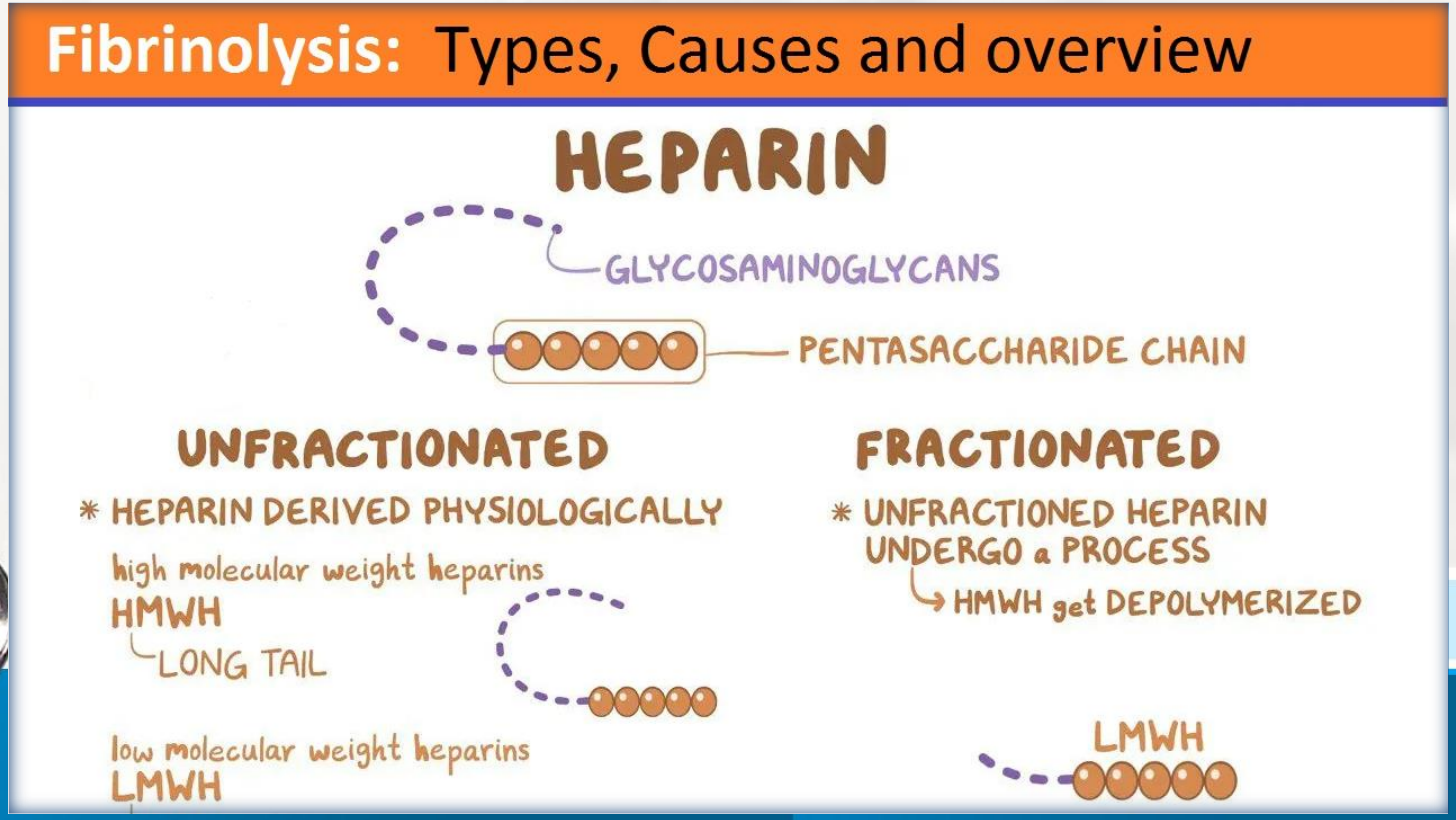


Preventing Deep Vein Thrombosis After Stroke

- Previous history of DVT,
- Dehydration
- comorbidities such as malignant diseases or clotting disorders.
- Patients with an increased risk of DVT should receive prophylactic treatment

- Patients should be **mobilized** as soon as possible and should be kept well **hydrated**

- Administered **low-dose unfractionated** heparin is preferred to unfractionated heparin



Intracerebral hemorrhage

- **low-dose subcutaneous low-molecular-weight heparin** is probably safe after documentation of cessation of active bleeding, and may be considered on an individual basis after 3 to 4 days from stroke onset.

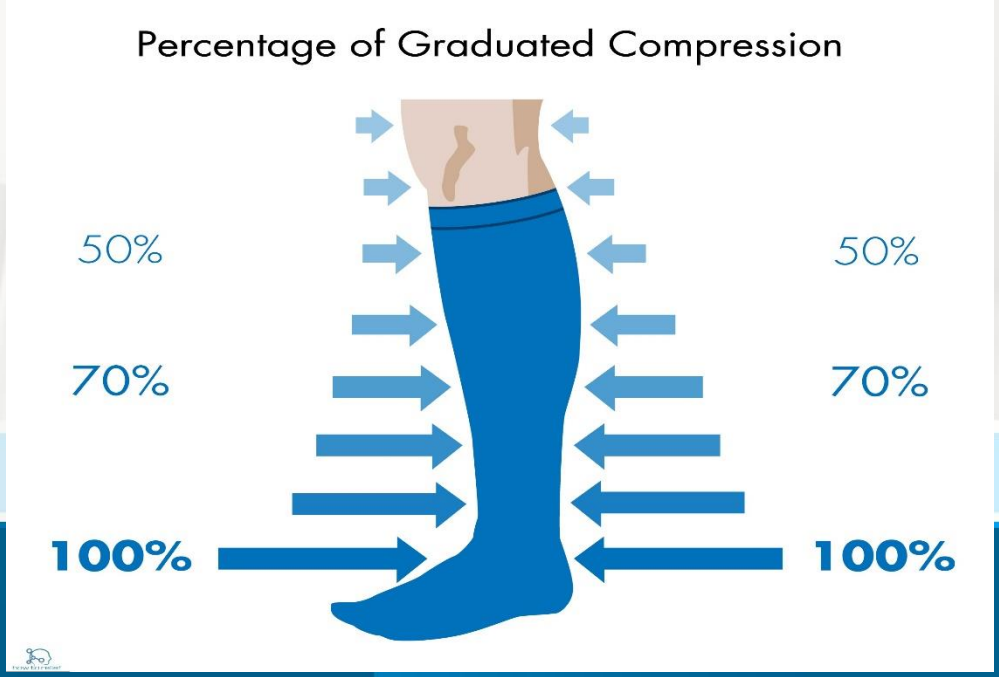
Physical methods

- wearing graduated compression stockings,
- intermittent pneumatic compression
- electrical stimulation of leg muscles

- The physical methods are used to increase the blood flow in the leg veins and reduce the risk of clots forming

Compression stockings

- Graduated compression stockings were no better than 'best medical treatment' in reducing the risk of DVT after stroke. Stockings caused more skin problems (for example ulcers and blisters) on the legs.



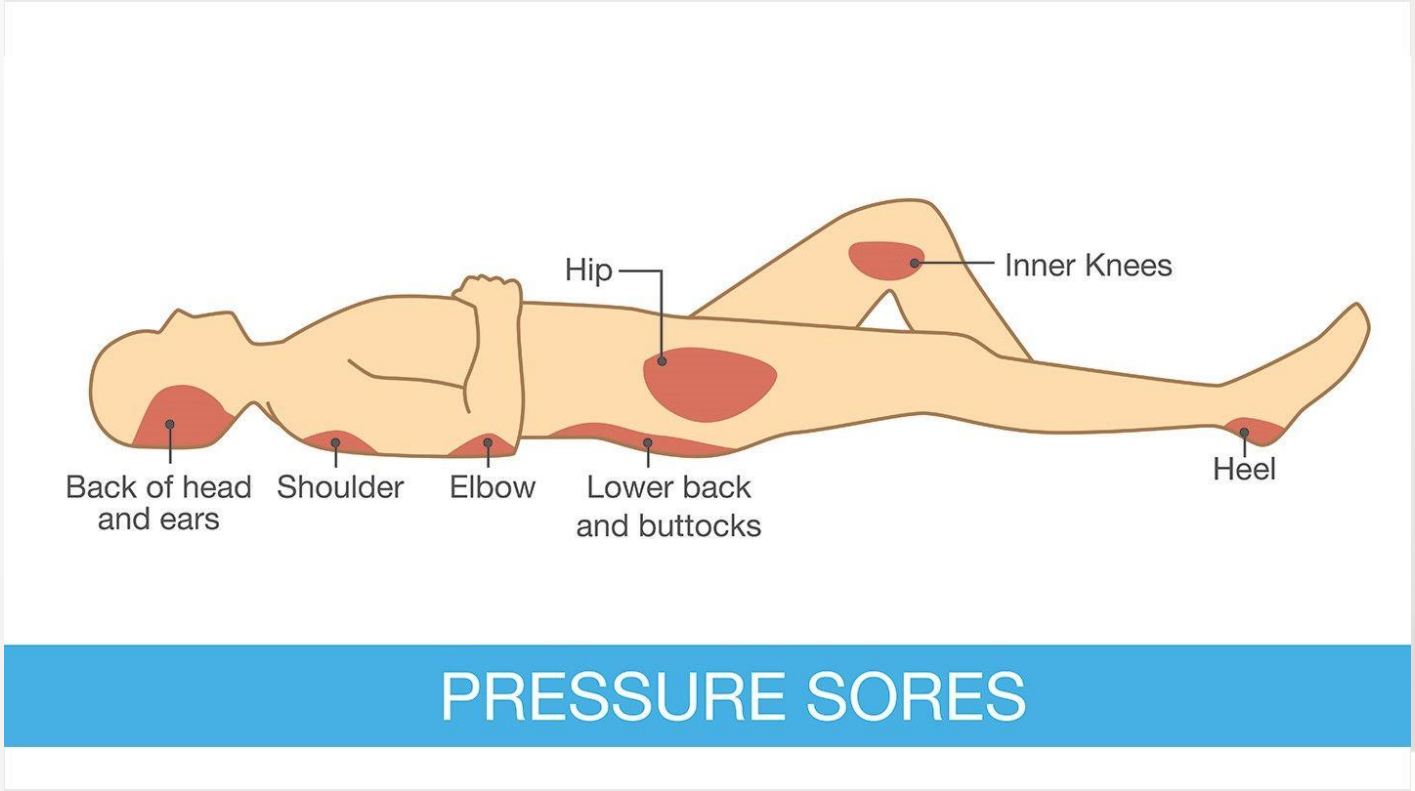
- Intermittent pneumatic compression appeared promising but was not proven to be definitely beneficial.

- **Aspirin** may also be effective for patients with ischemic stroke who have contraindications to anticoagulants

TREATMENT OF VENOUS THROMBOEMBOLISM

- Prompt administration of parenteral anticoagulation, followed by risk stratification, and initiation of long-term oral anticoagulation (OAC) to minimize recurrence VTE-associated morbidity (postthrombotic syndrome and chronic thromboembolic pulmonary hypertension).

Pressure Ulcers



- Pressure ulcers occur in **all health care settings**

- the prevalence of pressure ulcers in acute care units has declined by 1% to 2% over the last decade
- **Critical care** settings present increased risk with pressure ulcer **prevalence rates reported at 13%**

- Pressure ulcers generally occur **within the first 2 weeks of hospitalization** (the **first 5 days in critical care units**),
- And of those patients with an ulcer, more than half develop them after admission

Rehabilitation facilities

- Rehabilitation facilities present special concerns related to pressure ulcer development, because **patients in these facilities have conditions that limit mobility**,
- Such as **spinal cord injury (SCI), traumatic brain injury, cerebral vascular accident, burns, multiple trauma**, or a **chronic neurologic disorder**.

MORBIDITY ASSOCIATED WITH PRESSURE ULCERS

- Pain
- Septicemia
- Prolonged hospitalization
- Increased death rates
- Quality issue
- Costly

Detection of Pressure-Induced Tissue Damage

- High-resolution ultrasound
- Thermography
- Measurement of the water content of the skin and underlying tissue can be accomplished using surface electrical capacitance devices

SEM

- Pressure ulcers are caused by **mechanical force** compressing tissues between the bony skeleton and external

- Surfaces **occluding capillaries and lymphatics** with resultant ischemia
- Buildup of metabolic cellular waste products,
- Release of **oxygen free radicals** from reperfusion injury,
- **Cellular apoptosis** from cell deformation.

Pathophysiology

- 1. Ischemia caused by capillary occlusion
- 2. Impairment in lymphatic flow with increase in metabolic waste products
- 3. Reperfusion injury (damage that occurs because of the inflammatory response that occurs when blood flow resumes to the ischemic tissues)
- 4. Deformation of tissue cells

Presentation

- The first clinical sign of pressure ulcer formation, blanchable erythema, presents as discoloration of a patch or flat, nonraised area of the skin larger than 1 cm.

Prevention

- Prevention includes **screening for risk** followed by **risk assessment** using standardized risk assessment tools to determine individual-specific risk and implementing targeted prevention interventions based on identified risk factors.

Movement at night

- Individuals with more than **50 movements** a night did not develop pressure ulcers compared to 90% of individuals with 20 or fewer spontaneous body movements at night who developed a pressure ulcer.

- **Incontinence, malnutrition, impaired mental status,** and **altered sensation** or response to pain and discomfort are all risk factors with strong relationships to pressure ulcer development in prospective studies.

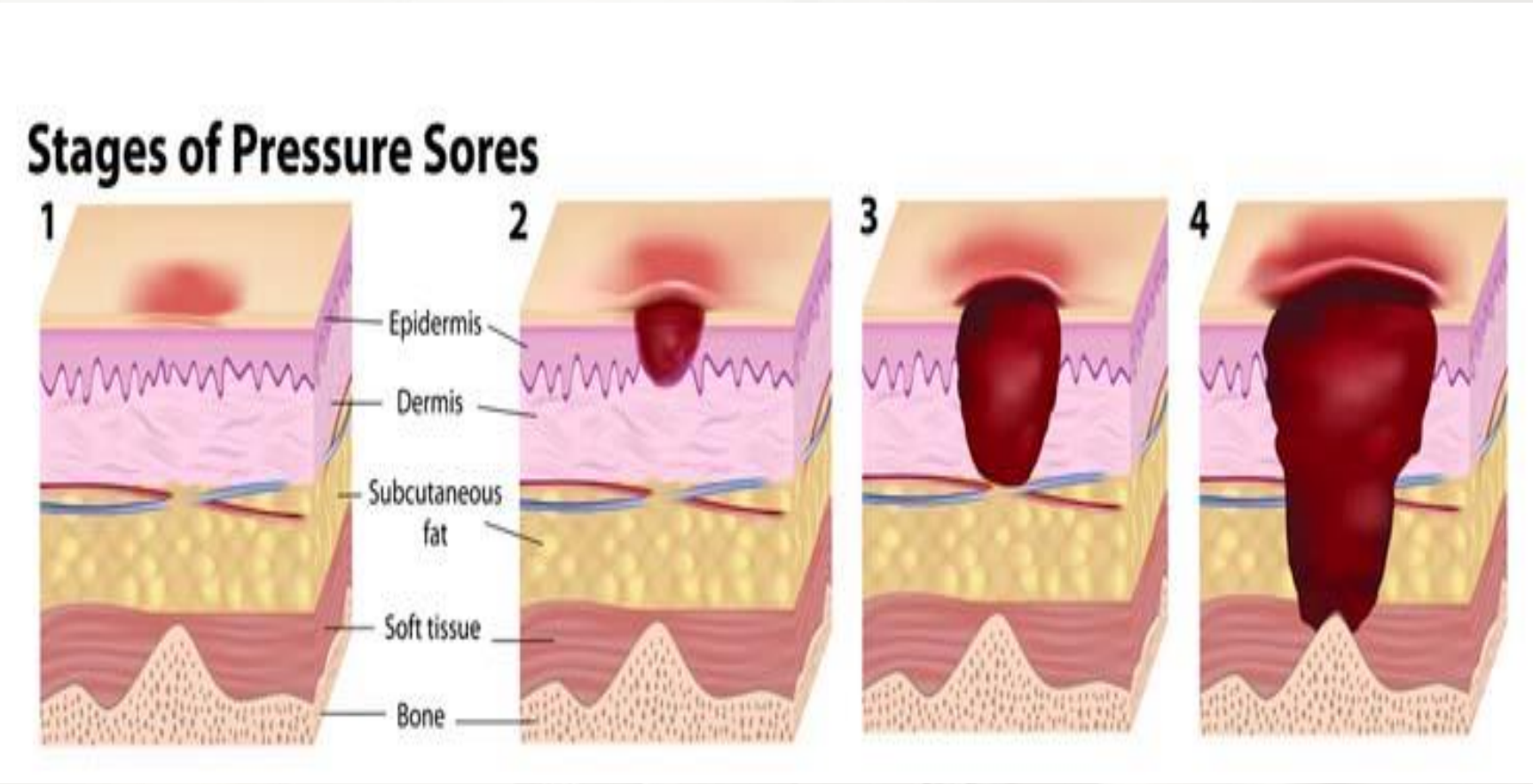
Common sites

- Pressure ulcers are most commonly found over bony prominences subjected to external pressure.
- The most common locations are **sacrum, ischial tuberosities, trochanters,** and **heels**

- **with sacral and heel sites most frequent**

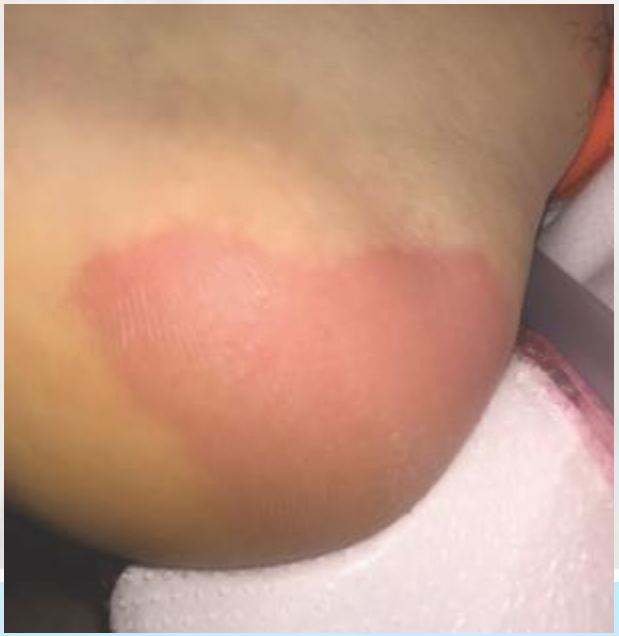
Medical record documentation

- Pressure ulcer risk status,
- Prevention strategies,
- Pressure ulcer assessment (size, stage, location, and description of wound bed minimally),
- Treatment plan
- Evaluation of treatment success


Stages of Pressure Sores
1
2
3
4
Epidermis
Dermis
Subcutaneous fat
Soft tissue
Bone

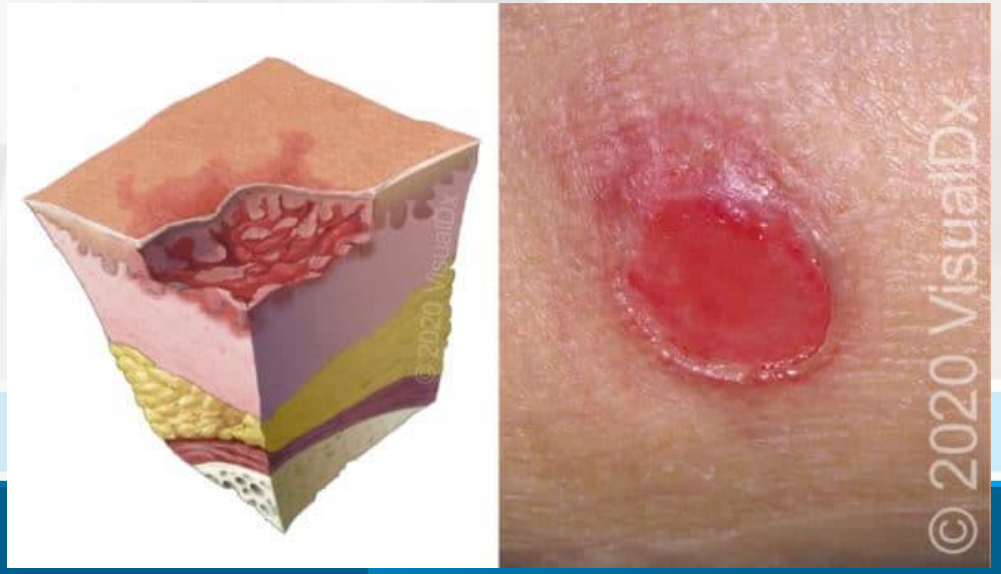
Stage 1

- **Intact skin** with a localized area of **non-blanchable erythema**, which may appear differently in darkly pigmented skin



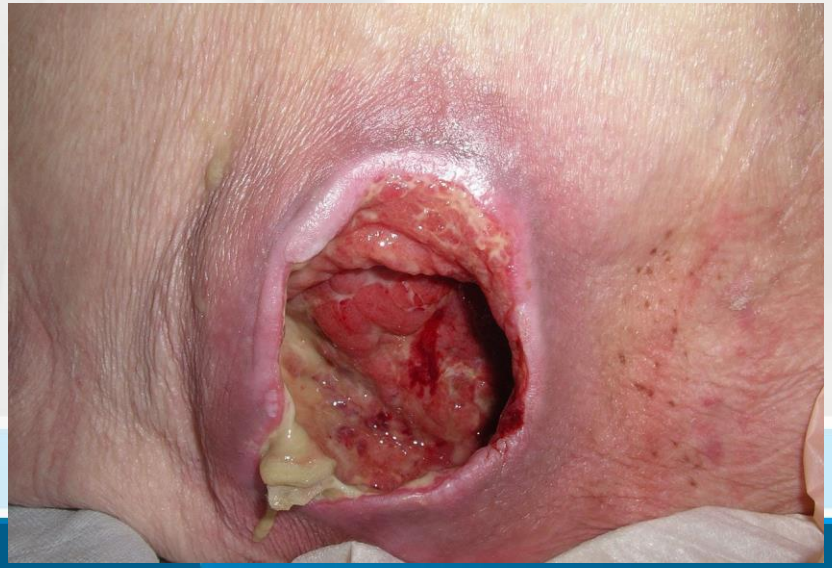
Stage 2

- **Partial-thickness loss of skin** with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. **Adipose (fat) is not visible** and deeper tissues are not visible.



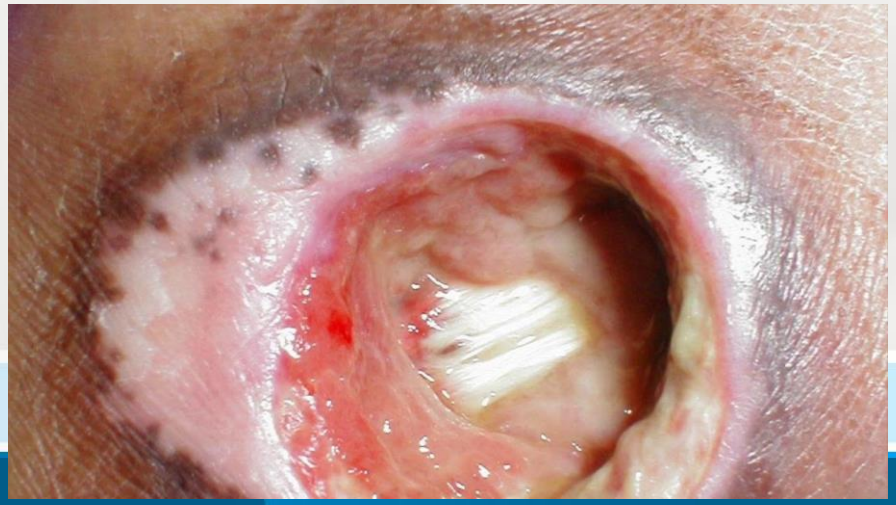
Stage 3

- **Full-thickness loss of skin**, in which **adipose (fat) is visible** in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible.



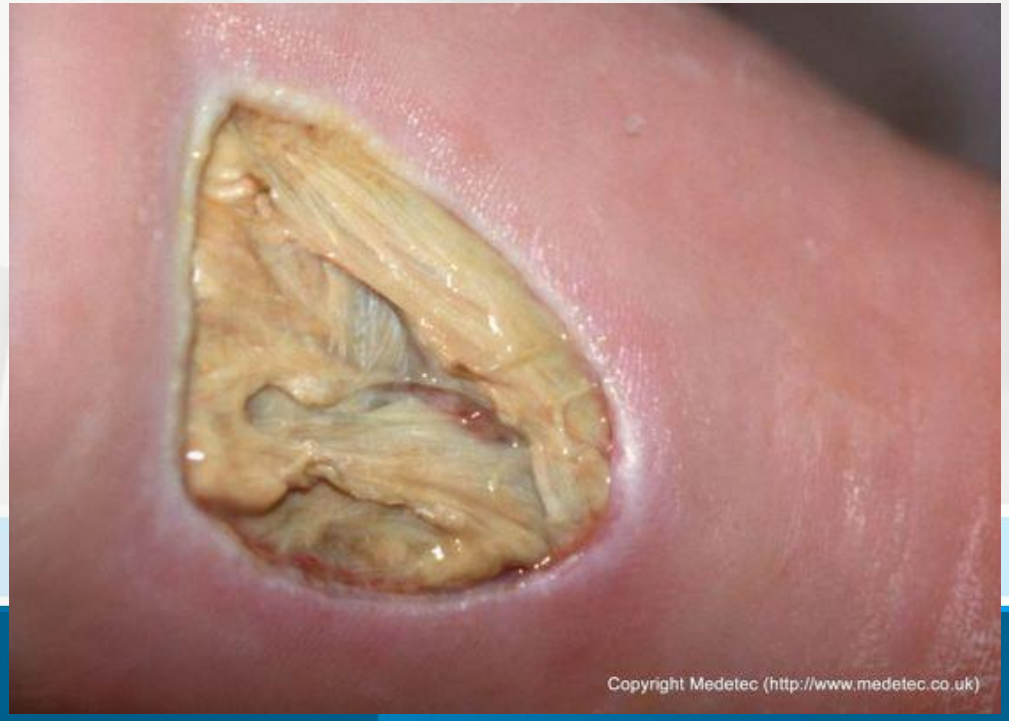
Stage 4

- Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer.



Unstageable

- Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar.



- If slough or eschar is removed, a stage 3 or stage 4 pressure injury will be revealed.
- Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on an ischemic limb or the heel(s) should not be removed.

Eschar

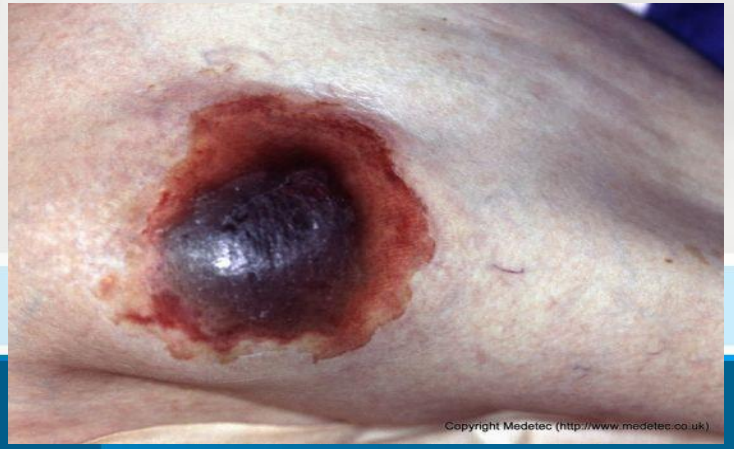
- Eschar, pronounced es-CAR, is **dead tissue that sheds or falls off from the skin**. It's commonly seen with pressure ulcer wounds (bedsores). Eschar is typically tan, brown, or black, and may be crusty

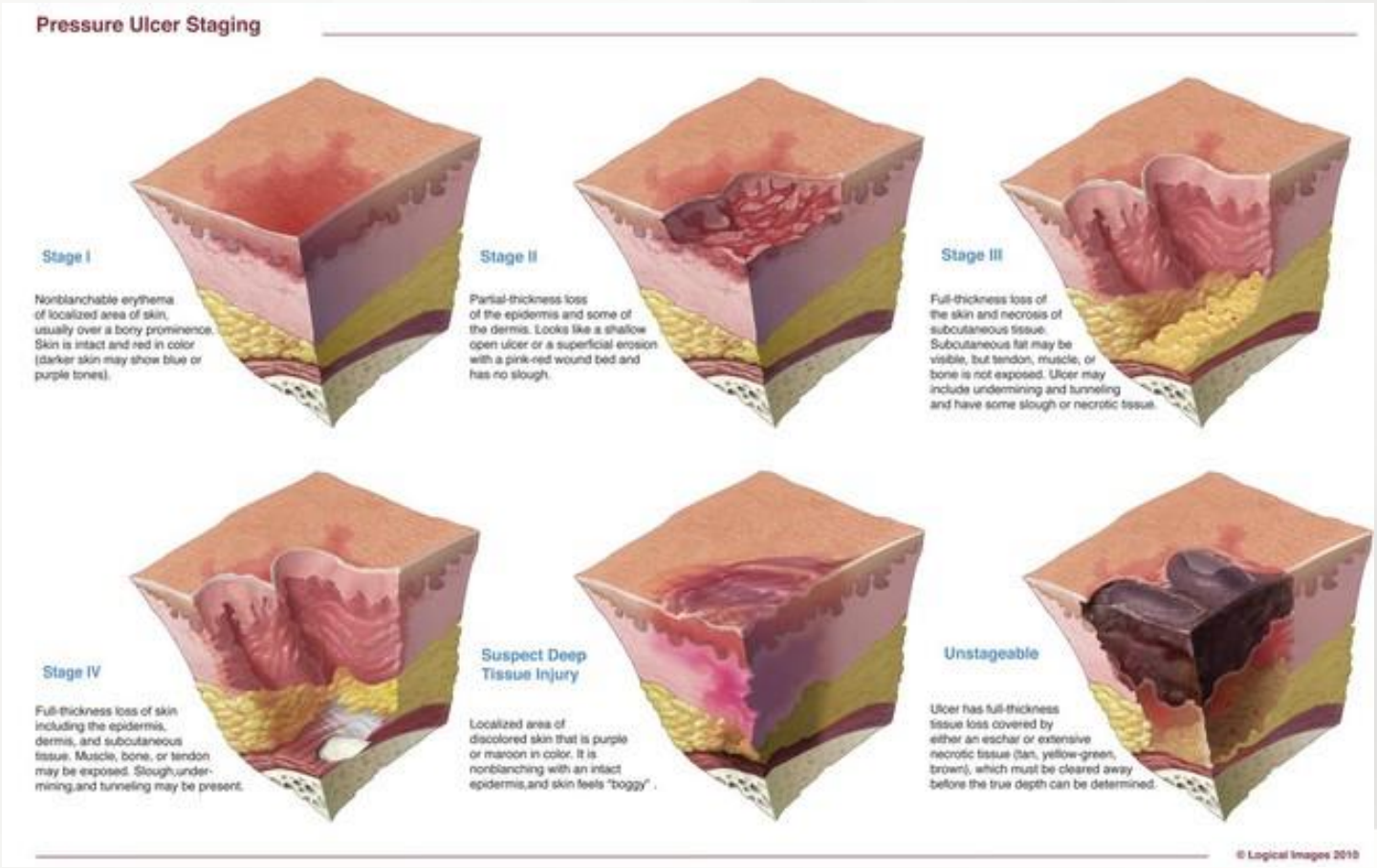
Slough

- Slough is essentially the by-product of the inflammatory phase of wound healing comprising of fibrin, leucocytes, dead and living cells, microorganisms and proteinaceous material
- The appearance of slough is typically a **pale yellow**, viscous fibrinous tissue and can range from yellow to tan, usually, but not always,

Suspected deep Tissue injury

- Intact or non-intact skin with localized area of persistent **non-blanchable deep red, maroon,**
- **purple** discoloration or epidermal separation revealing a dark wound bed or blood filled blister.




Pressure Ulcer Staging
Stage I
Nonblanchable erythema of localized area of skin, usually over a bony prominence. Skin is intact and red in color (darker skin may show blue or purple tones).
Stage II
Partial-thickness loss of the epidermis and some of the dermis. Looks like a shallow open ulcer or a superficial erosion with a pink-red wound bed and has no slough.
Stage III
Full-thickness loss of the skin and necrosis of subcutaneous tissue. Subcutaneous fat may be visible, but tendon, muscle, or bone is not exposed. Ulcer may include undermining and tunneling and have some slough or necrotic tissue.
Stage IV
Full-thickness loss of skin including the epidermis, dermis, and subcutaneous tissue. Muscle, bone, or tendon may be exposed. Slough,under-mining,and tunneling may be present.
Suspect Deep Tissue Injury
Localized area of discolored skin that is purple or maroon in color. It is nonblanching with an intact epidermis,and skin feels "boggy" .
Unstageable
Ulcer has full-thickness tissue loss covered by either an eschar or extensive necrotic tissue (tan, yellow-green, brown), which must be cleared away before the true depth can be determined.
© Logical Images 2010

- Partial-thickness pressure ulcers (stage 2) should heal within 60 days maximum;
- full-thickness pressure ulcers (stage 3/4/unstageable) should demonstrate improvement in overall ulcer status every 2 to 4 weeks

Risk assessment

- The most commonly used risk assessment tools are the **Norton** Scale and the **Braden** Scale for predicting pressure sore risk.

Assessment of Pressure Ulcer Healing

- **Bates- Jensen** Wound Assessment Tool (BWAT)
- NPUAP's Pressure Ulcer Scale for Healing tool
- **(PUSH)**

Stroke recovery

- The primary goals of stroke management are to reduce brain injury and promote maximum patient recovery.

- Once a patient is medically stable, the focus of their recovery shifts to rehabilitation

When can a stroke patient begin rehabilitation?

- Rehabilitative therapy typically **begins in the acute-care hospital** once the condition has stabilized,
- Often **within 48 hours** after the stroke.
- The first steps often involve promoting **independent movement** to overcome any paralysis or weakness

- A stroke victim may need to learn how to **sit up** and **move between the bed and a chair to standing and walking**, with or without assistance

- Some patients are transferred to **in-patient** rehabilitation programs,

- While others may be referred to **out-patient** services or home-based care

In patient

Interdisciplinary team :

- Physician,
- Nurse,
- Pharmacist,
- Physical therapist,
- Occupational therapist,
- Speech therapist,

- Primary goals of this sub-acute phase of recovery include preventing secondary health complications, minimizing impairments, and achieving functional goals that promote independence in activities of daily living

Activities of Daily Living

iStudentNurse.com/ADLs

Toileting

Bathing

Eating/Feeding

Dressing

Grooming

Transfering

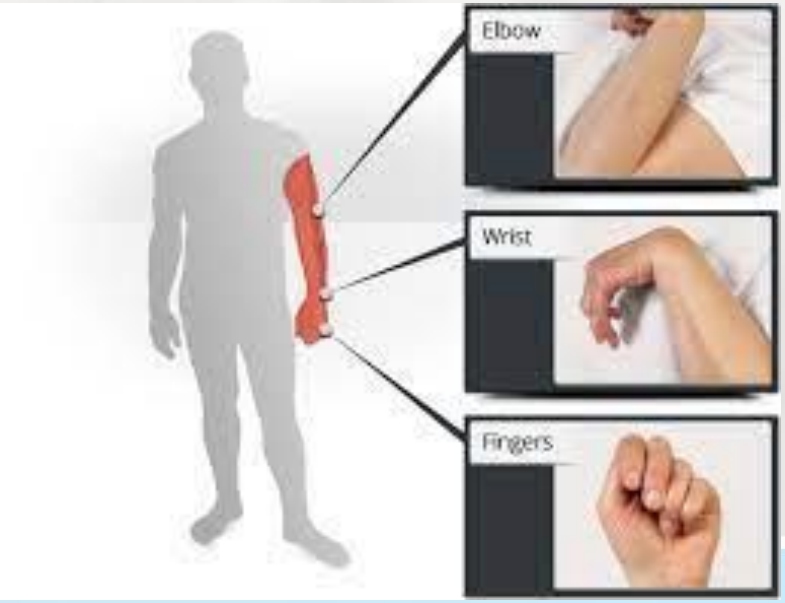
iStudentNurse

- Contractures after stroke are characterized by **stiff, tight muscles and joints**.
- It often occurs in the upper extremities and can lead to clenched hands after stroke. However, the condition is not limited to the hands.

Causes of Contractures After Stroke

- Contractures are a form of spasticity, a condition where muscles become stiff and tight after stroke.
- When **spasticity is left unmanaged**, contractures can develop.

- Spasticity is best understood as a **miscommunication** problem between the **brain and the muscles.**



Rehabilitation for Contractures After Stroke

- Stretching
- Range of Motion Exercises
- Massed Practice
- Passive Exercise
- Electric Stimulation
- Botox
- Rehab Technology
- Orthoses
- Surgery

سپاس از توجه شما