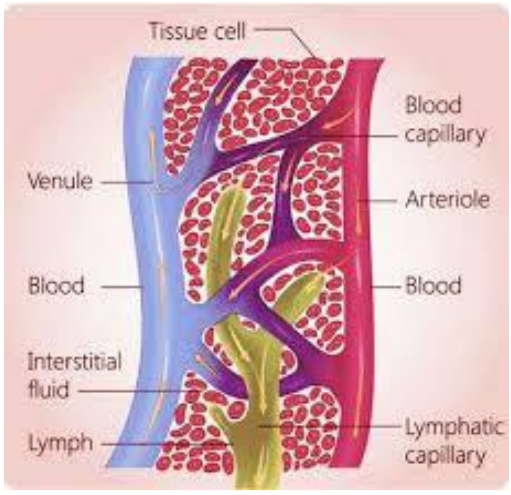

Physical Therapy for vascular and microcirculation

By

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Introduction

Physical therapy plays an important role with major peripheral vascular diseases related to metabolic disorders and provides different techniques in evaluation and management of such disorders.

This book promotes the reflective, critical, objective, and analytical practice of physical therapy applied to internal medicine health problems. All physical therapy students should possess strong foundational knowledge about vascular and internal medicine diseases and be able to apply this knowledge to a variety of patients.

Table of contents

Vascular anatomy of lower limb

Femoral artery: -

It is the main Arterial supply of the lower limb and branch of external iliac artery.

Location:-

Behind the inguinal ligament in the midway between Superior anterior iliac spine and the pubic symphysis.

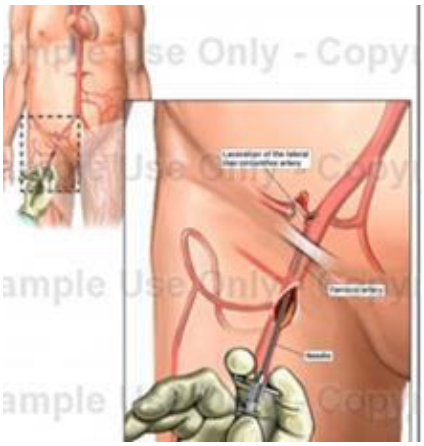
It ends at the opening of adductor Magnus as the popliteal artery.

Origin:

It is the continuation of the external iliac artery. It descends vertically towards the adductor tubercle. It terminates by passing through the adductor hiatus (in adductor magnus) and entering the popliteal space as the popliteal artery.

Branches of the femoral artery:

- superficial epigastric
- Superficial Circumflex iliac
- Superficial external pudendal
- Deep external pudendal .
- Profunda femoris — (Deep artery of thigh)



Cannulation of Femoral Artery:

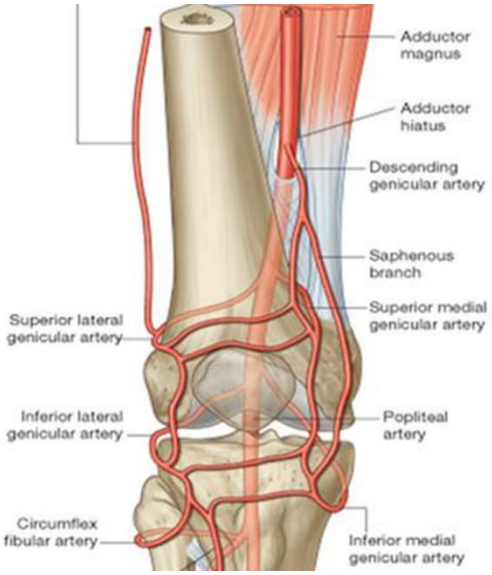
It is used for left cardiac angiography. A long catheter is inserted percutaneously into the artery and passed up the external iliac artery, common iliac artery and aorta to the left ventricle.

The profunda femoris artery

Supplies the medial compartment of the thigh. Arises from the lateral side of the femoral artery (4 cm below the inguinal ligament). Passes medially behind the femoral vessel

Anastomosis around knee

Genicular anastomosis is formed from the genicular arteries. It compensates for the narrowing of the popliteal artery during prolonged flexion of the knee.



Trochanteric Anastomosis

Formed from anastomosis of medial & lateral circumflex femoral arteries.

Its main function is to supply the head & neck of femur.

Cruciate anastomosis

It supplies blood to the lower limb in case of ligation of the femoral artery.

It is formed by the union of:

-superiorly: Inferior gluteal

-Inferiorly: First perforating

-transversely: Medial & Lateral circumflex femoral

-Provides connection between internal iliac and femoral arteries

Popliteal artery

It is the continuation of the femoral artery. It enters the popliteal fossa through an opening in the adductor magnus.

Relations and branches:

1-anterior:

A- Popliteal surface of the femur.

B- knee joint.

C- popliteus muscle.

2-posteriorly:

A- Tibial nerve.

B- Skin and fascia.

C- Popliteal vein.

3-Branches:

Muscular & Articular (five genicular branches to the articular capsule and ligaments of the knee joint).

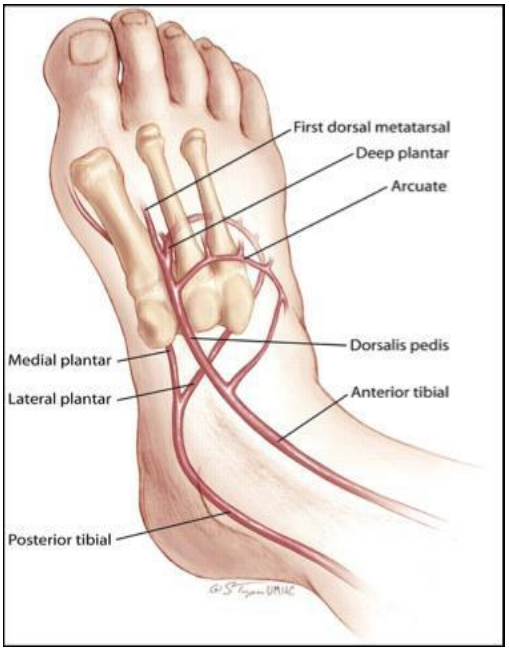
4-Termination:

It ends at the lower border of the popliteus muscle, it divided into two branches:

Anterior tibial artery: Small branch of the popliteal artery.

Posterior tibial artery: Large branch of the popliteal artery.

Anterior tibial artery:



It enters the anterior compartment of the leg through an opening on the upper part of the interosseous membrane (smaller than posterior tibial artery). It descends with the deep peroneal nerve. It's upper part is deep. It's lower part (in the front of the lower end of the tibia) is superficial.

Branches: muscular& anastomotic.

It supplies structures in the anterior compartment of the leg & dorsum of foot. It ends at the ankle joint midway between the malleoli where **it becomes the dorsalis pedis artery** (dorsal artery of the foot).

Posterior tibial artery:

It is the large branch of the popliteal artery. Provides the main blood supply to the posterior compartment of the leg & sole of the foot.

Above: lies on the posterior surface of the tibialis posterior.

Below: lies on the posterior surface of the tibia.

Its lower part is covered with skin and fascia.

Location :

Passes behind the medial malleolus. Deep in the flexor retinaculum.

Termination: it terminates when it divides into:

- **medial plantar artery**
- **lateral plantar artery**

Branches:

1- Peroneal (fibular) artery [a largest and most important branch that descends behind the fibula].

(The artery of lateral compartment of the leg) which gives:

A- Nutrient artery to the fibula.

B- Perforating branch (to lower part of front of the leg)

C- Shares in anastomosis around the ankle joint.

D- Muscular branches to the muscles of the lateral and posterior compartments of the leg.

2- Nutrients artery to the tibia [a largest nutrient artery of the body]

3- Anastomotic branches for anastomosis around the ankle joint

4- Medial and lateral plantar arteries.

5- Calcaneal arteries: supply the heel.

Plantar arteries

Medial plantar arteries:

Arises beneath the flexor retinaculum. The smaller terminal branch of the posterior tibial artery.

Branches: Muscular, Articular and Cutaneous.

Ends by supplying the medial side of the big toe. It supplies mainly the muscles of the great toe, and gives most of plantar digital arteries. Its superficial branch supplies the skin of the medial side of the sole.

Lateral plantar arteries:

The larger terminal branch.

Branches: Muscular, Articular & Cutaneous branches.

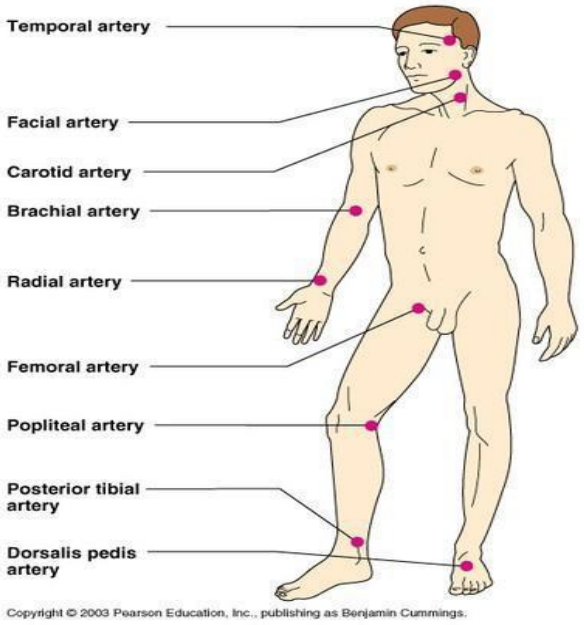
The plantar arch gives plantar digital arteries. At the base of the 5^{th} metatarsal bone, it curves medially to lateral plantar artery form the plantar arch which is completed by the medial plantar artery and branch from dorsalis pedis artery. Joins the dorsalis pedis artery at the proximal end of the 1^{st} intermetatarsal space. The arch supplies the skin, fascia and muscles in the sole and plantar digital arteries to the adjacent digits.

Where to feel the peripheral arterial pulse?

Femoral:

Inferior to the lingual ligament and midway between the anterior superior iliac spine and symphysis pubis.

How to stop blood flow in the femoral artery? By pressing the femoral artery directly posterior against the superior pubic ramus and the femoral head.



Popliteal:

Deep in the popliteal fossa medial to the midline (weakening or loss of the popliteal pulse is a sign of femoral artery obstruction).

Posterior tibial:

Posteroinferior to the medial malleolus in the groove between the malleolus and the heel(flexor retinaculum must be relaxed by inverting the foot) is essential for examining patients with occlusive peripheral arterial disease.

Dorsalis pedis:

Over the tarsal bones between the tendons of extensor hallucis longus and extensor digitorum ,Some people have congenitally non palpable A diminished or absent dorsalis pedis pulse usually suggests vascular insufficiency.

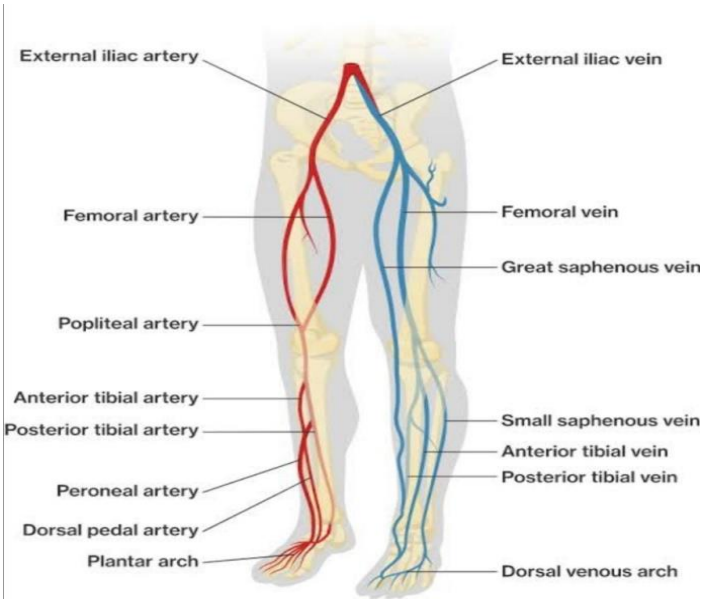
The venous system of the lower limbs:

The veins of the lower limb are classified into: Superficial (in the subcutaneous tissue) & deep (to the deep fascia and accompany all major arteries).

Superficial veins:

Dorsal venous arch (network) receives most of the blood of the foot through digital and communicating veins.

Drained on:



- Medial side by the great saphenous vein.
- Lateral side by the small saphenous vein

Deep veins of the lower limb:

The major deep veins of the lower extremities follow the course of the corresponding arteries. The deep venous system of the calf includes the anterior tibial, posterior tibial, and peroneal veins, Popliteal and femoral veins.

. In the calf, these deep veins present as pairs on both sides of the artery.

The posterior tibial vein

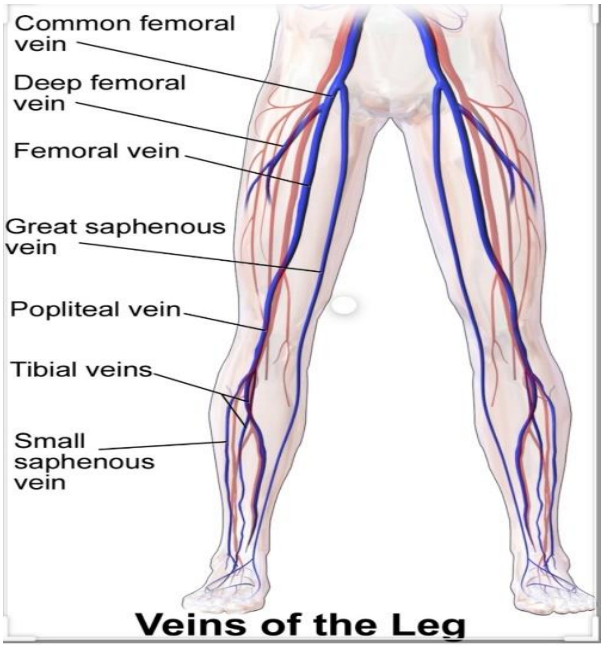
The posterior tibial vein receives blood from the medial and lateral plantar vein and drains the posterior compartment of the leg and plantar surface of the foot. This vein lies behind the tibia and joins the popliteal vein at the posterior knee.

The anterior tibial vein

The anterior tibial vein is the upward continuation of the dorsal pedal vein. It runs along the anterior compartment of the leg just above the interosseous membrane between the tibia and the fibula, and joins the posterior tibial vein to form the tibioperoneal trunk and popliteal vein.

The peroneal vein

The peroneal vein runs along the posteromedial aspect of the fibula and joins the posterior tibial vein



Popliteal vein: Formed by the union of venae comitantes around the anterior & posterior tibial veins. It ascends through popliteal fossa and pass through adductor canal and become femoral vein.

Femoral vein:

It enters the thigh by passing through the opening in the adductor magnus. It leaves the thigh in the intermediate compartment of the femoral sheath. Passes behind the inguinal ligament to become the **external iliac vein.**

Perforating veins:

Arise from the superficial veins and penetrate the deep fascia close to their origin. Connect the superficial veins (great saphenous vein) with the deep veins along the medial side of the calf. Their valves only allow blood to flow from the superficial to the deep veins.

Raynaud's disease

Raynaud's disease or phenomenon is episodic spasm of small arteries and arterioles.

Abnormal vasoconstrictor reflex exacerbated by exposure to cold or emotional stress; tips of fingers develop pallor, cyanosis, numbness and tingling. Typically, the fingers, and less commonly the toes, are involved. Rarely, the nose, ears, or lips are affected. Affects largely female.

Types of Raynaud's disease:

- **Primary Raynaud's** (Raynaud's disease), the cause is unknown.
- **Secondary Raynaud's** (Raynaud's Phenomenon) due to:
    - Connective tissue disorder, such as scleroderma or lupus,
    - Injuries to the hands.
    - Smoking and thyroid problems.
    - Birth control pills.
    - Diagnosis is typically based on the symptoms.

Risk factors for Secondary Raynaud's:

- Exposure to vibration from tools like rock drillers. Vibration from such tools may also damage nerves, muscles, joints of hands.
- Typists and pianists.
- Gripping hand tools too tightly.

Signs and symptoms:

When exposed to cold temperatures, the blood supply, is markedly reduced; the skin turns pale or white and becomes cold and numb. When the oxygen supply is depleted, the skin color turns blue (called cyanosis).

These events are episodic, and when the episode subsides or the area is warmed, the blood flow returns, and the skin color first turns red (rubor), and then back to normal, often accompanied by swelling, tingling, and a painful "pins and needles" sensation.

Physical therapy program

- Strengthening exercises
- Electrical stimulation
- Assistive devices
- Hydrotherapy

Strengthening exercises

- Hand grip
- Inverted hand Grip
- Resist A-Band
- Therapy putty
- Cando hand exercise web

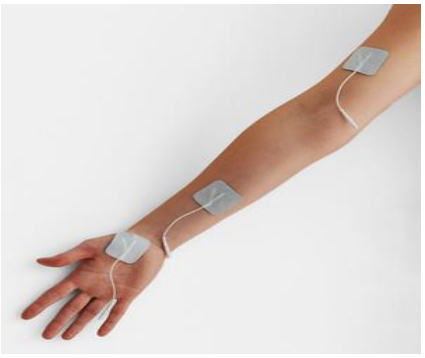
Range of motion and muscle power exercises.

Connective tissue manipulation: gentle massage of connective tissues can improve blood circulation to the affected area and decrease anxiety.

Superficial lymph drainage: is slow, delicate, rhythmical stretching of the tissues, which stimulates contraction of the lymphatic vessels, propels lymph through the collapsible superficial lymphatic system, increases local blood flow.

Hydrotherapy: Hydrotherapy in the form of a whirlpool or other pool therapy may also be helpful. It has been used to treat arthritis and joint injuries or replacements. It has also been used to relieve pain, support wound healing, and help with various neurological disorders. Physical training in warm water was well tolerated and seems to improve exercise capacity as well as muscle function in small muscle groups in older patients with chronic heart failure.

Electrical stimulation: Transcutaneous Electrical Nerve Stimulation (TENS): Pain relieving, stimulates the blood flow throughout the body, particularly in the treated area.



Life-style changes: Life-style changes including; staying warm, keeping the hands and fingers covered in cold weather, layered clothing, coats, and heavy socks.

Relaxation techniques to treat stress and anxiety.

Calcium channel blockers are the most effective treatment for Raynaud's phenomenon. Patients who do not tolerate or fail to respond to calcium channel blocker therapy can try other vasodilator drugs alone or in combination.

Chemical or surgical sympathectomy has been reported to be effective in relief of symptoms for very severe, unmanageable cases, however, results of this therapy may be short-lived.

Orthostatic hypotension

Definition:

- Orthostatic hypotension also referred to as postural hypotension is defined as a decrease in systolic blood pressure more than 20 mm Hg or a decrease in diastolic blood pressure more than 10 mm Hg within three minutes of standing when compared with blood pressure from the sitting or supine position.
- It results from an inadequate physiologic response to postural changes in blood pressure. Delayed vasoconstriction of lower limb blood vessels.
- When you change positions it causes a temporary decrease in blood flow and not enough oxygen to your brain. Usually your heart rate increases when you change positions so your body gets enough blood flow to prevent symptoms of orthostatic hypotension. Orthostatic hypotension occurs when something

  Interrupts body's natural process of counteracting low blood pressure (Baroreceptors reflex). Postprandial hypotension is believed to be caused by the autonomic nervous system not compensating appropriately, because of aging or a specific disorder.

Common symptoms:

- Dizziness
- lightheadedness
- blurred vision
- Weakness
- Fatigue
- nausea
- palpitations
- headache

Causes:

Inadequate intravascular volume, autonomic nervous system dysfunction, decreased

venous return, or inability to increase cardiac output in response to postural changes. Decreased cerebral perfusion produces the neurologic symptoms of orthostatic hypotension.

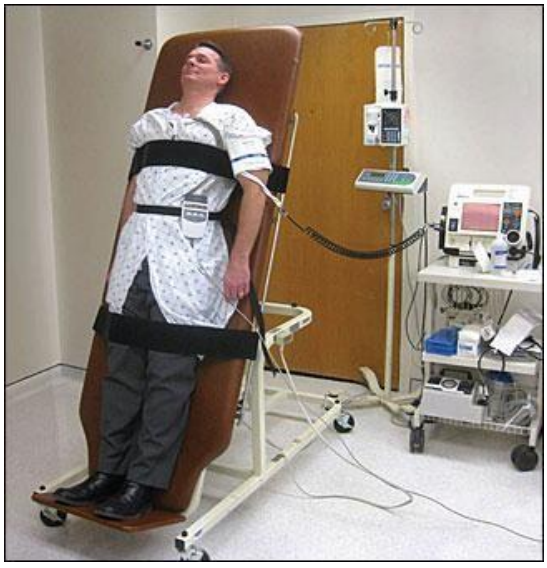
- ☐ dehydration
- ☐ blood loss
- ☐ Neurological disorder
- ☐ cardiovascular
- ☐ endocrine systems
- ☐ several classes of medications
- ☐ older patients

Evaluation:

Identifying reversible causes and underlying associated medical conditions.

1- **Blood pressure monitoring.** The diagnosis of OH can be made by measuring heart rate and BP supine (or seated) and after 1 and 3 minutes of standing Confirmation of diagnosis if patient has a drop of 20 mm Hg in systolic blood pressure or a drop of 10 mmHg in diastolic blood pressure and increasing HR, or if standing causes signs and symptoms.

2-Head-Up Tilt-Table Testing:



Perform tilt-table testing in a quiet room; the patient should rest while supine for five minutes before testing is started. Heart rate should be measured continuously and an automated device should measure blood pressure at regular intervals.

The table should be slowly elevated to an angle between 60 to 80 degrees for three minutes.

The test is considered positive if systolic blood pressure falls 20 mm Hg below baseline or if diastolic blood pressure falls 10 mm Hg below baseline.

If symptoms occur during testing, the patient should be returned to the supine position immediately.

Physical therapy treatment:

- ☐ Ensure adequate hydration, Older patients should consume a minimum of 1.25 to 2.50 L of fluid per day.
- ☐ Sodium may be supplemented by adding extra salt to food or taking 0.5- to 1.0-g salt tablets.
- ☐ Lower-extremity and abdominal binders used to decrease venous pooling on standing. It is best to use thigh- or waist-high stockings that produce at least 15- to 20-mm Hg pressure.

- ☐ Exercise prescription to improving cardiac output on standing:
  - moving from the supine to standing position in gradual stages(including continuous changing of the position throughout the day e.g. :half lying position using pillows ,semi sitting and sitting at the edge of the bed)
  - Breathing Exercises(that improve venous return so cardiac output increased)

- Isometric (tensing the leg muscles), lower-extremity exercise in the bed as ankle pump
- Squatting.
- Water exercises are particularly helpful because of the improvement in venous return produced by the pressure of the water.
- Reclining exercises (recumbent biking or rowing) are preferable to upright ones (treadmill).

☐ Education:

- Recognize symptoms that indicate your standing blood pressure is falling.
- Recognize the conditions that lower blood pressure, such as a heavy meal, positional changes, heat, drugs, or a hot bath.
- Learn the things you can do to raise your blood pressure.

Hypertension

Definition

Hypertension (HTN) is a persistent elevation of arterial blood pressureabove 140 mm Hg systolic or 90 mm Hg diastolic.

It is the most common cardiovascular condition in the world. The prevalence of HTN in Egypt is 26.3% among adult population. Its incidence increases with aging, around 50% of Egyptians above 60 years have hypertension.

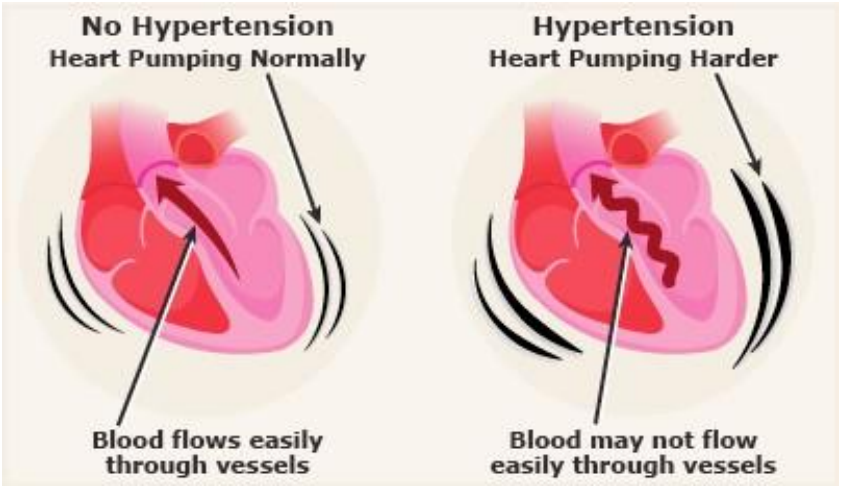
Classification of Blood Pressure in Adults

Classification	Systolic (mm Hg)	Diastolic (mm Hg)
Normal	<120	<80
Prehypertension	120–139	80–89
Stage 1 hypertension	140–159	90–99
Stage 2 hypertension	≥160	≥100

*Classification is based on the average of two or more readings on two or more occasions.

BP is determined by cardiac output and total peripheral resistance, or systemic vascular resistance, each of which are affected by a number of other factors. Cardiac output is affected by heart rate and stroke volume (SV). Total peripheral resistance is affected by the caliber of the arteriolar bed (i.e. peripheral vascular resistance), the viscosity of the blood, and the elasticity of the arterial walls.

BP rises when cardiac output, total peripheral resistance, or both are increased.



HTN develops when an imbalance occurs among the various systems that regulate BP: the sympathetic nervous system, the renin–angiotensin–aldosterone system, vasopressin, nitric oxide, and a number of vasoactive peptides (e.g., endothelin and adrenomedullin).

Regardless of cause, without effective treatment and control, HTN begets more HTN due to arteriolar remodeling, which leads to target organ damage (e.g., retinopathy, left ventricular hypertrophy [LVH], renal insufficiency, and encephalopathy), organ failure, and premature death. In addition, patients with HTN are more likely to be obese and to have insulin resistance, type 2 diabetes mellitus (DM), and dyslipidemia (high triglycerides and low levels of high-density lipoprotein cholesterol).

Types of HTN:

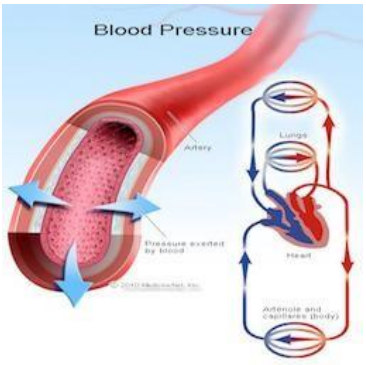
- Primary, or essential, HTN is diagnosed when no known cause can be identified (about 90% to 95% of cases).
- Secondary HTN can be directly attributed to a specific cause, such as renal, endocrine, vascular, and neurologic disorders and various drugs and toxins.
- Labile HTN occurs when BP is sometimes elevated and other times normal.
- White-coat HTN, which is defined as elevated BP that occurs in the clinic but not during normal daily life.

- Masked HTN exists when an individual has a normal clinic BP but high ambulatory BPs, which may occur in up to 10% of the general population and is associated with an increased rate of target organ damage and increased mortality.
- Malignant HTN involves markedly elevated BP (usually >160/110 mm Hg) causing retinal hemorrhages, exudates, or papilledema.

Risk factors:

Include age, ethnicity, obesity, glucose intolerance, smoking, stress, excess sodium or alcohol intake, decreased intake of potassium, calcium, and magnesium, and lack of exercise.

Pathophysiology:



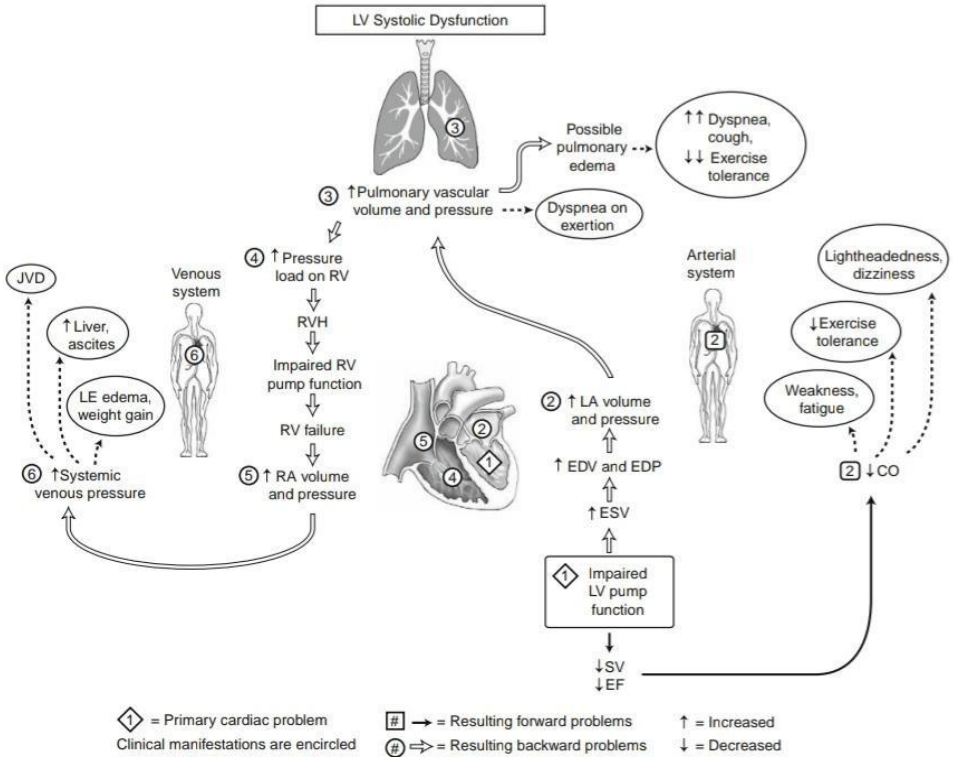
Alterations in endothelial and arteriolar structure, mechanical properties, and function lead to HTN, which creates a pressure load on the left ventricle (LV) followed by compensatory LVH. In addition, stiffening of the large systemic arterieswith age increases LV afterload and widens the pulse pressure, leading to greater LVH and eventual heart failure.

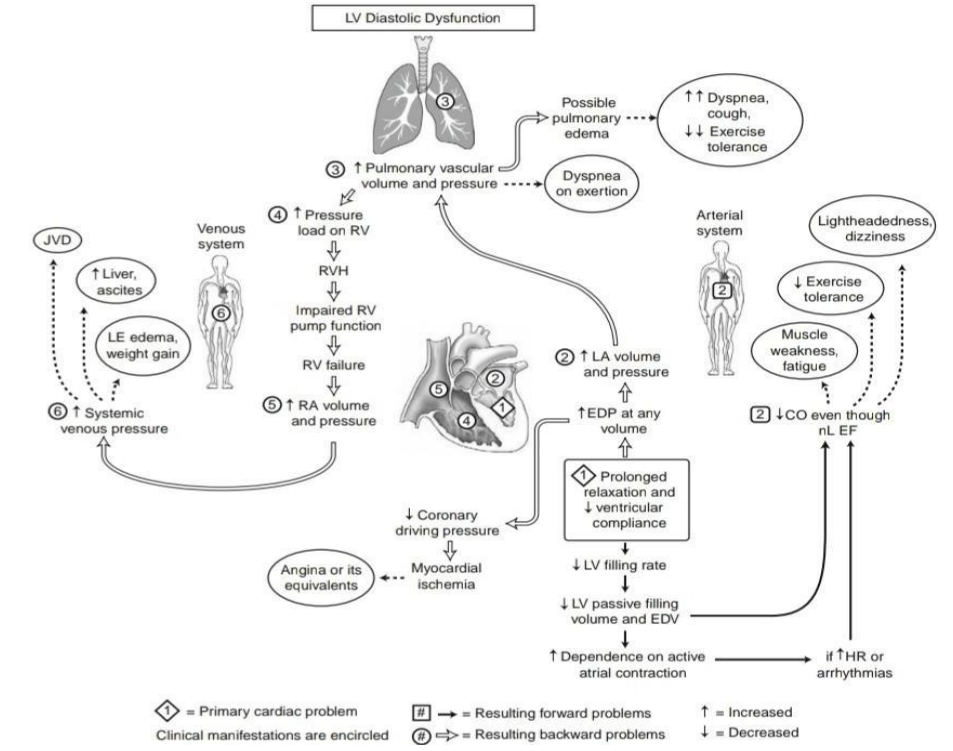
- Initially, normal LV systolic function is maintained by the hypertrophied LV, but diastolic dysfunction develops early in the disease process.
  - LVH and associated myocardial fibrosis results in prolonged relaxation time as well as a stiffer (less compliant) LV, both of which produce higher LV end-diastolic, left atrial, and pulmonary venous pressures, which leads to pulmonary congestion.
  - To achieve adequate filling, the stiffer LV becomes more dependent on active atrial contraction and is adversely affected by tachycardia and

arrhythmias where active atrial contraction is absent (e.g., atrial fibrillation)

- When filling volume is inadequate, SV is reduced and symptoms of pulmonary congestion and inadequate cardiac output may develop (i.e., diastolic heart failure)
- Higher filling pressures inhibit coronary flow and reduce coronary reserve, increasing the risk of myocardial ischemia and arrhythmias.

- As HTN becomes more severe and/or prolonged systolic dysfunction, defined as an LV ejection fraction (EF) of 40% to 50%, develops.
  - LVH, although initially adaptive and desirable, has long-term deleterious effects on cardiac energy balance and contractile function. Ventricular remodeling, and LV systolic function becomes impaired with resultant decrease in SV and increases in end-systolic and end- diastolic volumes (ESV and EDV, respectively).
  - In the presence of LVH with its reduced compliance, this increase in EDV causes a rise in end-diastolic pressure (EDP), which is reflected back to the LA and pulmonary vessels, resulting in pulmonary edema if the pressure rises high enough to produce transudation of fluid from the capillaries into the interstitial spaces.
  - Initially systolic dysfunction is manifested as reduced LV functional reserve during exercise, but later symptoms can develop even at rest (i.e., CHF). However, systolic dysfunction is asymptomatic in up to onehalf of patients.
- HTN is associated with an increased incidence of all-cause and CVD mortality, stroke, coronary artery disease, peripheral arterial disease, and renal insufficiency. Both increased systolic blood pressure (SBP) and pulse pressure (the difference between SBP and diastolic blood pressure [DBP])if $\geq$ 60 mm Hg are strong predictors of CVD risk.


LV Systolic Dysfunction
Possible pulmonary edema
↑↑ Dyspnea, cough, ↓↓ Exercise tolerance
③ ↑Pulmonary vascular volume and pressure
Dyspnea on exertion
④ ↑ Pressure load on RV
RVH
Impaired RV pump function
RV failure
⑤ ↑ RA volume and pressure
JVD
Venous system
↑ Liver, ascites
LE edema, weight gain
⑥ ↑Systemic venous pressure
② ↑ LA volume and pressure
↑ EDV and EDP
↑ ESV
① Impaired LV pump function
↓SV
↓EF
Arterial system
Lightheadedness, dizziness
↓Exercise tolerance
Weakness, fatigue
② ↓CO
① = Primary cardiac problem
Clinical manifestations are encircled
→ = Resulting forward problems
(#) ⇨ = Resulting backward problems
↑ = Increased
↓ = Decreased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② ↑ LA volume and pressure
↑EDP at any volume
① Prolonged relaxation and ↓ ventricular compliance
↓ Coronary driving pressure
Myocardial ischemia
Angina or its equivalents
↓ LV filling rate
↓ LV passive filling volume and EDV
↑ Dependence on active atrial contraction
if ↑HR or arrhythmias
Arterial system
Lightheadedness, dizziness
↓ Exercise tolerance
Muscle weakness, fatigue
② ↓CO even though nL EF
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Clinical manifestations

HTN is generally asymptomatic until complications develop in target organs, resulting in:

- Cerebral vascular accident (cerebral embolism or hemorrhage)
- Hypertensive heart disease
- Atherosclerotic heart disease
- Renal insufficiency or failure, nephrosclerosis
- Aortic aneurysm, simple or dissecting
- Peripheral vascular disease
- Retinopathy

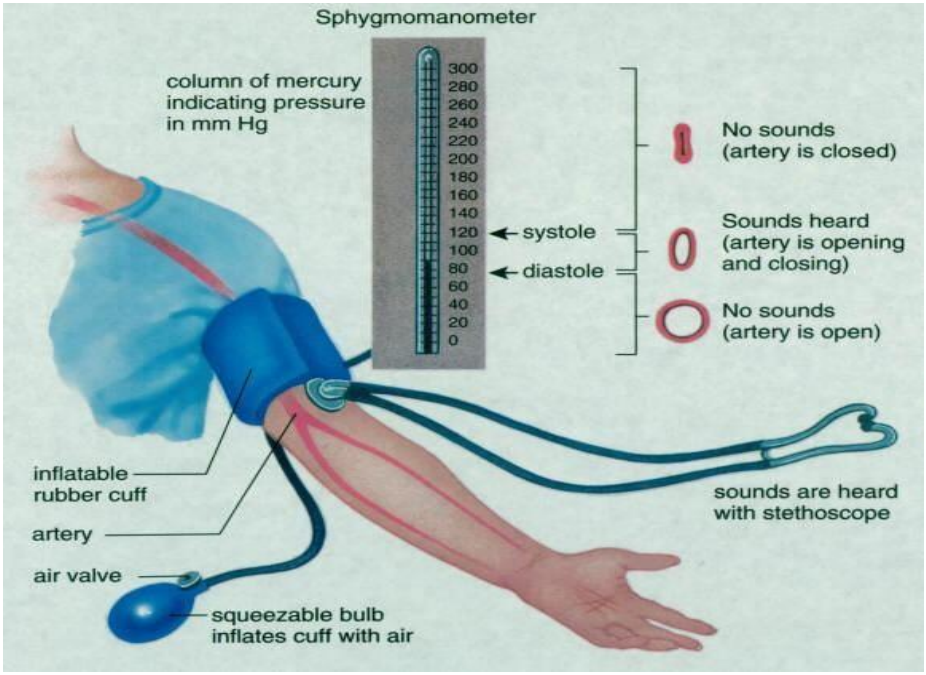
The clinical manifestations of hypertensive heart disease include the following:

- Exertional dyspnea
- Fatigue
- Impaired exercise tolerance
- Increased symptoms with tachycardia and loss of active atrial contraction
- Exertional chest discomfort
- Signs and symptoms of heart failure.

Accurate blood pressure measurement:

- The equipment should be regularly inspected and validated.
- The operator should be trained.
- The patient must be properly prepared and positioned and seated quietly for at least 5 minutes in a chair.
- The auscultatory method should be used.
- Caffeine, exercise, and smoking should be avoided for at least 30 minutes before BP measurement.
- An appropriately sized cuff should be used.

- The device should be on the same level of the heart.
- At least two measurements should be made and the average recorded.



Treatment:

Successful treatment of HTN usually requires two or more medications along with lifestyle modifications. For most patients the goal is to achieve a BP lessthan 140/90 mm Hg; however, lower levels (<125–130/75–85 mm Hg) are recommended for patients with comorbidities, such as diabetes mellitus, renaldisease, and possibly heart failure. Lowering SBP by 20 mm Hg reduces cardiovascular risk by half.

- **Pharmacologic therapy:**
  - Diuretics
  - ß-Blockers
  - α-Blockers
  - Calcium channel blockers
  - Angiotensin-converting enzyme inhibitors

- Angiotensin receptor blockers

- **Lifestyle modifications:**
  - Weight reduction (SBP and DBP decrease 1 mm Hg for every kilogram of weight loss)
  - Avoidance of tobacco
  - Diet modifications (e.g., low salt, higher potassium and magnesium, high in fruits and vegetables and nonfat dairy products, moderation of alcoholintake)
  - Physical activity
  - Stress management
  - Complementary/alternative medicine, including garlic, autogenic training, biofeedback, and yoga

- **Physical therapy:**

Benefits of exercise training:

- Reduced visceral fat, body weight and BMI.
- Alter renal function to increase elimination of sodium leading to reduce fluid volume.
- Lower cardiac output and peripheral vascular resistance at rest and submaximal exercise, Decrease HR, Decrease sympathetic and increase parasympathetic tone, Lower blood catecholamines and plasma renin activity
- After a bout of dynamic exercise, there is an immediate reduction in BP (i.e., post exercise hypotension, PEH), typically lasts for 4 to 10 hours and sometimes up to 22 hours after exercise cessation, with more prolonged duration induced by consecutive sessions.
- Aerobic exercise training decreases resting BP, with reductions averaging 7 to 11 mm Hg for SBP and 6 to 8 mm Hg for DBP, due to a reduction in systemic vascular resistance.

Precautions and contraindications to exercise:

- Monitoring of blood pressure before, during and after the exercise session.
- If a patient has a resting SBP greater than 200 mm Hg or DBP greater than 110, medical clearance should be obtained before initiating physical therapy or performing any exercise.
- If the patient has a history of retinopathy, renal failure, or LVH, BP must be controlled at rest (<125–130/75–85) and during exercise (≤ 10-mm Hg rise in SBP during dynamic exercise and ≤ 10-mm Hg rise in DBP) to avoid increased morbidity.
- Higher HRs or arrhythmias lacking active atrial contraction may increase symptoms caused by diastolic dysfunction.
- Most antihypertensive drugs are associated with orthostatic and post exercise hypotension, so caution is required when making quick changes to upright positions and an extended cool down is recommended after exercise. The application of heat modalities can also induce hypotension.
- Diuretic therapy may result in dehydration and electrolyte imbalance, including hypokalemia or hyperkalemia, both of which can triggerventricular arrhythmias, which can be serious and life-threatening. Hypokalemia can also cause muscle weakness, fatigue, and cramps.
- Antihypertensive medications affect HR and myocardial contractility, especially during exercise, blunting HR and BP responses. Other common side effects that can limit tolerance for exercise are muscle weakness, fatigue, and cramps, and sometimes bronchospasm.
- Breath holding and the Valsalva maneuver should be avoided because of the BP elevations they induce; instead, coordination of breathing with effort should be encouraged.
- Because many individuals with HTN are overweight or obese, exercise programs should also be designed to promote weight loss.

Exercise prescription:

- **Intensity:** moderate-intensity exercise (40% to 70% of VO2 reserve, i.e., the difference between resting oxygen consumption[VO2] and maximal oxygen consumption [VO2 max]) is effective inachieving antihypertensive benefit, minimize the risk of CV and musculoskeletal complications, and improve adherence.
- **Frequency:** between 3 and 5 days/week; however, because of post exercise hypotension, daily or near daily exercise is recommended,
- **Duration:** It is recommended to be 30 to 60 minutes of continuous or intermittent exercise per day (numerous bouts of moderate intensity exercise of at least 10 minutes in duration accumulated throughout the day to total 30 to 60 minutes of exercise).
- **Mode:** Aerobic exercise. Resistance exercise of moderate intensity has also been shown to produce small reductions in resting SBP and DBP in adults without causing adverse effects.

- Training HR ranges: The exercise intensity that has traditionally been recommended is 65% to 80% of HRmax, which corresponds to 50% to 70% of maximal oxygen consumption (VO2 max). HRmax is determined either by performance of a maximal exercise stress test orby calculation based on age (HRmax = 220 - age).
- Another method of prescribing exercise intensity uses a percentage of the heart rate reserve (HRR), which takes into account the resting HR and more accurately relates to oxygen consumption. This calculation, called **Karvonen's formula** (Target HR = (MHR – RHR)× (%) + RHR).Using this method for training, 60% to 80% of the HRR is equivalent to 60% to 80% of VO2 max.

  Unless the client is taking medications that affect HR either resting or exercising heart rates (e.g., ß-blockers) a rating of perceived exertion (RPE) scale can be used to guide exercise intensity.

Rating of Perceived Exertion:

Rating of perceived exertion (RPE) uses a numerical scale in relation to subjective perception of exertion, taking in to account personal fitness level, environmental conditions, and general fatigue levels, to provide an estimate of exercise intensity. Although ratings can be influenced by psychological factors, mood states, environmental conditions, exercise modes, and age, when used correctly they correlate well with exercise HRs and work rates. The RPE Scale uses numerical ratings of 6 to 20, with the following correlations:

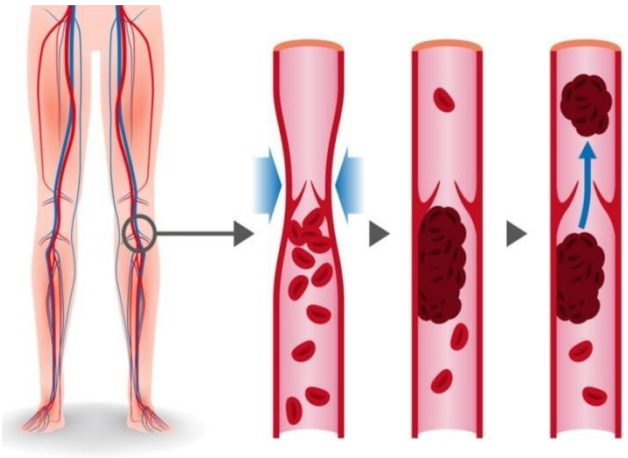
- No exertion at all is rated 6
- Extremely light exertion is rated 7
- Very light exertion is rated 9
- Light exertion is rated 11
- Somewhat hard exertion is rated 13
- Hard or heavy exertion is rated 15
- Very hard exertion is rated 17
- Extremely hard exertion is rated 19
- Maximal exertion is rated 20

Deep vein thrombosis

Definition:

Deep vein thrombosis, commonly referred to as ‑DVT,‖ occurs when a blood clot or thrombus, develops in the ‑deep veins‖ of the legs, thighs, and pelvis.

Thrombus is composed predominantly of fibrin and red cells. DVT in the lower limb can be classified as proximal, when the popliteal vein or thigh veins are involved or distal, when the calf veins are involved.



A clot that forms in the large, deep veins is more likely to break free and travel through the vein. It is then called an embolus. When an embolus travels from the legs or pelvic areas and lodges in a lung artery, the condition is known as a ‑pulmonary embolism,‖, a potentially fatal condition if not immediately diagnosed and treated.

If the clot breaks, loose and travels to the lungs, causes a pulmonary embolism. The symptoms of pulmonary embolism include:

- Shortness of breath
- Chest pain
- A feeling of apprehension
- Fainting
- Coughing
- Sweating
- Rapid pulse

- Bloody phlegm (coughing up blood)

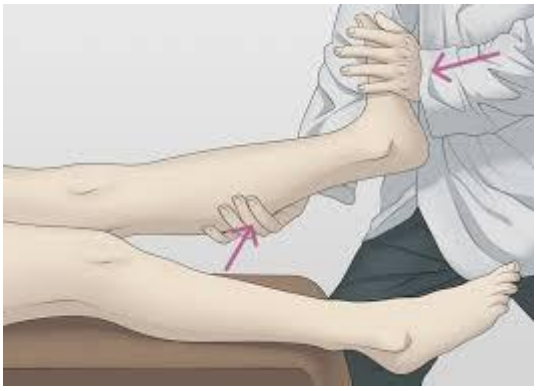
Classic symptoms of DVT include:

Lower extremity DVT can be symptomatic or asymptomatic. The symptoms may develop slowly or suddenly. Homans' sign may be demonstrable in DVT.

- Warmth of the skin
- Swelling of the limb
- Pain or tenderness
- Change in color (blue, red or very pale)
- Fullness of the veins just beneath the skin

Homans' sign:

1. The patient actively extend his knee.
2. Once the knee is extended, the examiner raises the patient's straight leg to 10 degrees, then passively dorsiflexes the foot and squeezes the calf with the other hand.



3. Deep calf pain and tenderness may indicate presence of DVT.

Causes:

Rudolph Virchow described three conditions that predispose to thrombus, the so-called Virchow's triad. This triad includes endothelial injury, stasis or turbulence of blood flow, and blood hypercoagulability. Stasis and endothelial injury are important in DVT following trauma or surgery while hypercoagulability is responsible for most cases of spontaneous DVT.

Risk factors for DVT:

1. Genetic: there are a number of genetic disorders that cause blood to clot more easily. These usually involve deficiencies in blood clotting factors such as protein C, protein S and factor V Leiden.
2. Acquired conditions – some medical conditions increase a person's risk of developing blood clots. These include:
    - prolonged immobility, stroke or paralysis
    - Pregnancy
    - Obesity
    - Smoking
    - Heart conditions (atrial fibrillation, mechanical heart valves)
    - Increased age
    - Previous episodes of DVT's
    - Cancer
    - Kidney problems
    - Blood disorders such as polycythemia vera
    - Medications – especially birth control pills and hormone replacement therapy
    - Surgery – surgical procedures, especially involving the hip, pelvis, or knee, increase the risk for clots to form. This risk continues through recovery from surgery as the person is less active.
    - Trauma – especially if blood vessels are injured.
    - Inactivity – prolonged sitting, especially sitting for six or morehours on a plane or in a car; or prolonged bed rest increase the risk for a clot to form.

Diagnosis:

If a patient's symptoms, history, and exam suggest the presence of a DVT; the most common test are:

- Venous ultrasonography of the leg, it is noninvasive, safe, available, and relatively inexpensive.
- D-dimer blood test
- MRI
- CT
- Contrast venography

A specific blood test to measure ‑D-dimer‖ which is a sign of recent clotting. When this test is negative, it is unlikely that DVT has occurred.

D-dimer is a degradation product of cross-linked fibrin that is formed immediately after thrombin-generated fibrin clots are degraded by plasmin.

Treatment of DVT:

The goal of therapy for DVT is to prevent the extension of thrombus, acute pulmonary empolism, recurrence of thrombosis, and the development of late complications such as pulmonary hypertension and post-thrombotic syndromes.

The primary treatment for blood clots is the use of anticoagulants (heparin). The anticoagulant helps reduce clots from forming. These medications have been referred to as ‑blood thinners‖; however they do not actually cause the blood to become less thick, only less likely to clot.

Other treatments can be used in specific situations and include filter placement (Vena cava filters).

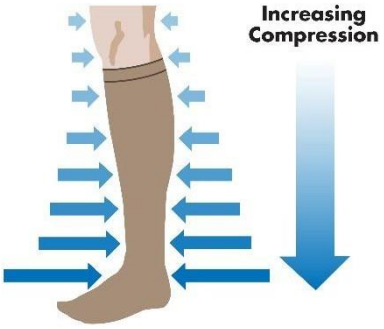
Prevention:

1. Mechanical methods of prophylaxis against DVT include:

- Intermittent pneumatic compression device: Intermittent pneumatic compression enhances blood flow in the

deep veins of the leg, preventing venous stasis and hence preventing venous thrombosis

- Graduated compression stocking. These stockings apply pressure to the lower legs with the greatest pressure at the ankle.



- Venous foot pump (ankle pump).

2. Surgery – at the time of surgery, certain high-risk patients may be given anticoagulants to decrease the risk of clot formation. This treatment isstarted shortly after the surgical procedure. It is used mostly in bone and joint surgeries and cancer surgeries.
3. Extended travel – since sitting for prolonged periods of time during travel increases the risk for clot formation, precautions to help prevent DVTinclude:
    - Stand up and walk around every hour or two when traveling in a plane or stop at rest stops and walk every couple of hours when traveling by car
    - Avoid smoking prior to travel
    - Elevate legs if you are sitting for moderate periods of time
    - Wear loose-fitting, comfortable clothing
    - Flex and extend the ankles and knees and change positions frequently. Avoid crossing the legs
    - Avoid dehydration by drinking plenty of fluids
    - Avoid medications (sleeping pills) or alcohol, which would impair the ability to get up and move around
    - Consider wearing knee-high compression stockings

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Student activities

Course title: P.T for Vascular and Microcirculation Diseases and its Surgeries. level : Third

Academic year: 2024/2025 - First semester

Student name: …….. ID #:……Lab section #:…….

1. **Attendance**

Lectures														
Practical sections														

2. **Quizzes :**

Quiz 1	**Quiz 2**	**Quiz 3**	**Quiz 4**	**Quiz 5**	**Quiz 6**	**Quiz 7**	**Quiz 8**	**Quiz 9**	**Quiz 10**
Student average mark									

2. Practical assignment:

……..

3. Teamwork research assignment

…….

Student total mark	

Lab section demonstrator **lecturer of the course**

…… **…….**