4.1.5

Disorders of Venous Circulation

Varicose Veins

Varicose veins are dilated veins found in the lower extremities. They can lead to chronic venous insufficiency where venous blood flow decreases and leads to pooling of the blood in the veins and subsequent edema, among other effects mentioned below. Varicose veins normally occur after the age of 50 and are more common in females. This predisposition in women is most likely due to the increased blood volume and stasis of blood flow experienced during pregnancy.

There are two subclasses of varicose veins:

1. **Primary varicose veins** are caused by some inherent weakness in the walls of veins that is most likely due to genetic mutations in the genes for connective tissue. They arise from anatomical anomalies such as malformations or vascular blemishes that can be present even at birth. This type of varicose vein tends to run in families.
2. **Secondary varicose veins** are varicose veins that arise from acquired injury or trauma to the veins. Anything that increases back pressure through a vein will tend to cause a progressive compromise of the venous valves and can make varicose veins more pronounced. Secondary varicose veins can be caused by damage associated with many different events:

1. Conditions that increase intra-abdominal pressure such as pregnancy and occupations that require frequent heavy lifting are associated with increased occurrence of varicose veins. This is because increased compression and pressure on vessels of the lower abdomen and pelvic region can cause backflow of blood to the lower extremities.
2. Prolonged standing where gravity causes blood to pool in the lower extremities and increases pressure on venous valves.
3. Obesity, which reduces the relative amount of fascia to support veins and can also increase intra-abdominal pressure.
4. Deep vein thrombosis (DVT) where a thrombus (or thrombi) blocks the vein and leads to increased back pressure on the valves.

Treatment of varicose veins focuses on improving venous flow and preventing tissue injury. Support stockings are commonly used and can be effective in compressing superficial veins and preventing distention. Sclerotherapy is another form of treatment used where a sclerosing chemical solution is injected directly into the vein and causes it to scar and shrink. This forces blood to reroute through healthier veins and the collapsed vein is reabsorbed and fades. Surgical treatment is also used to remove varicosities and the incompetent perforating veins but is only effective in patients with patent (open) deep venous channels that can sufficiently return blood to the heart in the absence of the excised veins.

Note: “spider veins” are different from varicose veins. They are smaller and generally not detrimental to overall health. They can occur simultaneously with varicose veins because of predisposition to develop them or because of a venous disease, but spider veins themselves do not develop into varicose veins.

Venous Thrombosis and DVT

Venous thrombosis refers to the presence of a thrombus in a vein and the subsequent inflammatory response in the vessel wall. This condition is also called **thrombophlebitis**. If the thrombus is located in a deep vein (deep inside the body, not on a superficial surface), then it is considered to be a **deep vein thrombosis (DVT)**. DVTs most often occur in the lower extremities and can be complicated by pulmonary embolism, recurrent DVT, and the development of chronic venous insufficiency.

There is a triad of risk factors associated with venous thrombosis. It includes stasis of blood, increased blood coagulation, and vessel wall injury. The greater the number of these risk factors that a person experiences, the greater the risk of developing a DVT.

1. **Stasis of blood** is associated with the lack of movement of an extremity or of the whole body. Patients who are immobilized by a hip fracture, joint replacement, or spinal cord injury are particularly prone to DVT.
2. **Increased blood coagulation** can be due to deficiencies in naturally occurring anti-clotting substances and inherited disorders such as factor V Leiden and prothrombin gene mutations. Pregnancy, the postpartum state, the use of oral contraceptives, and hormone replacement therapy after menopause all increase the circulating levels of clotting factors and therefore increase the risk of venous thrombosis. This is because increased levels of estrogen stimulate the liver to make more proteins, including clotting factors. Cancer cells also release substances that can activate the clotting cascade. Finally, dehydration concentrates clotting factors and can potentially increase their activation.
3. **Vessel wall injury** that often leads to DVT is damage to iliac and femoral veins that happens during hip replacement surgery. Smoking is another cause of vessel damage because it introduces toxic chemicals that cause chronic inflammation in the vessels. These chemicals cause endothelial cell dysfunction and death.

Many patients suffering from venous thrombosis are asymptomatic. This is most likely because the vein is not totally occluded. When symptoms are present, the most common manifestations of venous thrombosis are signs associated with inflammation such as pain, swelling, and deep muscle tenderness. Fever and elevated WBC count may also be present. Physical manifestations occur upstream of the DVT. The most common site of DVT formations is in the venous sinuses in the soleus muscle and posterior tibial and peroneal veins.

In an ideal world, venous thrombosis should be prevented rather than treated. Preventative measures include appropriate initiation of early ambulation after surgery or childbirth, exercising the legs, wearing support hose, limiting body positions that favor venous pooling, and use of pneumatic compression devices in individuals who are immobile. Although immobility can cause thrombus formation, for those that have DVT, bedrest is important until swelling has gone down to prevent embolization of thrombi.

It is very unlikely for a DVT to cause a stroke because the clot is found far away from the brain in a deep vein of the leg, pelvis, and sometimes the arm. If these clots break loose, they may travel to the small capillary beds of the lungs and cause a pulmonary embolism, but not to the brain. Arterial thrombi are responsible for many heart attacks and strokes, not venous thrombi.

Chronic Venous Insufficiency

Chronic venous insufficiency is a condition that presents in the lower extremities and is characterized by venous hypertension and venous blood flow insufficiency. Common causes of chronic venous insufficiency are reflux through incompetent veins (e.g. varicose veins), venous outflow obstruction (e.g. DVT or damaged vein), and impaired function of skeletal muscle pumps.

Net pressure in the veins of the legs can be determined by two factors: a hydrostatic component related to the weight of a column of blood below the level of the heart, and a hydrodynamic component related to the action of the skeletal muscle pump. Right-sided heart failure or blockage of large veins in the body cavities can increase the hydrostatic component as blood is unable to continue its unimpeded journey back to the heart. Immobility and prolonged sitting or

bedrest can increase the hydrodynamic component as the skeletal muscles are not contributing to the muscle pump movement of venous blood.

Venous insufficiency leads to tissue edema, congestion, and eventual impairment of tissue nutrition. Manifestations of venous insufficiency include edema, varicose veins, necrosis of subcutaneous fat, hemosiderin deposits from the breakdown of erythrocytes (induces browning of skin), and sclerosis of lymph vessels which further exacerbates the edema. With progression of the condition, thrombophlebitis (a clot that causes inflammation in vein walls) and venous ulcers appear. Common skin characteristics of stasis dermatitis are shiny, thin, bluish/brown, and irregularly pigmented skin that may have an ulceration. Minor injuries increase risk of developing relatively painless ulcers that are most often found at the lower part of the leg, specifically over the medial ankle. Common treatments for venous ulcers include compression therapy and skin grafting for large or slow-healing ulcers. Growth factors may also be administered.

Comparing a venous wound to a wound caused by arterial insufficiency (discussed in next section), we see that venous ulcers often weep and drain more due to the edema and increased venous blood pressure caused by chronic venous insufficiency. Arterial wounds are caused by a lack of arterial blood supply that reduces the hydrostatic pressure in the capillary bed and causes less fluid to leave. As a result, the wounds tend to be drier. Due to decreased blood flow, arterial wounds also present with hair loss and shiny skin in the affected area, not to mention they are quite painful. Venous wounds tend to be less painful than arterial wounds because they don't experience ischemia like arterial wounds do.

	VENOUS INSUFFICIENCY:	**ARTERIAL INSUFFICIENCY/PAD:**
DEFINITION:	Decreased ability to move venous blood back to heart, often due to venous valve dysfunction or DVT.	Obstruction/lack of arterial blood supply, often due to atherosclerosis.
EFFECTS:	Backup of blood leads to tissue edema, varicose veins, necrosis of SC fat, hemosiderin deposits, sclerosis of lymph vessels, thrombophlebitis, and ulcers.	Hydrostatic pressure is reduced in capillary, pain occurs due to tissue ischemia, claudication.
APPEARANCE OF SKIN/ WOUND	Shiny, thin, bluish/brown, irregularly pigmented skin. Less painful, wet ulcers that weep/drain more.	Hairless, shiny skin. Dry, painful ulcers due to tissue breakdown from insufficient blood proliferation.
COMMON WOUND LOCATION:	Lower parts of leg, often over medial ankle.	Most often in lower extremities.
TREATMENT:	Compression therapy, skin graft for large/slow healing ulcers, and growth factors.	Improved self-care like cessation of tobacco use, increased exercise, and healthy diet. Medication and surgery if needed.

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