The heart pumps blood to the aorta, which divides into arteries and is responsible for blood supply to various organs of the body. The primary artery in an organ is divided into smaller arterioles, which further divide into capillaries at the tissue level. As the capillary wall is only one-cell thick, rapid diffusion of gases and nutrients occur. Capillaries combine to form venules, which drain into larger veins. All veins of the body drain either into the superior or inferior vena cava.
Venous pressure refers to the average blood pressure in the venous compartment. **Central venous pressure (CVP)** is the blood pressure in the thoracic part of the vena cava near the heart; the pressure correlates with that in the right atrial chamber of the heart. CVP is measured to approximate the pressure in the right atrium and the amount of blood returning to the heart and is known as the venous return/preload of the right ventricle. The pressure can be determined using radiological measurements and is thus useful in clinical applications.

Walls of the veins have less smooth muscle compared to those of arteries. The *myogenic tone* in veins is therefore less. In addition, veins have more *collagen fibers* and less *elastic fibers* in their outer layers; therefore, they are more distensible compared to arteries. Veins typically have a larger diameter compared to arteries and offer less resistance to blood flow.

They serve as *reservoir vessels* owing to their increased capacitance. During the resting state, when there is a low demand for blood, the surplus blood is stored in the veins. Most capillary beds are closed and thus, more blood sweeps into the venous system.

When venous capacity decreases due to external or internal factors, venous pressure increases and causes an increase in venous return.
Vascular smooth muscle cells are tethered at one end to collagen filaments. At low filling pressures, the collagen filaments are folded. An increase in filling pressure causes the vein to expand and collagen filaments to unfold. Sympathetic stimulation causes smooth muscles to contract, thereby reducing venous capacity. Although collagen tends to be taut, it limits the extent to which the internal diameter of the vessel can be reduced.

**Regulation of Venous Pressure and Venous Return**

**Right atrial pressure**

As blood moves away from the heart, the blood pressure and resistance to blood flow decrease. The blood pressure in the veins is reduced to an average value of 17 mm Hg. Although this pressure is low, it is still greater than the right atrial pressure. Therefore, blood travels from a region of higher to lower pressure, i.e. from the veins into the right atrium.

**Sympathetic activity**

The smooth muscle layer of the veins is abundantly supplied with sympathetic nerve fibers. Sympathetic stimulation of vascular smooth muscle leads to vasconstriction. A decrease in the diameter of the veins causes a decrease in capacitance and an increase in venous pressure and peripheral resistance. This increases the pressure gradient and in turn, increases venous return to the heart.

**Cardiac output**
Sympathetic stimulation not only causes vasoconstriction but also increases the force of cardiac contractility and heart rate. As ventricles pump blood with greater force, the stroke volume increases and in turn decreases the pressure in the right atrium. Blood moves down the pressure gradient and venous return increases. This phenomenon is often referred to as the 'suction effect.'

A decrease in cardiac output increases atrial pressure. This causes the backflow of blood into the vena cava causing an increase in central venous pressure and a decrease in the pressure gradient.

**Blood volume**

An increase in the total blood volume during renal failure or during the activation of the renin-angiotensin-aldosterone system increases average venous pressure.

Central venous pressure (CVP) is measured by dividing the blood volume (ΔV) by compliance (Cv) of the thoracic veins and can be represented as follows:

\[
CVP = \frac{\Delta V}{Cv}
\]

An increase in blood volume increases CVP. However, if the thoracic veins are distensible, they will have more compliance, which can maintain CVP. However, large thoracic veins such as the vena cava do not undergo large changes in compliance; therefore, CVP almost always increases with an increase in blood volume.

**Skeletal muscle activity**

Many of the large veins of the lower limb lie between bulky skeletal muscles. Physical activity causes contraction of the calf muscles, which exerts external pressure on the veins, decreases capacitance, and increases venous pressure. Blood then moves down the concentration gradient, i.e. towards the heart.

The pressure caused by muscle contraction is responsible for the opening of venous valves in the lower limb, above the level of the muscles. The valves lying below the skeletal muscles remain closed due to low blood pressure in that region.

**The gastrocnemius** is a powerful calf muscle, which effectively increases venous return
during physical activity. It is therefore referred to as the ‘second heart.’

Venous valves

Large veins of the body are equipped with one-way valves at a distance of every 3–4 cm. When pressure is applied at the center of a vein, blood is displaced in both directions. However, due to the presence of valves, blood does not flow in both directions. The valve lying above the pressure point opens due to increased blood pressure, which leads to blood flow in one direction, i.e. towards the right atrium. The valve lying below the pressure point closes. The structure of a valve is such that pressure applied from above causes its leaflets to close.

Respiratory activity

During inspiration, the ribcage moves upwards and outwards while the diaphragm moves downward. This decreases intra-thoracic pressure and increases intra-abdominal pressure.

The portion of the vena cava lying above the diaphragm experiences low pressure from the outside, thus, venous blood pressure is also low. On the other hand, the sub-diaphragmatic portion of the vena cava experiences compression, which reduces venous compliance; consequently, venous pressure increases.

During this process, a pressure gradient is created, which allows blood to move towards the thorax. This mechanism, known as the ‘respiratory pump,’ facilitates venous return.

Gravity

During the sitting or supine positions, the effect of gravity on the column of blood in vessels is minimal. However, prolonged periods of standing or sitting cause pooling of blood in the veins of the lower extremities. Blood capacitance increases, which reduces central venous pressure and venous return.
To counteract the effect of low venous pressure and venous return, the sympathetic nervous system is activated, which causes vasoconstriction and increases venous return, as previously explained. This phenomenon is known as reflex venous vasoconstriction.

In the upright (orthostatic) position, calf muscles contract causing an increase in blood pressure in the specified venous column. This movement creates a pressure gradient, causing blood to return to the heart.

Clinical Applied Physiology

As discussed, CVP is an estimation of the blood in the right atrium. It may be raised in right-sided heart failure or volume overload.

Jugular venous pressure (JVP) is the clinical measurement of blood pressure in the internal jugular vein. It is raised in right heart failure. JVP is easier to measure compared to CVP.

Portal venous pressure refers to the pressure in the portal vein and can be measured using ultrasound guidance. It is raised in conditions like liver cirrhosis and Budd-Chiari syndrome. Increased pressure in the portal vein results in extravasation of fluid into the peritoneal cavity, a condition known as ascites.

References


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