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# PHYSIOLOGY OF VENOUS RETURN AND THE DEVELOPMENT OF VARICOSE VEINS

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Varicose veins are a common disorder affecting the venous system, most commonly affecting the superficial veins of the lower extremities. The development of varicose veins is closely related to disturbances in the physiological mechanisms that regulate venous return. Under normal conditions, venous blood from the lower limbs is transported toward the heart through the coordinated action of venous valves, the skeletal muscle pump, respiratory pressure changes, and pressure gradients within the venous system. Venous valves maintain unidirectional blood flow and prevent retrograde circulation, while contraction of the lower limbs skeletal muscles compresses deep veins and facilitates the upward movement of blood against gravity. When these mechanisms become impaired, venous reflux and blood pooling occur, leading to increased hydrostatic pressure within the venous system. Persistent venous hypertension contributes to dilation of the venous wall and the progressive development of varicose veins.

The aim of this study is to analyze the physiological mechanisms regulating venous return and to determine how disturbances in these mechanisms contribute to the development of varicose veins.

According to Raffetto and Khalil (2008), structural and functional changes in the venous wall contribute to the progressive dilation of veins and the development of varicosities. A study published in Phlebology (2023) further demonstrates that chronic venous hypertension leads to alterations in venous hemodynamics and promotes inflammatory and structural changes in the vessel wall. In addition to valve dysfunction, prolonged standing, reduced physical activity, aging, and genetic predisposition may increase venous pressure and aggravate venous insufficiency. Persistent elevation of hydrostatic pressure results in elongation and dilation of superficial veins, which are characteristic features of varicose veins.

Another important physiological mechanism involved in the development of varicose veins is the calf muscle pump and ambulatory venous pressure regulation. The calf muscles assist venous return from the lower extremities. During walking or muscle contraction, these muscles compress the deep veins, propelling blood toward the heart while venous valves prevent backward flow. When muscle pump efficiency is reduced, venous emptying becomes incomplete, and venous pressure in the lower limbs remains elevated during movement, a condition known as ambulatory venous hypertension. This contributes to venous stasis and promotes progressive dilation of superficial veins, thereby facilitating the development of varicose veins. (Barrett et al., 2019)

Chronic venous hypertension can cause endothelial dysfunction and alterations in the structure of the venous wall, leading to increased vascular permeability and activation of inflammatory pathways. These processes stimulate the release of enzymes such as matrix metalloproteinases, which degrade important structural components of the venous wall including collagen and elastin. As a result, the mechanical strength and elasticity of the veins decrease, making them more susceptible to dilation and valve separation. (Urbonavicius et al., 2023)

In conclusion, disturbances in the physiological mechanisms regulating venous return, particularly venous valve incompetence and increased hydrostatic pressure in the lower limbs, play a crucial role in the development of varicose veins. Understanding these physiological processes provides important insights into the pathogenesis of chronic venous disease and may contribute to improved preventive and therapeutic approaches.