Vascular Buffer System of the Scrotal Organs

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Natural arterial and venous anastomoses between three main vessels of the scrotal organs and spermatic cord serve as the anatomic basis for the formation of a complex natural vasculogenic stabilizing mechanism for the protection of the testicle, epididymis, and vas deferens from the negative exo- and endogenous factors -- the vascular buffer system of the scrotal organs. Its functioning is based on the unity of three hemodynamic components: antiischemic (redistribution and reduction of arterial blood deficiency), antihyperemic (adequate temporary deposition of arterial and venous blood), and hypotensive (reduction of critical pressure of the arterial and venous blood) mechanisms.

Key Words: *vascular buffer system of scrotal organs; hemodynamic mechanisms; intersystem fusion of testicular arteries; intersystem venous communicants of scrotal organs*

The anatomy of arterial and venous systems of the scrotal organs and spermatic cord was studied not once [1-4]. However, many problems concerning the specific features of functional mechanisms of the testicular, epididymal, and vas deferens (VD) vascular system in health and disease, pathogenesis of vasculogenic disorders of fertility in men, essential for modern practical and preventive andrology in order to develop new technologies for the treatment of sterility and improve measures for prevention of generative dysfunction, remain unsolved.

The data on the functional mechanisms of the testicle and spermatic cord vascular systems are scanty; these problems are discussed only in clinical studies of, mainly, varicocele [6,9]. Unfortunately, in fact no basic studies in this sphere were carried out.

We studied the structure and function of the natural vasculogenic stabilizing system, protecting the male generative function from negative exo- and endogenous factors.

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MATERIALS AND METHODS

The anatomy and microanatomy of the arterial and venous systems of the scrotal and spermatic cord organs were studied on 122 anatomical cadaveric complexes including the testicle and epididymis with membranes and VD with membranes. The arterial and venous network was stained by injection of stain; anatomical and microanatomical preparation of the vessels and their anastomoses with subsequent glass printing and X-ray contrast angiography were carried out.

The function of the arterial and venous systems of the scrotal organs and spermatic cord in health were studied on 30 anatomical complexes. Dyscirculatory conditions of the scrotal and spermatic cord vascular system were simulated, including arterial testicular ischemia, arterial testicular hyperemia, venous testicular hyperemia, and combined venous testicular cremasteric hyperemia. Sixty anatomic experiments using functional chromovasography and X-ray contrast angiography were carried out.

RESULTS

Comprehensive anatomic, microanatomic, and anatomo-experimental studies of the vascular system

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of the scrotal organs and spermatic cord in health and disease showed an intersystem type of blood supply to the testis, epididymis, and VD from the basins of the testicular, cremasteric, and VD arteries, realized through an anastomosis of intricate anatomical structure, intersystem fusion of the testicular arteries (IFTA) [1,3]. The testicle, epididymis, and VD have an intersystem collateral venous outflow via the testicular, cremasteric, and VD veins, which is realized on the basis of intersystem venous communicants (IVC) of two levels, IVC-1 (testicular venous node) and IVC-2, including the extraorgan veins of the epididymis and intersystem collateral veins, forming three hemodynamic variants of blood outflow from the epididymis and representing the anatomical basis of collateral flow from the scrotal organs [2,3]. Disorders in blood supply via the testicular artery (arterial testicular ischemia) lead to the formation of pathological compensatory circulation, consisting in blood transfer from the VD and cremasteric artery basins through IFTA to the testicle. Arterial testicular hyperemia (hypervascularization) leads to retrograde shunting of arterial blood from the testicular artery basin to the VD and cremasteric artery basins through IFTA. Venous testicular block and, more so, combined venous testicular-cremasteric block lead to severe disorders in local circulation, consisting in functional overloading of intact veins, including the intersystem venous communicants of two levels, and to the development of secondary arterial ischemia of the testicle and epididymis because of interarterial blood shunting from the testicular artery. The degree of changes in intraorgan testicular perfusion by stained solutions and X-ray contrast agent depended on the type of local circulation disorders — from devastation of the testicular parenchymatous vessels in arterial testicu-lar ischemia to plethora, extravasation (infarction) in arterial testicular hyperemia and venous testicular and combined testicular-cremasteric blocks.

We hypothesized a universal physiological mechanism, called "vascular buffer system of the scrotal organs" (VBSSO). It is a physiological local hemocirculatory mechanism for protection of the scrotal and reproductive organs from unfavorable exo- and endogenous factors (physical, iatrogenic, *etc.*), causing acute or chronic disorders in local circulation; the mechanism is realized on the basis of intersystem arterial and venous physiological anastomoses characterized by an intricate constant structure (Fig. 1).

The functioning of VBSSO is based on the unity of three hemodynamic mechanisms for local circulation stabilization: antiischemic (redistribution and reduction of arterial blood deficit), antihyperemic (adequate temporary deposition of arterial and venous blood), and hypotensive (reduction of critical pressure of arterial and venous blood).

The antiischemic VBSSO mechanism is realized in acute or chronic conditions associated with disorders in the testicular and epididymal trophics. Acute circulatory disorders can result from iatrogenic (surgical ligature of the testicular artery, its compression in plastic repair of the inguinal channel after herniotomy), urgent (testicular artery embolism, spermatic cord torsion), and other causes. Among the probable causes of testicular and epididymal ischemia are, for example, atherosclerotic involvement of the testicular artery or its compression by cicatricial tissues in the inguinal channel.

The antiischemic mechanism of VBSSO is realized only at the level of the arterial system of the scrotal organs mainly due to the natural anastomosis (intersystem fusion of testicular arteries). The arterial system provides the arterial collateral bloodflow and redistributes the blood from the cremasteric and VD artery basins in favor of the testicular basin in order to reduce the acute oxygen starvation of the testicular and epididymal tissues and hence, maintain the fertile function. The "upper" anastomoses between the two arteries of the scrotal organs, presented in anatomical variants, are extremely rare and their significance for realization of the VBSSO antiischemic mechanism is in fact negligible.

We consider that arterial hyperemia (hypervascularization) of the scrotal organs most often develops as a result of exposure to professional (industrial) factors. These dyshemocirculatory states emerge during exposure to hypergravitation and high temperature as a result of arterial wall atonia, during prolonged exposure to magnetic fields of high and medium intensity. The causes of venous hyperemia (most often of iatrogenic origin) are surgical ligation of the main venous collectors of the scrotal organs — the most prevalent method for surgical treatment of varicocele.

One of venous hyperemias is the testicular venous plethora. The testicular venous collector naturally plays the leading role in the maintenance of blood outflow from the scrotal organs.

Some interventions (for example, surgical treatment of varicocele by resection of the varicose testicular vein) lead to disorders in the anatomic integrity of the cremasteric vein (V. V. Yakovenko, Bergman's operations). Hence, it is essential for clinicians to know how blood is deposited and flows from the scrotal organs under conditions of testicular or cremasteric hyperemia.

The greatest negative consequences for fertility develop in arterial hyperemia caused by plethora in the testicular artery (the main vessel feeding the sexual gland and the epididymis).

The arterial testicular antihyperemic mechanism of VBSSO is realized at the levels of arterial system of the scrotum, intraorgan vascular network of the testicle, epididymis, and venous system of the scrotal organs and spermatic cord (Fig. 2). The mechanism of this defense includes three stages: arteriogenous, intraorgan, and venogenous (Fig. 2).

Simulation of arterial hypervascularization of the scrotal organs showed that the main anatomical components realizing the function of temporary deposition of arterial blood are the testicular artery and its first-order branches (epididymal arteries), structural components of intersystem fusion of the testicular artery, and the cremasteric artery with its branches. However, high hypertension in the testicular artery basin can lead to testicular tissue damage (infarction, extravasation). For this reason the scrotal adaptor vessel (the cremasteric artery) is the main anatomic structure provisionally depositing arterial blood excess at the expense of retrograde bloodflow in this artery, dilatation of the main vessel, specific reaction of the helix, and first-order return branches.

The venous testicular and cremasteric antihyperemic mechanisms basically differ, primarily by loading of the compensatory potential of the scrotal organs. Impairment of the blood outflow via the main venous collector of the scrotal organs (testicular vein) causes functional overload of the intersystem venous communicants of all levels, at the expense of which the collateral blood drainage is realized. This causes pronounced dilatation of the communicant veins, venous plexuses of the VD and cremasteric veins, and a rarely seen intermediate cremasteric vein plexus, depositing venous blood. Analysis of the hemodynamic picture of venous testicular hyperemia indicates that the anatomic functional potentialities of the cremasteric and VD

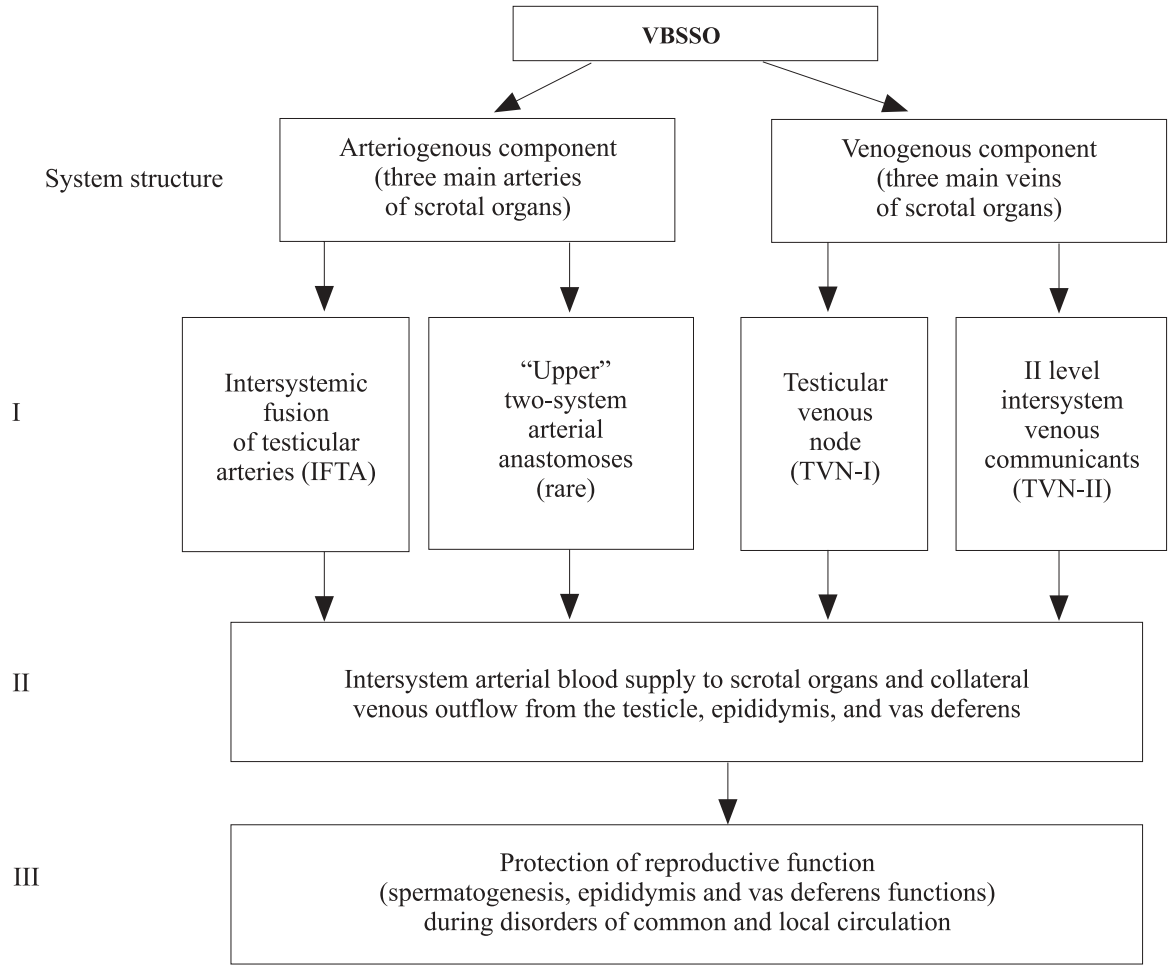

Fig. 1. Anatomic and functional structure of vascular buffer system of scrotal organs. *I*: anatomic basis of collateral bloodflow formation; *II*: hemodynamic result of system functioning; *III*: biological and clinical results of the system functioning.

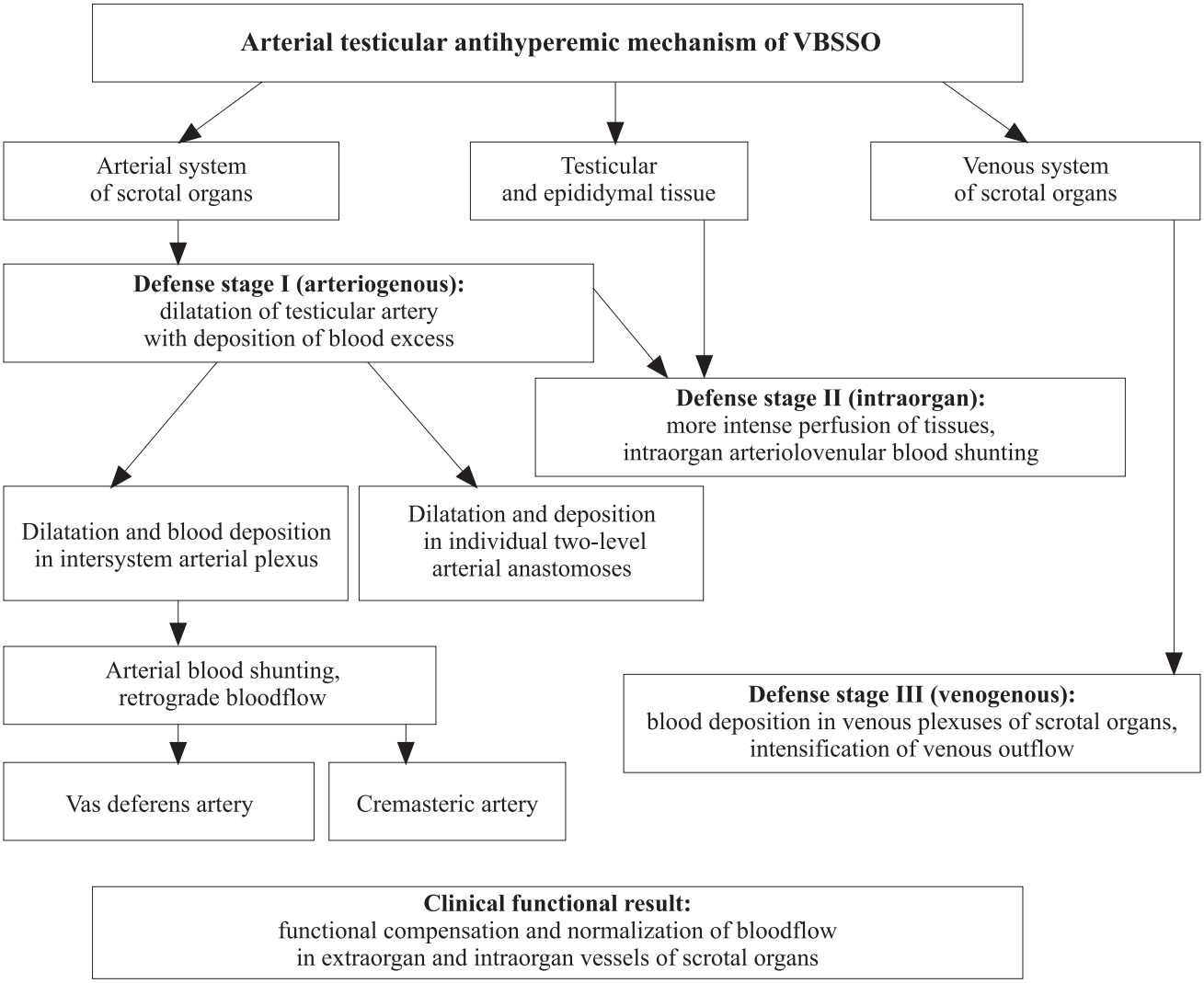

Fig. 2. Anatomic and functional characteristics of arterial testicular antihyperemic mechanism of VBSSO.

vein basins in deposition of testicular "extra blood" are limited and fail to provide a lasting adequate outflow from the testicle and epididymis (Fig. 3).

Damage (partial or complete) to blood outflow via the cremasteric vein does not involve appreciable changes in the total hemodynamics of the scrotal organs. High potential cumulative capacity of the plexus pampiniformis provides deposition and adequate outflow of "extra" venous blood from the cremasteric collector without appreciable functional overloading.

In both cases the mechanism of temporary deposition of venous blood is realized by means of the collateral bloodflow through intersystem venous communicants of two levels, while venous plexuses (pampiniformis, epididymal, VD plexus, and rarely observed intermediate plexus of the cremasteric vein) serve as the key vascular formations realizing this mechanism.

These data indicate that venous plexuses of the scrotal organs are principally significant anatomic components of intricate physiological and pathophysiological mechanisms of local hemodynamics stabilization.

The arterial testicular hyperemia and hypertension in the testicular artery basin are parallel pathophysiological phenomena, closely linked with each other. The hypotensive mechanism of VBSSO and its actual functioning are extremely important primarily for the protection of the testicular and epididymal tissue from vasculogenic damage.

Hypertension in the cremasteric and VD artery basins is less significant for the reproductive system.

The hypotensive mechanism of VBSSO is realized at the arteriogenous, intraorgan, and venogenous levels. The arteriogenous mechanism is functionally the most significant; it consists in shun-

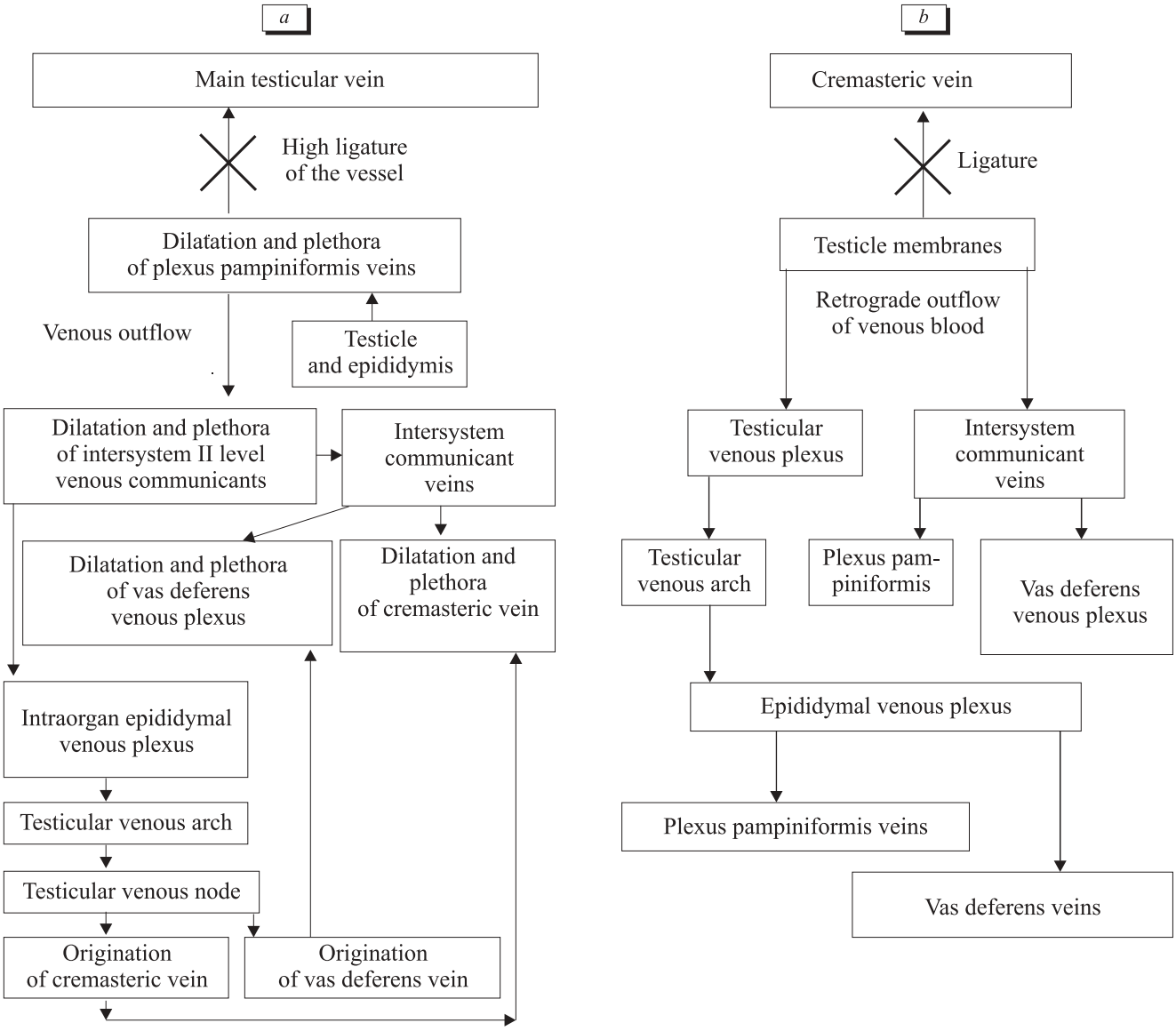

Fig. 3. Anatomic functional characteristics of venous testicular (*a*) and cremasteric (*b*) antihyperemic mechanisms of VBSSO.

ting of arterial blood from the testicular basin into the cremasteric and VD arterial basins. Shunting is realized through intersystem fusion of the testicular arteries by modification of the bloodflow in these vessels. The cremasteric artery and its structural components are principally important for reduction of critical arterial pressure, which suggests regarding this artery as an adaptor vessel. An original hypotensive function of this artery is realized at the expense of the helix (a sort of a hemodynamic coil) functioning and reaction of the first-order return branches, providing pressure reduction at the expense of abrupt "arms". Transition of arterial testicular hypertension into hypertension of the cremasteric and VD artery basins reduces significantly the risk of vasculogenic involvement of the testicular and epididymal tissue. The intraorgan level consists in intensification of tissue perfusion, arteriovenular blood shunting, while the clinical and functional significance of the venogenous level (more intense venous outflow from the scrotal organs) is secondary.

The VBSSO starts functioning under unfavorable conditions leading to modification of the natural functional modes of circulation. Normally, when the total body and the scrotal organs are under physiologically comfortable conditions, no signals for triggering the mechanisms of VBSSO are delivered. The VBSSO can be activated in traumatic and surgical injuries to the main vascular system of the scrotal organs (ruptures of and injuries to the main veins and arteries, deliberate or accidental ligature of extraorgan vessels of the spermatic cord, testicle, and epididymis, such as Ivanissevich, Palomo, or

Marmar's operations, herniotomy, *etc.* [5,7,8]), acute and chronic vascular diseases leading to involvement of the main vessels of the scrotal organs (atherosclerotic stenosing of the testicular artery, extrafunicular varicosis resultant from diseases of the lower limb veins, secondary varicocele in stenosed left renal vein, *etc.*), excessive exercising (heavy athletics), and exposure to negative industrial factors (high gravitation, vibration, high ambient temperature), changes in intraorgan bloodflow in the testicle and epididymis as a result of exposure to negative factors of unfavorable environment (magnetic fields, vibration, *etc.*) or acute diseases (acute orchitis, epididymo-orchitis, *etc.*). These mechanisms of arterial or venous failure compensation can sometimes normalize the local hemodynamics and maintain the normal function of the testicle, epididymis, and VD. However, in some situations VBSSO fails to control the pathological exposure or a long exposure to a negative factor leads to failure of the system's work. For example, ligature of the cremasteric vein does not cause pronounced disorders in the testicular, epididymal, and VD function, while combined or selective ligature of testicular vessels leads to severe impairment of the scrotal organ trophics.

Hence, four functions of VBSSO are distinguished.

The state at rest is functioning of the scrotal vascular system under normal conditions, when the mechanisms of compensation for circulatory disorders are not involved in the hemodynamic processes. The state of VBSSO compensation indicates that exposure to a factor leading to disorders in local circulation triggers the defense mechanisms which provide adequate hemodynamics of the scrotal organs. The subcompensation state implies the threshold permissible parameters of the defense vascular mechanisms functioning, when more intense or further exposure to the negative factors leads to failure in the systems' work and deterioration of local circulation in the scrotal organs. Decompensation of VBSSO means a drastic impairment of the local hemodynamics of the scrotal organs, despite the activity of the compensatory mechanisms, which fail to control the negative factors.

Hence, the development and course of dysorders in hemocirculatory states of the scrotal organs should be regarded from the universal functional defense system viewpoint. Due to this we can clearly see the mechanisms compensating for the vascular dysfunctions, predict their outcome, and develop measures aimed prevention of these disorders.

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