Experimental Anatomic Modeling of Venous Dyshemocirculation in the Scrotal Organs

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Disorders in venous outflow from the testis and epididymis lead to the formation of pathological compensatory hemodynamics, development of high intratesticular pressure with impairment of the integrity of the intra-organ vessels, up to extravasation (testicular venous infarction), to local arterial hypertension, inter-arterial shunting of stained solutions through the intersystem fusion of the testicular arteries (shunting of arterial blood under vital conditions), and hence, trigger the mechanism of secondary arterial ischemization of the testis and epididymis. The severity of circulatory disorders depends on the volume of venous collectors excluded from circulation: from pronounced disorders in case of testicular venous outflow blockade to extremely severe ones in case of combined testicular-cremasteric venous block (hemodynamic collapse).

Key Words: *dyshemocirculation; testicular venous block; testicular-cremasteric venous block; intersystem venous communicants; ischemia*

Surgical methods for the treatment of varicocele (varicosity of veins of the plexus pampiniformis) aimed at impairment of the anatomical integrity of the testicular vein (TV) [3] and other structural venous formations in the scrotal organs [2,4,5] are widely used in clinical andrology, urology, and surgery. These operations result in improvement of circulation in the testicular tissue, normalization of spermatogenesis, and fertile characteristics of the ejaculate. On the other hand, there are no persuasive data on the hemodynamic pattern, forming as a result of exclusion of TV from circulation, combined ligature of TV and cremasteric (CV) and other veins, and on the status of the testicular parenchyma as a result of venous (passive) hyperemia.

We studied the structure and function of the compensatory reaction of extraorgan vascular system of the scrotal organs, intraorgan perfusion by stained solutions and X-ray contrast agent during simulation of venous dyshemocirculatory conditions.

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MATERIALS AND METHODS

Fresh anatomical complexes including the testicle, epididymis, and full-length spermatic cord with all membranes were used in the study. The anatomical integrity of the arterial and venous systems of the scrotal organs and spermatic cord were spared during preparation of the complex to experiment, and therefore the preparation was not "precise", but just "superficial" and was carried out as follows. The main vascular stems were mobilized, after which the common tunica vaginalis testis was opened in vessel-free zone, the edges of the tunic were stretched, and the preparation was thus fixed on the preparation table.

Two series of experiments, 15 per series, were carried out. Venous testicular hyperemia (venous testicular block) was simulated in series I and combined venous testicular-cremasteric hyperemia (combined venous testicular-cremasteric block) in series II.

High ligature of TV trunk was performed in series I, ligature of the main TV and CV trunks in series II.

This was followed by cannulation of the testicular artery main trunk, into which colored solution (china ink; 1:10) or verographin (water-soluble iodine-containing three-atom 76% X-ray contrast agent) was injected with a syringe infusion pump (1235N ATOM). Warm (36-38°C) solutions were injected at rate of 2 ml/min (normal volume blood flow velocity in human testicular arteries during intraorgan testicular blood perfusion at 12.35 ml/min/100 g organ tissue [6]). The duration of each experiment was 0.5-1.0 h until the start of discharge of colored solution from the deferent duct (DD) vein stump. The main testicular arterial trunk stump was then ligated. Additional "superficial" preparation was carried out and the extraorgan vascular system of the testicle was described. After the entire vascular system of the scrotal organs and spermatic cord was filled, colored solutions were discharged from the CV and DD vein stumps in experimental series I and from the DD vein stump in series II. Before injection of solutions into the testicular artery, the cremasteric and DD arteries were ligated in order to prevent leakage in the least resistant (retrograde) direction through the intersystem fusion of the testicular arteries.

RESULTS

Isolated ligature of the TV caused manifest hemodynamic disorders in the scrotal organs, consisting in sharp dilatation of the venous structures, and formation of pathological collateral hemodynamics: changes in the direction of colored solutions outflow from the testicle and other scrotal organs (Table 1).

Combined ligature of the TV and CV led to virtually complete blocking of blood outflow from the scrotal organs, because the DD vein, as the only functioning collector, was obviously unable to maintain adequate blood drainage (Fig. 1).

Venous testicular and combined testicular-cremasteric blocks, causing pathological compensatory venous collateral circulation, trigger, in parallel with this, the mechanisms of arterial shunting of colored solutions with the formation of retrograde pathways of testicular arterial blood discharge into other main vessels of the scrotal organs (the cremasteric and DD arteries) via the intersystem fusion of the testicular arteries (Fig. 2).

Hence, the opinion that exclusion (clipping, resection, ligation) of venous vessels involved in the scrotal organs circulation has no negative impact on local circulation and even improves it is erroneous. Disorders in the venous outflow after exclusion of the main vein not only directly leads to testicular and epididymal hypoxia due to developing "passive" (venous) hyperemia of these organs, but also triggers the mechanism of arterial ischemia at the expense of formation of pathological compensatory shunting of the arterial blood flowing via the testicular arterial trunk. Hence, combined ligature of TV and CV, causing virtually complete venous block, leads to hemodynamic collapse under conditions of an anatomical experiment, decompensating the arterial and venous components of the scrotal vascular system.

The degree of intraorgan testicular infusion under conditions of anatomical experiment was evaluated visually by the pattern and intensity of the

TABLE 1. Visual Reaction of Extraorgan Arterial and Venous Systems of the Scrotal Organs during Modeling of Venous Dyshemocirculatory Conditions (n=15)

Anatomical vascular structures	Venous testicular block	Combined venous testicular-cremasteric block
Intersystem fusion of testicular arteries	Sharp dilatation, intense staining	Sharp dilatation, intense staining
Testicular venous node	Intense staining	Intense staining
Testicular artery	Sharp dilatation of the main trunk and I-II order branches, intense staining	Sharp dilatation and staining
Level II intersystem communicants	Intense staining and dilatation	Intense staining and dilatation
Cremasteric artery	Moderate staining and dilatation	Retrograde flow of colored fluid, moderate staining
DD artery	Staining and dilatation	Retrograde leakage of colored substance
DD vein	Staining and dilatation	Slight staining
CV	Sharp dilatation and staining	Devastation

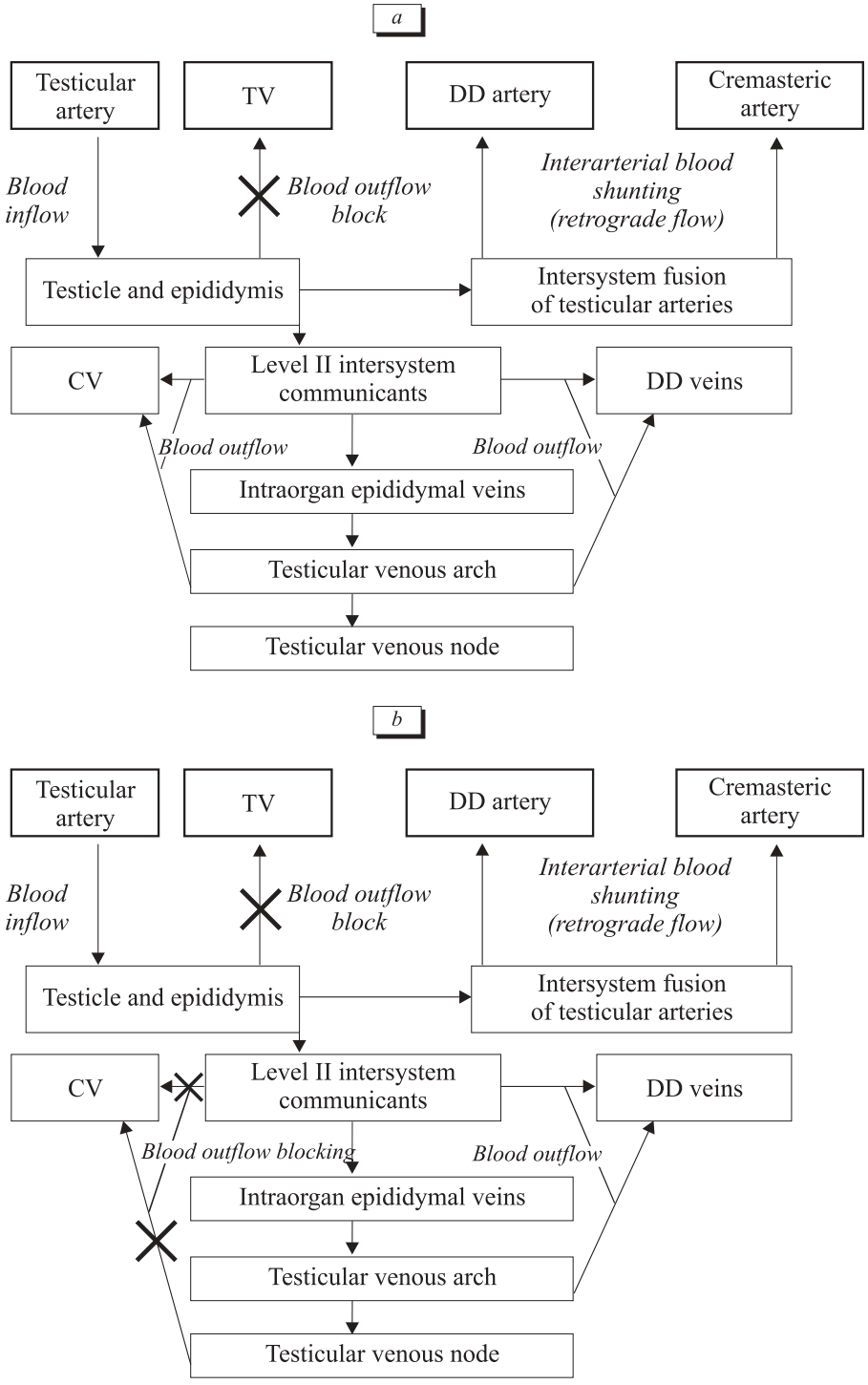

Fig. 1. Scheme of pathological compensatory hemodynamics of the scrotal organs under conditions of venous testicular (*a*) and combined venous testicular-cremasteric (*b*) blocks.

parenchyma staining at the site of the testicular tunica albuginea incision (3-4 cm incision). These studies were carried out directly after visual description of the extraorgan vascular system of the scrotal organs and completion of the main vascular experiment.

X-Ray study of anatomical preparations after injection of verografin into the vascular bed gave a more objective picture of the testicular intraorgan vascular system.

The degree of intraorgan perfusion of testicular parenchyma vessels depends on the type of ex-

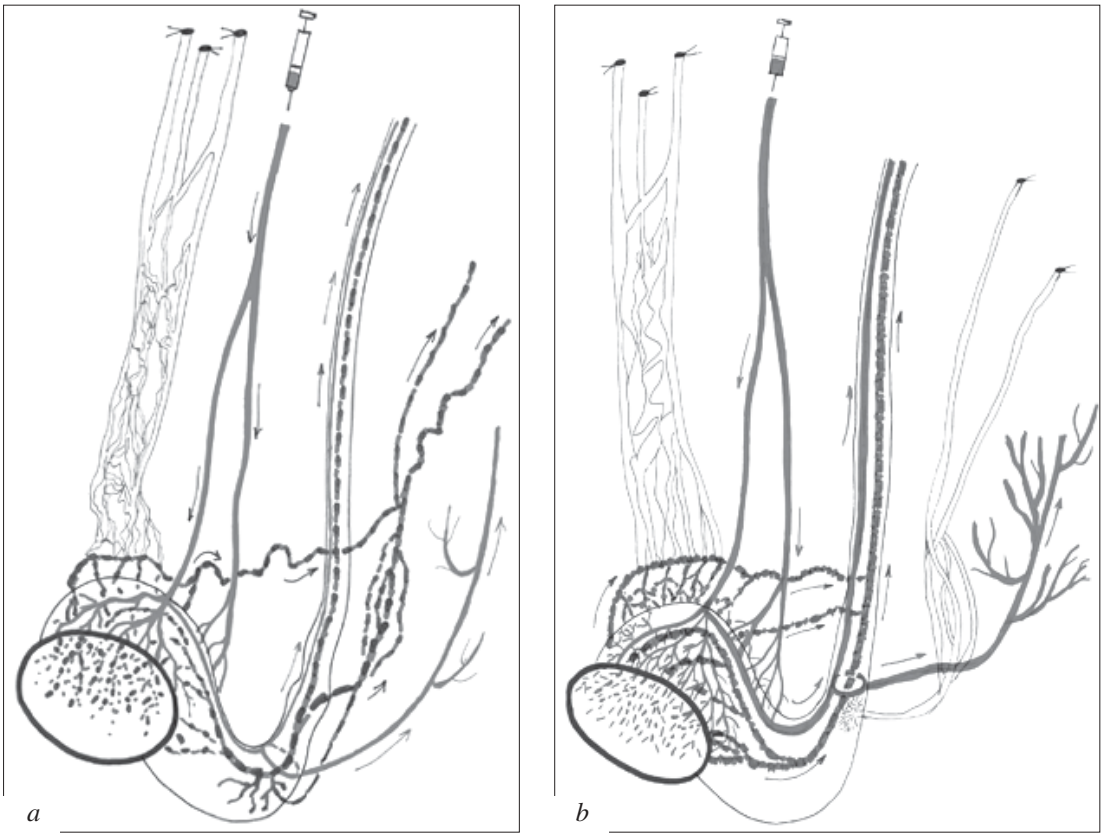

Fig. 2. Compensatory hemodynamics during modeling of venous testicular (*a*) and combined testicular-cremasteric (*b*) blocks. *a*) development of combined venous and arterial compensatory vascular reactions. Blood draining into CV and DD vein basin through dilated intersystem venous communicants of both levels. Inflow of colored substance via the testicular artery, preset at physiological parameters, becomes excessive. This leads to retrograde shunting in the cremasteric artery and DD artery basins through the intersystem fusion of testicular arteries. Arrows show the flow of colored solutions in vessels. *b*) hemodynamic collapse: pronounced dilatation of intersystem venous communicants of both levels, DD venous plexus, main TV trunk with branches, all anatomical components of intersystem fusion of the testicular, cremasteric, and DD arteries.

perimental models of disorders in venous outflow from the scrotal organs.

The severity of changes in the intraorgan perfusion by stained solutions in venous hyperemia is worthy of note (sharply pronounced intense staining of the testicular parenchyma, up to release of colored solution from the testicular tissue, extravasation) (Fig. 2). These data disagree with common opinion on the predominantly arteriogenous nature of vascular disorders in the testicular parenchyma and spermatogenesis. It is obvious that impairment of the blood outflow from the testicle leads to severe changes in the organ trophics and trigger the secondary mechanisms of arterial insufficiency of the testicle and epididymis.

Secondary arterial ischemia of scrotal organs developing in the presence of impaired outflow in the TV, CV, or DD vein is governed by the mechanism of pathological compensatory shunting of arterial blood through the natural anastomoses (intersystem fusion of testicular arteries) with the development of vascular hyperfunction and formation of the retrograde bloodflow in the DD and cremasteric artery basins (Fig. 3).

Hence, any violation of the natural balance consisting in the equilibrium between the arterial inflow and venous outflow leads to severe hemodynamic consequences for the sexual gland, epididymis, and DD.

It is obvious that surgical interventions arresting the bloodflow in the testicular vein basin [3] and additional ligature of the CV [2] and level II intersystem venous communicants [2,4,5] cannot create the conditions for improvement of spermatogenesis, because they lead to irreversible disastrous hemodynamic conditions for the sexual gland and epididymis trophics. Positive results of the ejaculate analysis after venoresecting surgery are explained not by the positive effect of the intervention proper on the gametogenesis, but by oral treatment with potent stimulants of spermatogenesis (androgenic hormones, gonadotropins, phyto- and biogenic stimulators, oral enzymes, vitamins, antioxidants, *etc.*) [1].

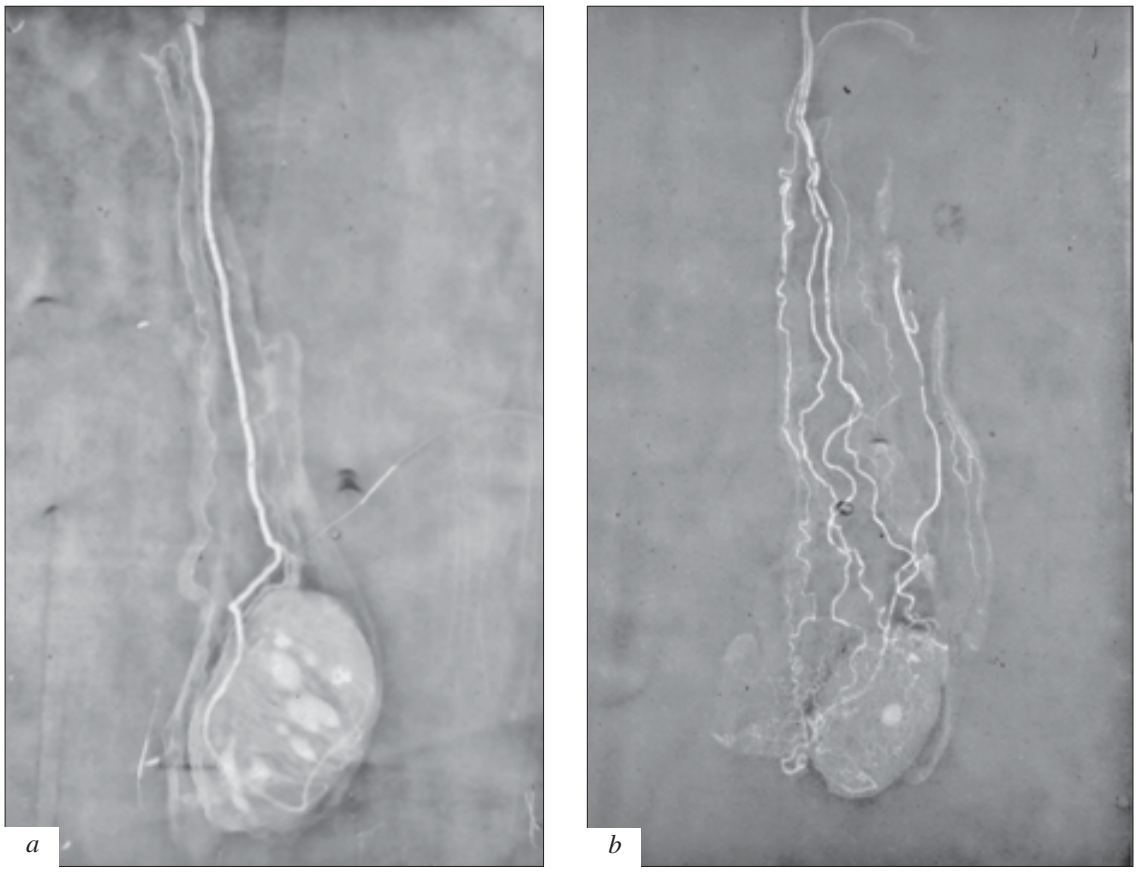

Fig. 3. Angiographic picture during modeling of venous testicular (*a*) and combined venous testicular-cremasteric (*b*) blocks. *a*) sharply dilated testicular, epididymal, *etc.* arteries, on which poorly discernible shadows of the main veins are superimposed. Testicular parenchyma is hyperemic, shadows of numerous extravasates are seen (the picture of testicular venous infarction). *b*) hemodynamic collapse. Intense twisting of 3 main testicular arteries (signs of inter-arterial shunting), onto which poorly discernible blurred shadows of the veins are superimposed. Intraorgan testicular and epididymal vessels are twisted and plethoric. Intratesticular extravasates (venous infarctions) are seen.

Hence, wide use of surgical interventions for varicocele, aimed at circulation disruption in varicose veins of the scrotum, is etiopathogenetically unjustified.

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