Institute of Experimental Morphology and Anthropology with Museum
Bulgarian Anatomical Society

Acta morphologica et anthropologica, 13
Sofia • 2008

Microvascular Mechanisms of Chronic Venous Insufficiency

M. Minkov

Department of Anatomy, Histology and Embryology, Prof. Paraskev Stoyanov
Medical University of Varna

The study covered a material of 152 affluents of the saphenous veins as well as an operative material of 60 patients. The considerable number of the direct and indirect affluents without any valves in the outfall allows us to consider that there is a possibility for a blood reflux into the *vasa vasorum* of the saphenous veins not from the lumen of this vein itself but from the lumen of the affluents that do not possess any valves in their outfalls. In case of hypertension in the principal trunk and its affluents certain conditions for the development of reflux to the dermal pool, too, are created which results in oedema, microangiopathies and skin ulcerations.

Key words: saphenous vein, valves, *vasa vasorum*, varicosis.

Introduction

Chronic venous insufficiency of the lower extremity is a widely disseminated disease that, according to some authors' opinion, affects up to and over 50% of the examined populations. There exists a united concept in the literature available that the venous hypertension and the stasis related to it represent the first clinical symptom of the chronic venous insufficiency. C r o t t y [2, 3, 4] accepts that the development of the varicose process is related with the blood reflux into the *vasa vasorum* of the venous wall from the lumen of the vein. According to V a n c o v [12, 13] and M a r i n o v [14], a direct drainage of the *vasa vasorum* into the lumen of the saphenous veins can be observed as an exception only.

Based on our own investigations of the valvular apparatus of the superficial affluents of the saphenous veins as well as on the studies of the *vasa vasorum* of the valvular sinus wall of the varicose veins we defined our purpose to clarify the microcirculatory mechanisms that control the chronic venous insufficiency.

Material and Methods

The study covered 152 affluents of the saphenous veins, i.e. 61 direct and 91 indirect ones. The investigation of the valvular sinus wall was carried out on an operative material taken from 60 patients.

The provision with valves of the superficial affluents and the localization of the valves in it underwent an examination by means of a stereomicroscope. The material for the light-microscopic observation of the valvular sinus wall was fixed in a combination of different fixers. Stainings with hematoxylin-eosin (HE), orcein, Azan and after the methods of Mallory and of Van Gieson were made use of. The histological sections were examined under OLYMPUS BX-500 microscope and filmed by a video-camera.

Results and Discussion

Our study demonstrated that the provision with valves of the affluents of the saphenous veins, and especially of the indirect affluents places them into an unfavourable situation towards the venous hypertension in case of disturbed venous outflow from the lower extremities (Fig. 1).

The 'valvular index' after V a n c o v [12] concerning the direct affluents amounts to 2,06 while that concerning the indirect ones — to 1,28. The great number of the direct and indirect affluents without any valves in the outfall allows us to consider that there is a possibility for a blood reflux into the *vasa vasorum* of the saphenous veins not from the lumen of this vein itself but from the lumen of the affluents that do not possess any valves in their outfalls. In case of hypertension in the principal trunk and its affluents certain conditions for the development of a reflux to the dermal pool, too, are created which causes oedema, microangiopathies and skin ulceration. According to the present understanding of the topic, the pathological alterations in chronic venous insufficiency result from a preliminarily programmed

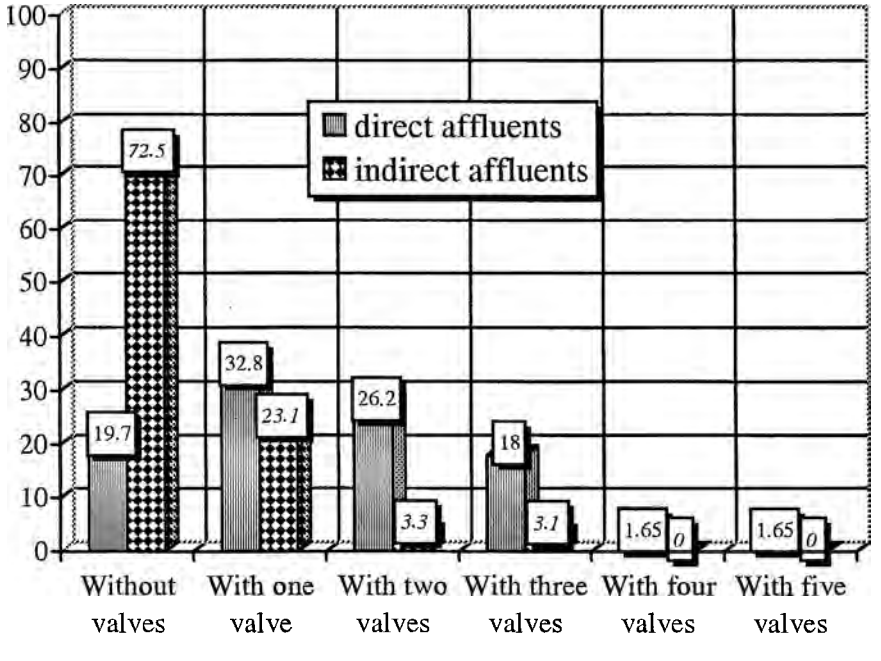

Fig. 1. Percentage distribution of the number of valves in the single affluents

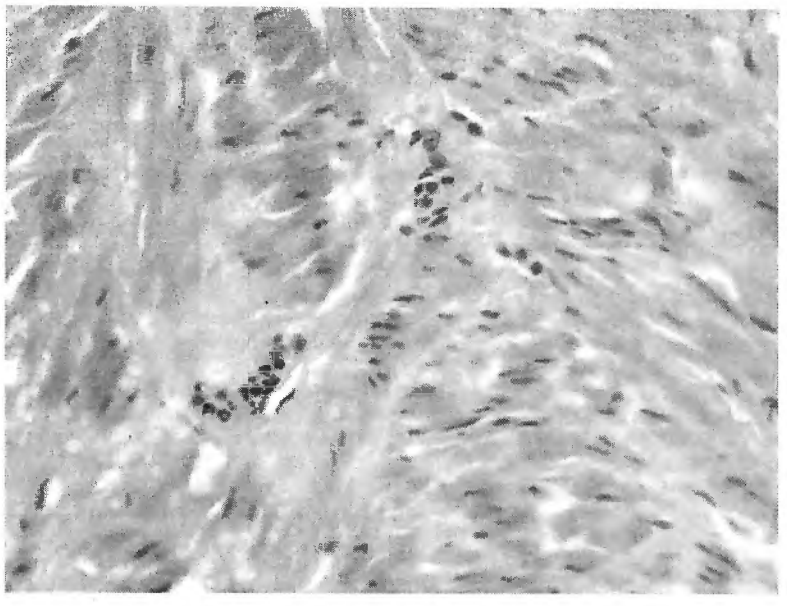

Fig. 2. Vena saphena magna. Vasa vasorum in the valvular sinus wall. HE, × 400

cascade of reactions with numerous signs of inflammation [1, 5, 6, 8]. The venous hypertension, blood stasis and hypoxia activate the endothelial cells and leukocytes towards an increased expression of adhesive molecules into their membranes through which an adhesion of the leukocytes on the endothelial surface and their penetration into the venous wall is accomplished (Fig. 2).

The enhancement of the tissue compression on the intramural vessels in the venous wall blocks the blood circulation in them which, on its part, leads to hypoxia of the venous wall. Besides the other source of oxygen provision of the venous wall, i.e. the blood stored in the lumen, presents with a lowered oxygen concentration.

Under the conditions of hypoxia, the endothelial and smooth muscle cells of the venous wall modify their physiological activity as some active substances such as growth factors and matrix metal proteinases are produced and thus the elimination of NO is suppressed [1, 7, 9, 10, 11]. In aggregate and by definite sequence of incorporation, these processes realize a venous wall remodeling. At the level of the principal subcutaneous veins and their affluents they manifest themselves with varicosities of the veins while at the level of the microcirculatory part of their pool - with skin alterations, including even with varicose ulcers.

References

1. Coleridge-Smith, P., J. Bergman. Inflammation in Venous Disease. — In: Molecular Basis for Microcirculatory Disorders (Eds. G. W. Schmid-Schönbein, D. N. Granger). Paris etc., Springer-Verlag, 2003, 489-500.
2. Crotty, T. The roles of turbulence and vasa vasorum in the aetiology of varicose veins. — *Med. Hypotheses,* **34**, 1991, 41-48.
3. Crotty, T. An investigation of the vasa venarum in a canine vein using radial reflux perfusion. — *Phlebology*, **10**, 1995, 12-18.

4. Crotty, T. The corrupted feedback hypothesis. — *Med. Hypotheses*, **61**, 2003, 605-616.
5. Jünger, M., S. Braun. Microvascular mechanisms controlling CVI. — In: Molecular Basis for Microcirculatory Disorders (Eds. G. W. Schmid-Schonbein, D. N. Granger). Paris etc., Springer-Verlag, 2003, 501-513.
6. Michiels, C. Role of the respiratory activity of vascular endothelial mitochondria in the pathophysiology of CVI. — *Phlebolymphology*, **39**, 2003, 105-112.
7. Nicolaides, A. Pathophysiology of chronic venous insufficiency. — *Medicographia*, **26**, 2004, 2, 128-132.
8. Schmid-Schönbein, G., Ph. Faha. Inflammation and the pathophysiology of chronic venous insufficiency. — *Phlebolymphology*, **39**, 2002, 95-104.
9. Schmid-Schönbein, G., S. Takase, J. Bergan. New advances in the understanding of the pathophysiology of chronic venous insufficiency. — *Angiology*, **52**, 2001, 27-34.
10. Takase, S., J. Bergan, S. Schmid-Schönbein. Expression of adhesion molecules and cytokines on saphenous veins in chronic venous insufficiency. — *Ann. Vasc. Surg.*, **14**, 2000, 427-435.
11. Weber, C. Novel mechanistic concepts for the control of leukocyte transmigration: specialization of integrins, chemokines, and junctional molecules. — *J. Mol. Med.*, **81**, 2003, 4-19.
12. Ванков, В. Строение и васкуляризация стенки вен и их клапанов (канд. дис.). Ленинград — Варна, 1968.
13. Ванков, В. Морфология на вените. София, Мед. и физк., 1989, 208 с.
14. Маринов, Г. Строеж и васкуларизация на вените на подбедрицата и задколянната яма (канд. дис.). Варна, 1969.