ORIGINAL PAPER

The valvular segment of the lower limbs venous system: anatomical, physiological and physiopathological aspects

F. CALOTĂ, S. S. MOGOANTĂ, MIRELA-MARIA VASILESCU, I. VASILE, M. PAŞALEGA, M. C. STOICEA, D. CAMEN, C. MEŞINĂ

II[nd] Clinic of General Surgery,
University of Medicine and Pharmacy of Craiova

Abstract

The valvular segment is a distinct venous structure, which, from a morphological point of view, is comprised of the following components: the valvular insertion, the valvular gorge entrance orifice, the valvular defile, the valvular gorge exit orifice, the valvular sinus. Endoscopic and echo Doppler examinations are used to identify the normal and the pathological morphology of the valvular segment, and the hemodynamic phenomena occurring at this level. Cusps' integrity and size as well as valvular dynamics are key elements directly involved in shaping the valvular segment in general, and the valvular sinus in particular. The valvular sinus shows an obvious hemodynamic determinism. Valvular segment pathology is the outcome either of a progressively long evolving process initialized by gravitational venous pressure overcharges, or of a rapidly evolving process such as the hemodynamic shock following intense physical efforts. Valvular defunctionalisation implies a different mechanism and a different type of cusp lesion.

Keywords: endoscopy, valvular segment, duplex color, reflux commissural canal, valvular lesions.

Introduction

The advance of the nowadays investigation technologies facilitated an extension to our clinical senses and a close observation of the most intimate structures of the human body. This, in turn, extended our informational horizon with key data, both practical and clinical, that corrected many of our mental, highly abstract representations. An evidence-based medicine cannot be conceived without the objectivity brought by the abovementioned technologies [1]. From this point of view, the complex duplex color and videoendoscopic examination of accessible sections of the venous system brought new insights regarding venous anatomy, physiology and physiopathology.

The central purpose of this study refers to an attempt to fully examine the valvular segment, which has a fundamental role in venous physiology, and a specific pathology as well.

Patients and Methods

Our observations include 46 patients with chronic venous insufficiency of the lower limbs (CEAP, classes 2–4) that were subject to surgical amendment or annulment of the venous reflux. Presurgical evaluation was performed using clinical examination, duplex color sonography (Aloca ultrasound with variable frequency probe) and biological data; surgical evaluation was performed using endoscopy, based on the patients' written consent following detailed information on the procedure. All patients received a prophylactic dose of 5000 U of low molecular weight heparin. Endovenous explorations were performed with a Storz endoscope. We used glucose 5% to wash and visualize. Images were initially stored as VHS recordings, and later transferred on digital devices (CDs). Duplex color and endoscopic examinations collected specific and many times complementary information.

Results

Information was processed and later revised repeatedly for further clarifications. Correlating duplex color and endoscopic information allowed us to identify the normal as well as the pathological anatomy of the valvular segment, certain interesting phenomena and hemodynamic mechanisms encountered at this particular level. All these pieces of information sustain the *venous segment* concept. Duplex color and endoscopic examinations allowed a detailed description of the valvular segment morphology with all its components. We selected the most evocative images to illustrate this study. Based on our endoscopic observations, we propose a pathogenic classification of valvular lesions.

The valvular segment is a distinct venous structure, which, from a morphological point of view, is comprised of the following components:

▪ *the valvular insertion ring*, equivalent to an area of slight but obvious strangling of the venous lumen. It represents the cusps' anchorage ring and, from a histological point of view, consists of a collagen-muscular densification. In cranial endoscopic examination, it looks like an obvious subvalvular threshold (Figure 1).

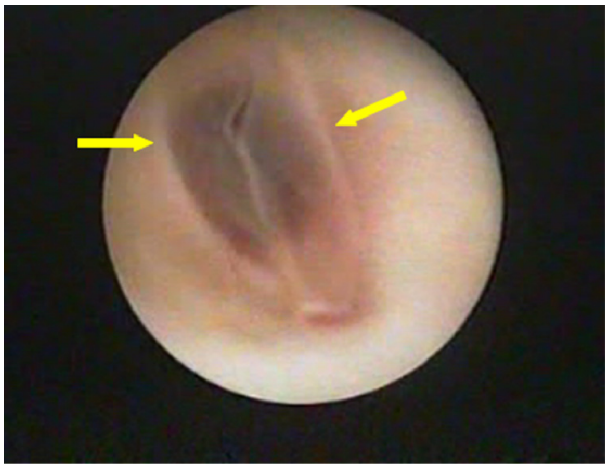

Figure 1 – *The valvular insertion ring (venous endoscopy, 5×).*

▪ *the entrance orifice* into "the valvular defile", approximately elliptical, lined by the cusps' inferior contours and commissures;

▪ *the valvular gorge* or *the valvular defile*, delimited by the cusps' axial surfaces. It has an elliptical appearance when the valves are open and that of a flat frustrum of a cone when they are closed.

▪ *the exit orifice* of the valvular gorge is limited by the cusps' free edge. Its circumference is larger than that of the entrance orifice and usually it is not two-dimensional. Natural contrast echo Doppler examination can efficiently reveal the ejection phenomenon (Figure 2) occurring as blood leaves the valvular defile through the exit orifice.

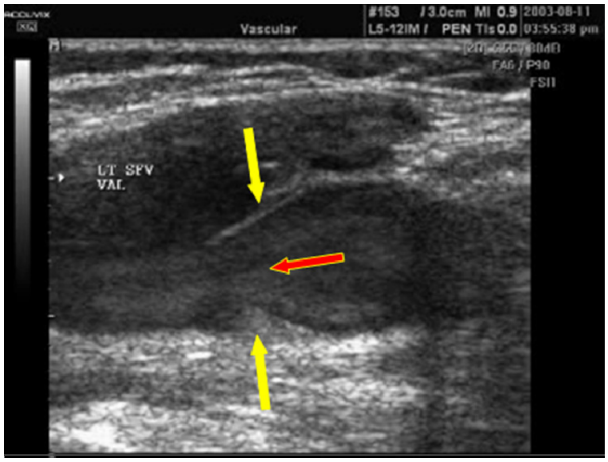

Figure 2 – *The ejection phenomenon (red arrow) between the valves (yellow arrows) (natural contrast echo Doppler).*

Frequently, the cusps' free edge shows a thickened border (Figure 3), with a marginal hemstitch. Occasionally, polyps can be seen at this particular level. Our histological observations indicate that valvular endothelial cells are disposed predominantly perpendicular on the streamlines, especially if compared to the parallel disposition of the endothelial cells outside the valvular segment.

▪ *the valvular sinus*, delimited by the venous wall, the parietal-cuspal angle and the cusps' parietal surface, is shaped like a bulb, with the maximal diameter above the cusps' insertion ring. The size of the valvular sinus is proportional to the cusps' size, and this specific correlation determines the morphology of the hemodynamic singularity (of the valvular segment as a whole), the amplitude of the hemodynamic events that take place at intravalvular and sinusal levels. The endothelial cells of the sinus have a cobblestone arrangement with no particular pattern, most probably caused by the intense turbulences described above.

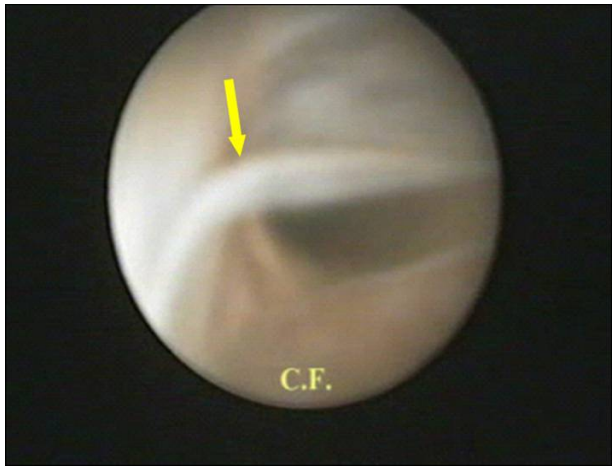

Figure 3 – *Thickened border of one cusps free edge (venous endoscopy, 5×).*

An interesting aspect is seen with complex sinuses, where three affluents converge. Usually, this type of sinus is large and shows a transversal exuberant ostial valve helix or "S"-shaped. While flexing, one end closes an affluent; as the other counter-flexes, it closes the second affluent; the transitional segment closes the central affluent. The gradual reduction in bloodstream pressure allows the central, transitional segment of the valve to open.

These successive valvular segments render venous courses a slightly moniliform appearance; many times, valvular sinuses can be identified clinically in replete superficial veins.

Beyond these normal morphological aspects venous endoscopy of the lower limbs allowed us to directly observe valvular lesions *in vivo*.

The first group of lesions was the functional one formed by the commissural slit and canal of reflux (Figures 4 and 5). In this type of lesion the valves are not altered from the morphological point of view, but they become more or less competent subjected to a progressive increasing pressure that produces the dilatation of the insertion ring and not only, as we will discuss later.

The second type of lesions, consisting in valvular ruptures at the insertion level or on the free edge (Figures 6 and 7), was considered to have a traumatic determinism – brutal increase of the pressure in the valvular segment. We found those lesions also in high performance athletes, especially weight-lifters or volleyball players at young ages.

Another type of lesions that we can describe in venous endoscopy includes the inflammatory reshuffling of the valves. The valves are distorted or partially twisted, with a thickened border (Figure 3), in some cases resembling polyps, occasionally with a marginal

hemstitch or local thrombosis. It is pretty difficult to make a clear distinction between these two last types of lesions from an ethiopathogenic point of view because every traumatic lesion determines inflammation, and inflammation predisposes to traumatic lesions by reducing flexibility and resistance of the valves, their spatial arrangement or dynamics.

Figure 4 – ***The commissural slit shaped like a tennis racket with the tail oriented towards the venous axis (venous endoscopy, 5×).***

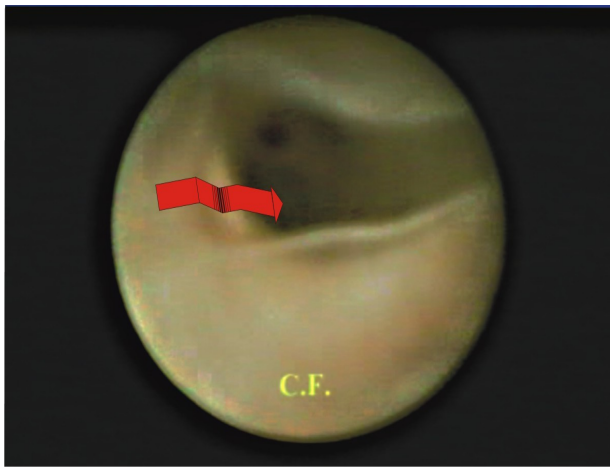

Figure 5 – ***The reflux commissural canal (venous endoscopy, 5×).***

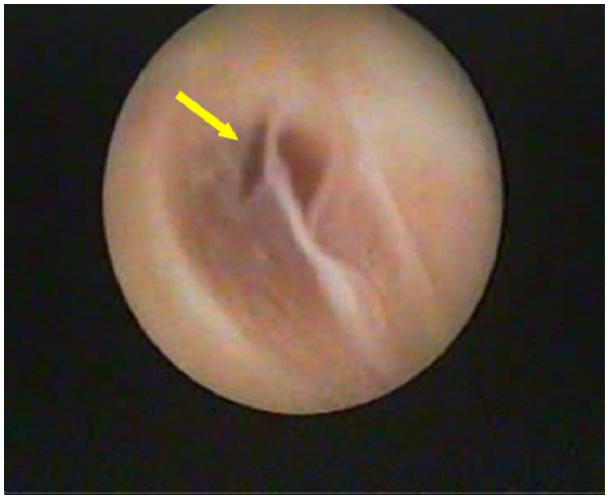

Figure 6 – ***Valvular commissural ruptures (venous endoscopy, 5×).***

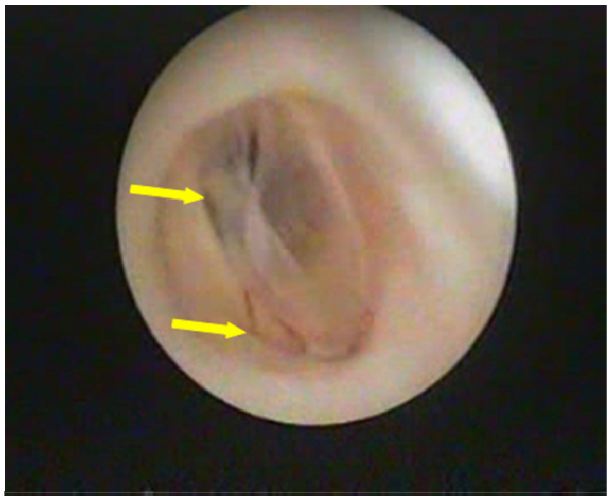

Figure 7 – ***Cusp insertion ruptures (venous endoscopy, 5×).***

In some cases, we identified, in the venous lumen, fringes of endothelium, anchored on both sides of the wall, floating on the fluid like banners in the wind (Figure 8). We recognized those fringes as being fragments of valves by their position in an area resembling a valvular segment but with a dimmed morphology. We consider those lesions the most advanced that we have encountered before the full disappearance of the valves and of the valvular segment. We called them valvular vestiges.

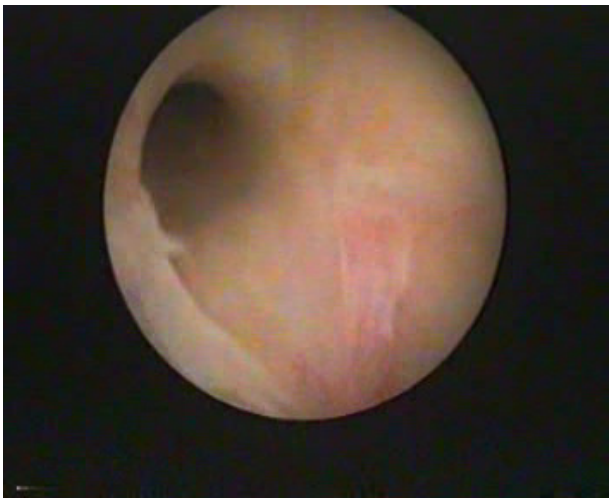

Figure 8 – ***Valvular vestiges (venous endoscopy, 5×).***

Discussion

The caliber reduction produced by the valvular gorge, comparable to a diaphragm type singularity in fluid mechanics, induces an increase in bloodstream speed according to Bernoulli's equation. The section area delimited by the cusps' luminal surfaces represents up to 2/3 of the venous section above valvular level [2].

From this point of view, valves can be considered "bloodstream accelerators"; on the other hand, valves function as "bloodstream stabilizers" as well, because the valvular defile transitorily restores the blood flow laminarity.

The secondary pressure decrease on the cusps' luminal surface is transmitted to the sinusal surface as

well, and thus also to the valvular sinus. Following this phenomenon, a volume mass of blood is drawn within and pressures the cusps preventing them from plating against the venous wall [3].

Bloodstream acceleration within the valvular gorge can be objectified easily with natural contrast echo Doppler examination.

Just as they leave the valvular defile, blood streamlines generate a jet constriction (from a mechanical point of view); this phenomenon is precisely defined in physical and mathematical terms; moreover, in hemodynamics there is even a so-called jet constriction coefficient (C). Following this event, the bloodstream section area widens as the surrounding fluid is drawn in, generating the so-called *ejection phenomenon*. On the laminar flow border exiting the valve along with the surrounding fluid inertia resistances are generated, which reverse marginal flow vectors; this induces whirls next to the venous wall, with no apparent mass blood dislocation. The valvular sinus represents a segment where complex flows are generated. Among the consequences, on one hand, there is an increase in the endothelial permeability for macromolecules, as well as an increase in ICAM-1 molecular expression responsible for leukocytic adherence and migration; on the other hand, there is an increase in the endothelial cellular *turnover* rates [4]. From a strictly hemodynamic point of view, a *dead water sector* emerges, which, following the static pressure decrease, will occupy the valvular sinus.

In this space, at the fluid-wall interface, whirls are being generated vividly, demonstrated with natural contrast echo Doppler examination; these whirls prevent the concentration of procoagulant factors and blood cells at this point (according to the classical theory). *The dead water sector in the valvular sinus leaves the cusps in a "waiting" position, ensuring readiness in valvular closure when bloodstream changes occur* [1, 3].

The similarity between the valvular endothelial disposal and the vascular pattern in the aorta walls is quite interesting: the valvular endothelium functions like a distinct tissular structure, completely different from the remaining endothelium [5]. Unidirectional stress on the valvular endothelium represses all oxidative and inflammatory reactions in order to preserve this mature phenotype of endothelium, whereas reactivity is contrary to turbulent flow and an argument in favour of a heterogeneous kinase signaling [5].

In large veins, valve closure is achieved by the inertial action of the blood, while in small veins this process relies on blood viscosity [6]. The valvular closing mechanism in intermediate caliber veins may also involve viscose forces [7, 8]. Our endoscopic observations concluded that the opening-closing dynamics in valves forced by retrograde high pressure is produced eccentrically, starting at one of the commissures; the opening mechanism works exactly the opposite way. In closed position, the valve exerts a flap effect on the blood flow, dispersing the subvalvular vectors that direct blood circumferentially towards the wall. As the dynamic pressure increases above valvular level, it closes the cusps, and thus the closed valve functions like a wedge, or better, like "water shed", splitting the inverted blood flow. Under hydraulic (hemodynamic) shock, pending on the wall elasticity, the kinetic energy is stored as static pressure in the sinusal wall and thus opens the sinus (Windkessel-like phenomenon). When the valve opens and flow is resumed, secondary to the supravalvular pressure being annulled, this energy is transferred back to the blood flow. As the flux is inverted, turbulences occur both supra- and subvalvularly, with certain, but yet unclear, thermodynamic consequences.

The gradual and prolonged pressure increase in the main venous trunks following prolonged orthostatism (gravitational venous hypertension) induces spatial geometry alterations of these veins – from ellipsoid shape to cylinder. All these phenomena summed up determine maximal mechanical stresses in the cuspal-parietal sulcus (which constitutes a low resistance area at commissural level) and move the cusps' insertions along the circumference thus generating a *commissural slit*, shaped like a tennis racket with its tail oriented towards the venous axis (Figure 4), through which reflux occurs. Bergan JJ and Schmid-Schönbein GW consider that the earliest defect is represented by a commissural space increase that allows reflux along the venous border [8]. Persistence of venous hypertension generating factors increases the refluent blood volume gradually and, in time, a *reflux commissural canal* appears, delimited by the sinus wall, the cranial surface and then the axial border of the valvular insertion, and the cuspal opening (Figure 5). Refluent mass volume is projected from the insertion ring threshold against the opposite venous wall, thus inducing an eccentric subvalvular dilatation, easily identifiable in duplex color examination (Figure 9).

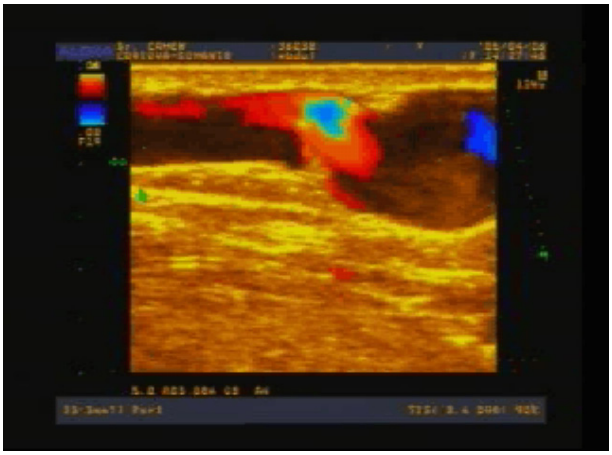

Figure 9 – *Eccentric reflux with secondary eccentric subvalvular dilatation (echo Doppler).*

This observation is consensual with histopathological findings regarding an immediately subvalvular aneurismal dilatation of the internal saphenous vein [9]. The reflux–flux collision, depending on the mass volume dislocated and on the variable dynamic pressures, deviates flux vectors towards the wall through a *nozzle-flap mechanism* [10]. As a result, on one hand, a mechanical stress distends the venous wall and, on the other hand, significant frictional forces are being generated between flux and reflux courses issuing

caloric dissipation (an increase in the number of particle collisions per volume unit) with a possible inhibitory effect on α_2-adrenal receptors, and supplementary venous dilatation [1].

Brutal pressure increases in different segments of the muscular-venous pump (intra-abdominal, leg, etc.) can generate valvular commissural ruptures (Figure 7) or cusp insertion ruptures (Figure 7). These types of lesions were described and classified by Hoshino S *et al.* [11]. The commissural reflux canal is shaping based on the direction and amplitude of pressure stresses. Isolated or even segmental valvular insufficiency is well tolerated, at least initially, if lesions occur on more valvular segments (an extremely rare occurrence). Gravitational pressure charges will later reshape the venous trunk, depending on other associated favoring factors as well.

At valvular level, pressure and inflammatory factors generate specific lesions. Based on our endoscopic observations, we propose the following classification for valvular lesions [1]:

1. Functional valvular lesions (type I) generated by gradual and prolonged venous pressure increases. These lesions can be subclassified in:
   - reflux commissural slit (type Ia);
   - reflux commissural canal (type Ib).

The difference between these two subtypes is clearly morphological, and they obviously correlate with the size of the refluent mass volume.

2. Organic traumatic valvular lesions (type II) – valvular ruptures:
   - commissures: splittings and stretchings (type IIa);
   - cusp insertions: linear perforations (type IIb).

These lesional subtypes do not seem entirely traumatic, leukocytic infiltration on the cranial surface of the cusps [10] generates breaches in their structure and stimulates the apoptosis of endothelial cells at this level. The traumatic factor, however, plays the decisive role, explaining the ruptures in cusp insertions or nearby when the reflux dynamic force is massively brutal. In these cases, the rupture direction is perpendicular to the reflux direction.

3. Organic inflammatory lesions (type III). Inflammatory structural reshufflings of the cusps were seen when the venous wall was affected extensively, with obvious caliber and shape alterations, with parietal stiffness, etc. Certain authors consider that inflammatory valvular lesions are primitive [12] and solitary; their argument resides in the highly increased expression of adherence molecules and in monocytes being discovered underneath the endothelium of the cusps' cardial surface.

4. Valvular vestiges (type IV). Here we included the valvular remains following reflux induced inflammatory or traumatic processes. Valvular vestiges (Figure 8) can easily be mistaken for endothelial folds and fringes. The sinus has been annulled, the vein caliber is either regular or irregular, and it does not increase at higher work-fluid pressures.

Conclusions

In our opinion, the valvular segment is an anatomic and physiological device with key roles in venous blood flow. The valvular sinus shows an exclusively hemodynamic, non-gravitational determinism; it also has a specific pathology. Valvular segment defunctionalisation, under specific pathological conditions, constitutes the basis for secondary venous parietal remodeling. The advance of the nowadays investigation technologies facilitated our knowledge of valvular segment morphology and functions.

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Corresponding author

Firmilian Calotă, Professor, MD, PhD, IInd General Surgery Clinic, Emergency County Hospital, 1 Tabaci Street, 200642 Craiova, Romania; Phone +40723–612 999, +40726–323 242, e-mail: ssmogo@yahoo.com

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