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The Descending or Saphenocentric Theory of Superficial Venous Insufficiency.

La théorie de l'insuffisance veineuse superficielle dite descendante ou saphéno-centrique.

Transcript from the last meeting of the French Society of Phlebology

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Summary

The aetiology and pathogenesis of varicose veins remains poorly understood. At present two popular theories exist; the ascending and descending theory of varicose vein development.

The descending theory proposes that the primary pathology arises from proximal abnormalities usually affecting the terminal valve at the saphenofemoral junction (SFJ) or saphenopopliteal junction (SPJ).

Incompetent valves in a proximal vein segment result in the reflux of venous blood and an increase in haemodynamic pressure on the valve immediately distal, resulting in subsequent incompetence and distal vein dilatation. The theory is supported by cadaveric studies, reporting reduced or absent valves in the external iliac and femoral veins in patients with evidence of venous insufficiency and in cadaveric studies and limbs undergoing superficial venous surgery in comparison to normal limbs, valves are significantly reduced overall and particularly reduced in the proximal two thirds of the vein. Varicosities are also found in consistent locations distal to valves. In addition, valvular incompetence occurs in the absence of venous symptoms, suggesting it may be the primary event.

A study of 4020 limbs investigated with venous colour duplex confirmed that in patients with varicose veins, the majority had incompetence at the SFJ, of those, 72% had SFJ incompetence with complete incompetence of the entire GSV and a further 19% had above knee GSV incompetence. Distal incompetence in the presence of SFJ incompetence was rare (4%) [11].

Although the descending theory cannot account for every observed pattern of venous reflux, the presence of an incompetent SFJ significantly influences patterns of reflux and provides support for the descending theory.

Keywords: *terminal valve, saphenofemoral junction, venous disease, truncal reflux.*

Résumé

L'étiologie et la pathogénie des varices restent mal comprises. Actuellement, deux théories existent : la théorie dite ascendante et la théorie descendante.

La théorie dite descendante *veut que l'origine primitive des varices soit liée à la présence d'anomalies proximales des veines superficielles qui affectent habituellement la valve terminale de la jonction saphénofémorale (JSF) ou celle de la jonction saphéno-poplitée (JSP).*

Les valvules incompétentes du segment proximal d'une veine conduisent à un reflux du sang veineux et à une augmentation de la pression hémodynamique sur la valve immédiatement sous-jacente, ce qui provoque son incompétence et ensuite une dilatation de la veine sous-jacente. Cette théorie s'appuie sur des études cadavériques, qui démontrent des valves en nombre réduit ou absentes dans les veines iliaque externe et fémorale, chez des cadavres présentant des signes d'une insuffisance veineuse. En plus, dans les études cadavériques et celles portant sur les membres des patients bénéficiant d'une chirurgie des veines superficielles, le nombre des valvules est significativement réduit globalement et en particulier dans les deux tiers proximaux de la veine saphène (par rapport à celle des membres sains). On trouve également et de manière significative des varices dans les veines immédiatement sous le siège des valvules. De plus, l'incompétence valvulaire survient en l'absence de symptômes veineux, ce qui suggère que ceci pourrait être l'évènement primaire.

Une étude portant sur 4 020 membres analysés par un écho-Doppler couleur, chez des patients atteints de varices, a confirmé que la majorité avait une incompétence de la JSF. Parmi ceux-ci, 72 % avaient une incompétence complète de la GVS sur tout son trajet et 19 % avaient une incompétence de la GVS au-dessus du genou. Une incompétence distale avec incompétence de la JSF a été trouvée rarement (4 %) [11].

Bien que la théorie dite descendante ne puisse pas expliquer tous les schémas de reflux veineux observés, la présence d'une JSF incompétente influence significativement les schémas du reflux et vient en appui de la théorie dite descendante.

Mots-clés : valve terminale, jonction saphénofémorale, insuffisance veineuse chronique, reflux tronculaire.

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Introduction

Despite their high prevalence, the aetiology and pathogeneses if varicose veins remains poorly understood.

Histological studies confirm that varicose veins contain abnormalities in the vein wall, including changes in the vessel intima, connective tissue and smooth muscle [1], although the precise mechanism of how this arises is incompletely understood.

A cycle of events develops and then progresses, leading to worsening disease. Venous hypertension leads to an increase in vein wall stress, increasing stretch and producing changes in shear stress. This potentiates changes in the regulation of intracellular signalling pathways, an example being the up-regulation of matrix metalloproteinases and tissue inhibitors of metalloproteinase that in turn, modify the vein wall reducing compliance and elasticity, causing vein wall weakening and relaxation.

This leads to further valvular incompetence and venous reflux, blood stasis and worsening venous hypertension [2]. ***However, the initiating factor in the cycle is unknown.***

At present, several theories of the development of this pattern exist, including the ascending, descending and multifocal theories of varicose vein development.

The evidence providing support for the descending or saphenocentric theory of superficial venous insufficiency is discussed below.

Cadaveric studies

For many years the development of varicose veins was thought to occur due to processes based around the descending theory, which proposes that the primary pathology arises from proximal abnormalities affecting the terminal valve at the saphenofemoral junction (SFJ) or saphenopopliteal junction (SPJ).

In 1891, ***Trendelenburg*** proposed that the venous valves acted to protect distal venous wall from the pressure of blood acting on the valves, and that valve failure lead to reflux of blood, stasis and venous hypertension, causing damage to the distal vein and resulting in secondary dilatation. Numerous published studies support this theory to some extent.

Among the earliest are cadaveric studies of 38 patients by ***Eger and Casper***, reporting reduced or absent valves in the external iliac and femoral veins in patients with evidence of venous insufficiency, implying that increased hydrostatic pressure and gravity lead to primary valvular failure that began proximally, and eventually resulted in distal varicosities.

A further study comparing limbs undergoing superficial venous surgery with normal limbs, cadaveric specimens and amputated limbs, reported that valves were significantly reduced in density and distribution particularly in the proximal two thirds of the vein in those with varicose veins compared to non varicose specimens [3].

Varicosities were also found at consistent locations distal to incompetent valves in this study [3].

The idea that valvular incompetence was the primary event in the development of venous disease was also supported by ***Ludbrook***, who proposed that it preceded the development of distal varicosities [4].

Venography studies

In 1980, ***Kistner*** published studies of descending venography evaluating superficial incompetence in patients with lower limb pain, swelling, and venous ulceration.

He noted valvular incompetence in the absence of symptoms in some cases suggesting it to be a primary event.

He proposed that incompetent valves in a proximal vein segment result in the reflux of venous blood and an increase in haemodynamic pressure on the valve immediately distal, resulting in subsequent incompetence and distal vein dilatation [5].

It is also known that the incidence of both superficial and deep venous reflux increases following deep venous thrombosis.

There is some evidence to suggest that this begins at the valve sinus and that valvular reflux subsequently develops at this site, therefore subclinical thrombosis may be a possible mechanism for primary varicose veins in some instances [6].

Histological studies

Histological evidence exists to support a primary valvular failure, including an absence of valves, or morphological abnormalities such as dilatation of the valvular annulus and increased thickening, scarring and tearing of valves that occur more frequently in varicose than non varicose veins [7, 8].

Valves in varicose veins contain reduced collagen, and the surrounding perivalvular vein wall shows a lack of the viscoelastic properties seen in normal veins [9].

Increased monocyte and macrophage infiltration has been observed in the valves of varicose veins compared to normal veins, and therefore leukocyte infiltration in the vein wall and valve leaflets may be an important cause of primary venous dysfunction [10].