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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].

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Clinical studies

In 2009, a retrospective study at Charing Cross Hospital London evaluated **4020 limbs** in patients with known venous disease who had undergone a venous colour duplex scan performed by an accredited vascular scientist between 01/01/2009 and 31/08/2009.

Data were examined with the aim of providing support for either the ascending or descending theory of venous disease and to determine the influence of junctional incompetence of the terminal valve on saphenous reflux [11].

The great and small saphenous veins were considered separately and patients were classified into those who had incompetence of the terminal valve and those who did not. Patients who had undergone previous treatment for superficial reflux, those with deep venous disease and those with congenital vascular malformations were excluded from the analysis.

A total of 2137 patients had incompetence of the SFJ, and of those 72% had incompetence of the great saphenous vein above and below the knee, 19% had incompetence of the great saphenous vein above the knee only and 9% had below the knee incompetence or no incompetence [11], showing a strong association between junctional incompetence and truncal reflux.

In total, 1883 patients had a competent SFJ, and of those, 32% had no reflux, (ie tributary, perforator, or deep venous incompetence), 36% had reflux below the knee, 11% had reflux above the knee and 21% had reflux above and below the knee [11], and hence, the pattern was less clear.

Of the 871 patients with SPJ incompetence, 68% had incompetence of the SSV, however, **630 patients** had incompetence of the SSV in the absence of SPJ incompetence. Patterns of reflux observed in the study were variable and limitations included the absence of clinical data to correlate with duplex findings.

However, an association between the presence of an incompetent terminal valve and the existence of truncal reflux was observed.

Conclusion

For over a century, the principles of diagnosis and treatment of superficial venous insufficiency have been based on upon the descending theory of venous disease.

The presence of valvular incompetence, detectable on hand held Doppler, was frequently used to confirm the diagnosis of superficial venous disease prior to great saphenous vein stripping. This has been superseded by venous duplex imaging.

Throughout the world, removal of the incompetent segment of vein, either by traditional surgery, or by endovenous thermal or chemical ablation forms the basis of the majority of treatments performed today [12].

In addition principles such as CHIVA (Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire) are based around the development of abnormal re-entry flow loops of blood, to which valvular incompetence is key [13], although, conversely, the concept of Ambulatory Selective Varices Ablation under Local Anaesthetic (ASVAL) is based upon the ascending theory of venous disease.

Nevertheless, although the descending theory cannot account for every observed pattern of venous reflux, the presence of an incompetent SFJ appears to impose a significant influence on patterns of superficial venous reflux. This support for the descending theory is likely to represent an important part of a multi-factorial disease process contributing to superficial venous insufficiency.

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